



LEKARZ WOJSKOWY

MILITARY PHYSICIAN



2026

NR 1 VOL. 104

ISSN 0024-0745



- The impact of bruxism on the health of stress-exposed individuals – soldiers: diagnosis, treatment, and prevention
- When myocardial infarction is not the actual diagnosis – diagnostic challenges in acute settings
- The evolving role of artificial intelligence in orthodontic diagnosis and treatment planning
- Doping – a path to chronic kidney disease. Case report of 45-year-old powerlifter



**WOJSKOWY
INSTYTUT MEDYCZNY
PAŃSTWOWY INSTYTUT BADAWCZY**

Informacje dla autorów

Informacje ogólne

„Lekarz Wojskowy” jest czasopismem ukazującym się nieprzerwanie od 1920 r., obecnie jako kwartalnik wydawany przez Wojskowy Instytut Medyczny w Warszawie.

1. „Lekarz Wojskowy” zamieszcza prace oryginalne (doświadczalne i kliniczne), prace poglądowe, doniesienia dotyczące zagadnień wojskowych, opracowania deontologiczne, opracowania ciekawych przypadków klinicznych, artykuły z historii medycyny, prace dotyczące aspektów prawa medycznego, opisy wyników racjonalizatorskich, wspomnienia pośmiertne, listy do Redakcji, oceny książek, streszczenia (przeglądy) artykułów z czasopism zagranicznych, szczególnie dotyczących wojskowej służby zdrowia, sprawozdania ze zjazdów i konferencji naukowych, komunikaty o zjazdach. Publikacja oryginalna może mieć także formę krótkiego doniesienia wstępnego.
2. Każda praca przed przyjęciem do druku jest oceniana przez dwóch niezależnych recenzentów, z zachowaniem anonimowości. Czas na odpowiedź na zaproszenie do recenzji wynosi 7 dni, czas na recenzję 30 dni, czas na recenzję poprawionej pracy 14 dni.
3. Przesyłając pracę kliniczną, należy zadbać o jej zgodność z wymogami Deklaracji Helsińskiej, w szczególności o podanie w rozdziale „Materiał i metody” informacji o zgodzie Komisji Bioetycznej, jak również o świadomej zgodzie chorych na udział w badaniu. W przypadku wykorzystania wyników badań z innych ośrodków należy to zaznaczyć w tekście lub podziękowaniu.
4. Autorzy badań klinicznych dotyczących leków (nazwa międzynarodowa) i procedur medycznych powinni przedstawić opis finansowania badań i wpływu sponsora na treść publikacji.
5. Autor ma obowiązek dostarczyć Redakcji zgodę właściciela ilustracji na ich użycie w artykule.
6. Prace należy nadsyłać przez Editorial System.
7. Redakcja zwraca się z prośbą do wszystkich autorów pragnących zamieścić swe prace na łamach „Lekarza Wojskowego” o dokładne zapoznanie się z niniejszymi zasadami i ścisłe ich przestrzeganie. Niestosowanie się do wymagań Redakcji utrudnia redagowanie, zwiększa koszty i opóźnia ukazywanie się prac. Prace napisane niezgodnie z niniejszymi zasadami nie będą publikowane, a przygotowane niewłaściwie, będą zwracane autorom w celu ich ponownego opracowania.

Maszynopis wydawniczy

1. Artykuły należy przygotować w edytorze tekstu WORD i przesłać przez Editorial System. Tekst musi być napisany czcionką Times New Roman 12 pkt, z podwójnym odstępem między wierszami (dotyczy to też piśmiennictwa, tabel, podpisów itd.). Nowy akapit zaczyna się od lewego marginesu, bez wcięcia akapitowego. Nie wstawia się pustych wierszy między akapitami lub wycięziami.
2. Liczba stron maszynopisu (łącznie z tabelami, rycinami i piśmiennictwem) nie może przekraczać w przypadku prac oryginalnych – 30, poglądowych – 20, kazuistycznych – 10, z historii medycyny – 20, racjonalizatorskich – 15 stron. Streszczenia ze zjazdów, kongresów itp. powinny być zwięzłe (do 5 stron) i zawierać tylko istotne informacje. W każdym przypadku piśmiennictwo nie powinno zawierać więcej niż 35 pozycji.
3. Tekst główny (z piśmiennictwem) umieszcza się w odrębnym pliku. Prace oryginalne powinny być przygotowane zgodnie z układem: wstęp, cel pracy, materiał i metody, wyniki, omówienie, wnioski, piśmiennictwo; prace kazuistyczne: wstęp, opis przypadku, omówienie, podsumowanie (wnioski), piśmiennictwo. Skróty i akronimy powinny być objaśnione w tekście przy pierwszym użyciu, a potem konsekwentnie stosowane.
4. W tekście głównym należy zaznaczyć miejsca wstawiania rycin i tabel, np.: „na rycinie 1”, „(tab. 1)”. Tabele, ryciny i zdjęcia załącza się w osobnych plikach. Liczba tabel należy ograniczyć do minimum. Zdjęcia cyfrowe powinny mieć min. rozdzielczość 300 dpi.
6. Prace powinny być przygotowane starannie i zgodnie z zasadami pisowni polskiej, ze szczególną dbałością o komunikatywność i polskie mianownictwo medyczne. Teksty niespełniające tych kryteriów będą odsyłane do poprawy.
7. Informacje afiliacyjne: imię i nazwisko autora lub autorów (maks. 10 osób), z tytułami naukowymi, pełną nazwą zakładu (zakładów) pracy umieszcza się w programie Editorial System. Należy wskazać także autora do korespondencji. Nie należy zamieszczać tych informacji bezpośrednio w wysłanym manuskrypcie.
8. Streszczenie (do 250 słów) w języku polskim lub angielskim, ze słowami kluczowymi w języku polskim i angielskim, także należy załączyć w programie Editorial System. Nie należy umieszczać streszczenia dodatkowo w pliku manuskryptu.
9. Piśmiennictwo powinno być ułożone zgodnie z kolejnością pojawiania się odsyłaczy w tekście. Jeśli artykuł ma nie więcej niż czterech autorów, należy podać nazwiska wszystkich; jeśli autorów jest więcej – trzech pierwszych, z dopiskiem „et al.” Numerację piśmiennictwa należy wprowadzać z klawiatury, nie korzystając z możliwości automatycznego numerowania. Przykłady cytowań:
Artykuły z czasopism:
Calpin C, Macarthur C, Stephens D, et al. Effectiveness of prophylactic inhaled steroids in childhood asthma: a systemic review of the literature. *J Allergy Clin Immunol*, 1997; 100: 452–457. doi: 10.1016/s0091-6749(97)70134-9
Książki:
Rudziński E. *Alergia na leki: z uwzględnieniem odczynów anafilaktycznych i idiosynkrazji*. Lublin, Wydawnictwo Czelej, 2002
Rozdziały książki:
Wantz GE. Groin hernia. In: Cameron JJ, ed. *Current surgical therapy*. St Louis, Mosby, 1998: 557–561
W wykazie piśmiennictwa należy uwzględnić tylko te prace, z których autor korzystał, a ich liczbę należy ograniczyć do 35. W tekście artykułu należy się powołać na wszystkie wykorzystane pozycje piśmiennictwa, a numer piśmiennictwa umieścić w nawiasie kwadratowym. Tytuły należy kopiować z medycznych baz danych w celu uniknięcia pomyłek.
10. Przesłany artykuł musi spełniać kryteria zgody na publikację zgodnie z zasadami przyjętymi w jednostce afiliacyjnej autora. Do pracy należy dołączyć: oświadczenie, że praca nie została wcześniej opublikowana i nie jest złożona do innego czasopisma, oświadczenie o konflikcie interesów oraz ewentualne podziękowanie.
11. Redakcja zastrzega sobie prawo poprawienia mianownictwa i usterek stylistycznych oraz dokonanie skrótów bez uzgodnienia z autorem.
12. W przypadku nieprzyjęcia pracy do publikacji Redakcja poinformuje autora pisemnie o takiej decyzji.

Zasady etyki publikacyjnej

1. Czasopismo „Lekarz Wojskowy” stosuje zasady etyki publikacyjnej mającej na celu przeciwdziałanie nieuczciwym praktykom.
2. Materiały zgłoszone Redakcji „Lekarza Wojskowego” do publikacji są weryfikowane pod kątem zgodności z zasadami etyki wydawniczej, opisanymi m.in. w następujących dokumentach:
 - 1) wytyczne Komitetu ds. Etyki Publikacyjnej (Committee on Publication Ethics – COPE), „Kodeks etyki pracownika naukowego” (opracowany przez Komisję ds. Etyki w Nauce, wydanie drugie, PAN, Warszawa 2017);
 - 2) „Dobre praktyki w procedurach recenzyjnych w nauce” (opracowane przez Zespół ds. Etyki w Nauce, który doradzał Ministrowi Nauki i Szkolnictwa Wyzszego w latach 2009–2010);
 - 3) „Zrzetelność w badaniach naukowych oraz poszanowanie własności intelektualnej” (Warszawa, 2012, MNIŚW).
3. Redakcja „Lekarza Wojskowego” w szczególności:
 - 1) przeciwdziała zjawiskom ghostwritingu oraz guest authorshipu, które są przejawem nierzetelności naukowej;
 - 2) przestrzega przepisów prawa autorskiego zgodnie z ustawą z dnia 4 lutego 1994 r. o prawie autorskim i prawach pokrewnych (Dz. U. z 2021 r. poz. 1062, z późn. zm.);
 - 3) dokłada należytej staranności, by zapobiegać plagiatom i autoplagiatom poprzez zastosowanie systemu autodiagnostycznego Crossref Similarity Check, przy użyciu oprogramowania iThenticate;
 - 4) ma prawo wycofać tekst (nawet po opublikowaniu), jeśli istnieją dowody świadczące o braku wiarygodności wyników badań i/lub fałszowaniu danych i/lub praca nosi znamiona plagiatu albo narusza zasady etyki wydawniczej;
 - 5) z chwilą wykrzycia nieuczciwych praktyk jest zobowiązana:
 - a) skierować sprawę do Rzecznika Dyscyplinarnego WIM, jeśli dotyczy utworu pracownika naukowego;
 - b) w uzasadnionych przypadkach niezwłocznie informować o tych praktykach stosowne instytucje naukowe, jak również odpowiednie organy ścigania;
 - 6) zapewnia profesjonalny proces wydawniczy;
 - 7) zapewnia poufność i bezpieczeństwo przetwarzania danych osobowych zgodnie z obowiązującymi przepisami (m.in. RODC).

Information for the authors

General information

“Military Physician” has been published continuously since 1920, currently as a quarterly of the Military Institute of Medicine in Warsaw, Poland.

1. “Military Physician” publishes original (experimental and clinical) articles, reviews, reports on military issues, deontological papers, interesting case reports, articles on the history of medicine, descriptions of rationalisation results, posthumous memoirs, letters to the editor, book reviews, article (reviews) summaries from international journals particularly on military health service, reports on meetings and scientific conferences, and announcements of events. An original publication may also have the form of a short temporary report.
2. Before publication, each article is reviewed by 2 independent reviewers while maintaining anonymity. The time to respond to a review invitation is 7 days, the time to complete a review is 30 days, and the time to complete the review of a corrected article is 14 days.
3. A clinical article for submission should be in accordance with the requirements of the Declaration of Helsinki, and the “Material and methods” section should contain both information on the approval of the Editorial Committee and the patients’ informed consent to participate in the study. In the case of using the results of studies conducted by other centres, such information should appear either in the text or in the acknowledgements.
4. Authors of clinical studies on medications (international name) and medical procedures should provide a description of the research funding and the influence of the sponsor on the content of the publication.
5. The author must provide the editorial board with the consent of the owner of an image to use the image in an article.
6. Please submit your article using the Editorial System.
7. All authors who wish to publish their papers in Military Physician are asked to carefully read and strictly follow the guidelines listed below. Failure to follow the requirements of the Editorial Board makes editing more difficult, increases costs and delays publication. Manuscripts not meeting the requirements will not be published, and those considered inadequately prepared will be returned to the authors for revision.

Manuscript

1. Articles should be in MS Word and sent by Editorial System. The text must be written in Times New Roman 12 point font, double spaced (this also applies to references, tables, captions, etc.). A new paragraph should be started from the left margin without paragraph indentation. Please do not insert blank lines between paragraphs or enumerations.
 2. The number of pages of the manuscript (including tables, figures and references) cannot exceed 30 pages for original articles, 20 for review articles, 10 for reports, 20 for articles on the history of medicine and 15 for rationalisation articles. Reports on meetings and conferences should be concise (up to 5 pages) and discuss only significant issues. The number of references should never exceed 35.
 3. Text (with references) should be uploaded as a separate file. Original articles should be prepared according to the following structure: introduction, aim, material and methods, results, discussion, conclusions, references; case reports: introduction, case description, discussion, summary (conclusions), and references. Abbreviations and acronyms should be defined when first mentioned in the text and consequently used in the paper.
 4. Figures and tables should be referenced in the body of the text as follows: “in Figure 1”, “(Tab. 1)”. Tables, figures and images should be saved in a separate file. The number of tables should be reduced to a minimum. Digital images should have a resolution of 300 dpi.
 6. Papers should be prepared carefully, in accordance with Polish spelling and with special attention to communicativeness and Polish medical nomenclature. Manuscripts that do not meet the criteria will be sent back to the authors for revision.
 7. Affiliation information: author’s or authors’ (max. 10 people) first and last names, including academic degrees, full name of affiliated institute (institutes) should be introduced in the Editorial System. Please indicate the corresponding author. Please do not include this information directly in the manuscript.
 8. An abstract (up to 250 words) in Polish or English with keywords in Polish and English should also be saved in the Editorial system. Please do not also include the abstract in the manuscript.
 9. References should be presented according to the order in which they appear in the text. If the article has up to four authors then all of them should be named, while if there are more, then name only the first three, followed by “et al.”. References should be numbered using the keyboard, please do not use automatic numbering.
- Examples of citations:
- Journal articles:
Calpin C, Macarthur C, Stephens D, et al. Effectiveness of prophylactic inhaled steroids in childhood asthma: a systemic review of the literature. *J Allergy Clin Immunol*, 1997; 100: 452–457. doi: 10.1016/s0091-6749(97)70134-9
- Books:
Rudziński E. *Alergia na leki: z uwzględnieniem odczynów anafilaktycznych i idiosynkrazji*. Lublin, Wydawnictwo Czelej, 2002
- Chapter of a book:
Wantz GE. Groin hernia. In: Cameron JJ, ed. *Current surgical therapy*. St Louis, Mosby, 1998: 557–561
- The list of references should include only those publications that were used by the author and should be reduced to 35. All references should be cited in the text and the numbers of references should be put in square brackets. In order to avoid errors, titles should be copied from medical databases.
10. The submitted article must comply with the criteria for consent to publication adopted in the author’s institution. The paper should be accompanied by: a declaration that the article has not been published before or simultaneously submitted to any other journal, Declaration of Conflict of Interest and acknowledgements, if applicable.
 11. The Editorial Board reserves the right to correct nomenclature and stylistic errors as well as to introduce abbreviations without consultation with the author.
 12. If the manuscript is not accepted for publication, the Editorial Board will inform the author in writing about the decision.

Principles of publication ethics

1. The journal “Lekarz Wojskowy” applies principles of publication ethics aimed at preventing unfair publication practices.
2. Materials submitted to the editorial board of “Lekarz Wojskowy” for publication are reviewed for compliance with the principles of publication ethics described in the following documents:
 - 1) the guidelines of the Committee on Publication Ethics (COPE), the “Code of Ethics for a Scientific Employee” (developed by the Commission on Ethics in Science, second edition, PAN, Warsaw 2017);
 - 2) “Good practices in review procedures in science” (developed by the Team for Ethics in Science, which advised the Minister of Science and Higher Education in 2009–2010);
 - 3) “Reliability in scientific research and respect for intellectual property” (Warsaw, 2012, Ministry of Science and Higher Education).
3. The editorial board of “Lekarz Wojskowy” in particular:
 - 1) counteracts the phenomena of ghostwriting and guest authorship, which are manifestations of scientific unreliability;
 - 2) observes the provisions of copyright law in accordance with the Act of 4 February 1994 on Copyright and Related Rights (Journal of Laws of 2021, item 1062, as amended);
 - 3) exercises due diligence to prevent plagiarism and self-plagiarism through the use of the Crossref Similarity Check anti-plagiarism system using the iThenticate software;
 - 4) has the right to withdraw a text (even after publication) if there is evidence of unreliability of research results and/or falsification of data and/or the work shows signs of plagiarism or breaches the rules of publishing ethics;
 - 5) as soon as it discovers unfair practices, it is obliged to:
 - a) refer the matter to the Disciplinary Ombudsman of the WIM, if it concerns the work of a researcher;
 - b) in justified cases, immediately inform the relevant scientific institutions, as well as the appropriate law enforcement authorities about these practices;
 - 6) ensure a professional publishing process;
 - 7) ensure confidentiality and security of personal data processing in accordance with applicable regulations (including GDPR).



■ Letter from the Editor-in-Chief

Dear Colleagues and Readers,

It is with great satisfaction that we present to you the first issue of *Military Physician* in 2026 – a year that will undoubtedly bring new challenges, intensive scientific work, and a continued search for answers in an ever-evolving medical landscape.

This issue features extensive review articles covering public health and psychiatry, as well as topics of particular importance to the military community – including the impact of stress on the prevalence of bruxism among soldiers, diagnostic challenges in emergency medicine, and the practical aspects of treating tetanus. I also encourage you to explore the articles devoted to modern technologies, such as the role of m-health in the management of atrial fibrillation and the application of artificial intelligence in orthodontic diagnostics and treatment planning.

A valuable addition to this issue is the second part of a study on the medical service during the 1939 Polish Campaign, serving as a reminder of the importance of past generations' experiences for the modern organisation of medical support.

Completing the issue is a scientific report from an international cardiology congress, offering the latest recommendations relevant to physicians across multiple specialties.

We hope that the content presented here will serve as a source of knowledge, inspiration, and practical support in your clinical and scientific work. We invite you to enjoy this issue and to actively contribute to the future editions of our journal.

Wishing you an inspiring read.

With best regards

A handwritten signature in blue ink, appearing to read 'B. Kalicki'.

Prof. Bolesław Kalicki, MD, PhD



Editor-in-Chief:
Prof. Bolesław Kalicki, MD, PhD

Deputy Editors-in-Chief:
Prof. Krzysztof Korzeniewski, MD, PhD
Lt. Col. Agata Będzichowska, MD, PhD

Editorial Assistant:
Ewa Jędrzejczak, MA



Subject Editors:

field: cardiology, internal medicine
field: neurology, medical rehabilitation
field: endocrinology, nephrology, internal medicine
field: nephrology, biostatistics, internal medicine
field: general surgery, surgical oncology
field: otorhinolaryngology, general surgery
field: anaesthesiology and intensive care, emergency medicine
field: psychiatry, psychology
field: biology, basic research
field: basic sciences
field: basic sciences

Beata Uziębło-Życzkowska, MD, PhD
Jacek Staszewski, MD, PhD
Col. Prof. Marek Saracyn, MD, PhD
Col. Prof. Arkadiusz Lubas, MD, PhD
Andrzej Kwiatkowski, MD, PhD
Lt. Col. Kornel Szczygielski, MD, PhD
Bartosz Rustecki, MD, PhD
Col. Radosław Tworus, MD, PhD
Katarzyna Czarnek, PhD in Biology
Robert Zdanowski, PhD in Biology
Wawrzyniec Kowalski, PhD in Law

Scientific Committee

Head:

Lt. Gen. Prof. Grzegorz Gielerak, MD, PhD

Honorary Members:

Prof. Teofan Domżał, MD, PhD
Prof. Eugeniusz Dziuk, MD, PhD
Prof. Anna Jung-Hauska, MD, PhD
Prof. Jerzy Kruszewski, MD, PhD
Jacek Siewiera, MD, PhD

Polish Members:

Prof. Andrzej Chciałowski, MD, PhD
Prof. Renata Duchnowska, MD, PhD
Prof. Mirosław Dziuk, MD, PhD
Prof. Maciej Gonciarz, MD, PhD
Prof. Wiesław W. Jędrzejczak, MD, PhD
Prof. Karina Jahnz-Różyk, MD, PhD
Prof. Kuba Jałoszyński, MD, PhD
Prof. Dariusz Jurkiewicz, MD, PhD
Prof. Grzegorz Kamiński, MD, PhD
Agnieszka Kobielałak, MD, PhD
Krzysztof Kobielałak, MD, PhD
Jolanta Korsak, MD, PhD
Col. Prof. Paweł Krzesiński, MD, PhD
Magdalena Markowska, MD, PhD
Łukasz Młynarkiewicz, PhD in Law
Prof. Stanisław Niemczyk, MD, PhD
Prof. Alojzy Z. Nowak, MD, PhD
Col. Prof. Witold Owczarek, MD, PhD
Prof. Robert Rejdak, MD, PhD
Col. Prof. Marek Rękas, MD, PhD
Prof. Piotr Rzepecki, MD, PhD
Prof. Adam Stępień, MD, PhD
Prof. Witold Tłustołowicz, MD, PhD
Prof. Paweł Włodarski, MD, PhD
Prof. Barbara Wróblewska, MD, PhD
Prof. Piotr Zaborowski, MD, PhD
Col. Prof. Grzegorz Zieliński, MD, PhD

Foreign Members:

Massimo Barozzi (Italy)
Elspeth Cameron Ritchie (USA)
Nihad El-Ghoul (Palestine)
Claudia E. Frey (Germany)
Anselm Jünemann (Germany)
Paweł Kaliński (USA)
Małgorzata Kloc (USA)
Frederic C. Lough (USA)
Marc Morillon (Belgium)
Arnon Nagler (Israel)
Tomasz Rozmystowicz (USA)
Marek Rudnicki (USA)
Valerii Savatskyi (Ukraine)
Daniel Schneditz (Austria)
Eugeny Tischchenko (Belarus)
Mario Damiano Toro (Italy)
Brenda Widerhold (USA)

Technical Editing:

Managing Editor:
Zuzanna Leszczuk, MA
Editing and proofreading:
Medical Communications,
Aleksandra Kwiatkowska-
-Przybysz, MA
English translation and proofreading:
Lingua Line Translation Agency,
ENSKA Agnieszka Kosarzycka
Cover design:
Krzysztof Gontarski MSc, Eng
Typography:
Krzysztof Gontarski MSc, Eng
DTP:
Medical Communications,
Marcin Haze

Editorial address:

Military Institute of Medicine –
National Research Institute
128 Szaserów St. 04-141 Warsaw
telephone +48 261 817 380
e-mail: lekarzwojskowy@wim.mil.pl
lekarzwojskowy.wim.mil.pl

TABLE OF CONTENTS

LETTER FROM THE EDITOR-IN-CHIEF	3
Bolesław Kalicki	

REVIEW ARTICLES

AN INTERNATIONAL APPROACH TO HOMELESSNESS. PART II. SLUMS DEMOLITION OR UPGRADING?	7
---	----------

Międzynarodowe podejście do problemu bezdomności. Część II.
Likwidacja slumsów czy ich usprawnienie?

Jarosław Richard Romaniuk, Pranit Lanjewar

PHARMACOLOGICAL STRATEGIES FOR REDUCING METABOLIC COMPLICATIONS INDUCED BY SECOND-GENERATION ANTIPSYCHOTICS IN PATIENTS WITH SCHIZOPHRENIA	14
---	-----------

Farmakologiczne strategie redukcji powikłań metabolicznych wywołanych przez leki przeciwpsychotyczne drugiej generacji u pacjentów ze schizofrenią

Hanna Weronika Wołodkiewicz, Weronika Caban

THE IMPACT OF BRUXISM ON THE HEALTH OF STRESS-EXPOSED INDIVIDUALS – SOLDIERS: DIAGNOSIS, TREATMENT, AND PREVENTION	21
---	-----------

Wpływ bruksizmu na zdrowie osób szczególnie narażonych na stres – żołnierzy: diagnostyka, leczenie i profilaktyka

Adam Łuczak, Adrianna Gliszczyńska, Konstancja Nosowicz, Patrycja Różak, Nina Szczepańska, Agata Tuczyńska

TETANUS – WHAT A SURGEON SHOULD KNOW ABOUT CLOSTRIDIUM TETANI	28
--	-----------

Tężec – co chirurg powinien wiedzieć o zakażeniu *Clostridium tetani*

Paweł Radkowski, Maciej Szewczyk, Tomasz Jędrzejak, Łukasz Banasiak, Julia Oszytko

WHEN MYOCARDIAL INFARCTION IS NOT THE ACTUAL DIAGNOSIS – DIAGNOSTIC CHALLENGES IN ACUTE SETTINGS	35
---	-----------

Kiedy zawał serca to nie zawał – diagnostyczne wyzwania w stanach nagłych

Hubert Kowal, Marta Mielniczuk, Jarosław Kowal, Paweł Krzesiński

ATRIAL FIBRILLATION – WHAT ARE THE BENEFITS OF M-HEALTH?	40
---	-----------

Migotanie przedsionków – co daje nam m-health?

Dawid Sidorczuk, Agata Galas, Paweł Krzesiński

THE EVOLVING ROLE OF ARTIFICIAL INTELLIGENCE IN ORTHODONTIC DIAGNOSIS AND TREATMENT PLANNING	45
---	-----------

Ewolucyjna rola sztucznej inteligencji w diagnostyce i planowaniu leczenia ortodontycznego

Konstancja Nosowicz, Agata Tuczyńska, Patrycja Różak, Adam Łuczak, Adrianna Gliszczyńska, Nina Szczepańska

THE EFFECT OF GARLIC ON CARBOHYDRATE METABOLISM AND GLYCAEMIC CONTROL: A REVIEW OF THE LITERATURE	50
--	-----------

Wpływ czosnku na metabolizm węglowodanów i kontrolę glikemii: przegląd literatury

Dominik Ireneusz Kret, Wiktoria Klaudia Szlachta, Antoni Anczyk, Karolina Handzel, Grzegorz Zaborowski, Jacek Wysoczański, Wiktor Kruczek, Filip Basta, Miłosz Tworek

ORIGINAL ARTICLE

MEDICAL SERVICES IN THE POLISH CAMPAIGN OF 1939 – A SYNTHESIS. PART II: PREPARATIONS AND WAR PLANS 54

Służba zdrowia w kampanii polskiej 1939 roku – synteza. Część II.
Przygotowania i plany wojenne

Aleksander Rutkiewicz

CASE REPORTS

DOPING – A PATH TO CHRONIC KIDNEY DISEASE. CASE REPORT OF 45-YEAR-OLD POWERLIFTER 61

Doping – droga do przewlekłej niewydolności nerek.
Studium przypadku 45-letniego trójboisty

Karolina Borkowska, Oliwia Mróz, Katarzyna Romejko, Elżbieta Araminowicz-Kierkło, Stanisław Niemczyk

SCHWANNOMA OF THE SMALL INTESTINAL MESENTERY IN A 38-YEAR-OLD WOMAN 65

Nerwiak ostonkowy (*schwannoma*) krezki jelita cienkiego u 38-letniej kobiety

Krzysztof Stanisław Winiarz

REPORT

CARDIOLOGIC CARE OF PATIENTS WITH CHRONIC KIDNEY DISEASE. SELECTED INSIGHTS FROM THE 29TH INTERNATIONAL CONGRESS OF THE POLISH CARDIAC SOCIETY 68

Pacjent z przewlekłą chorobą nerek u kardiologa. Wybrane zagadnienia
XXIX Międzynarodowego Kongresu Polskiego Towarzystwa Kardiologicznego

Katarzyna Romejko



AN INTERNATIONAL APPROACH TO HOMELESSNESS. PART II. SLUMS DEMOLITION OR UPGRADING?

Międzynarodowe podejście do problemu bezdomności.
Część II. Likwidacja slumsów czy ich usprawnienie?



Jaroslav Richard Romaniuk, Pranit Lanjewar

Jack, Joseph and Morton Mandel School of Applied Social Sciences, Case Western Reserve University, Cleveland, Ohio, United States

Jaroslav Richard Romaniuk –  0000-0002-8568-6587

Pranit Lanjewar –  0009-0005-0083-4042

Abstract

In search of the best approach to eradicating homelessness in underdeveloped countries with large populations, we conducted interviews with professionals involved in different models of helping the unhoused. Although it has been established that the Housing First model combined with human services is the best model to end homelessness, the cost of housing might be prohibitive for many national governments. In situations where governments are overwhelmed by the numbers of homeless people, communities and organizations develop specific methods and practices to meet the needs of unhoused individuals. For the purpose of this paper, phenomenological research was undertaken to learn how societies in different countries respond to homelessness. We examined how international cooperation and assistance might be a potential support or a hindrance, and evaluated the practicality of international voluntarism. Social responses to homelessness in different countries were studied, and the role of the local culture on the choices of practice methods was evaluated. The importance of an asset-based community approach in developing organic social programs was also demonstrated. The findings suggest that community assets and resources should be utilized by national programs to support access to safe housing.

Streszczenie

W pracy przedstawiono wyniki badań dotyczących metod radzenia sobie z bezdomnością w krajach rozwijających się z dużą gęstością zaludnienia. Powszechnie uznaje się, że najlepszą metodą wyjścia z bezdomności jest model „Najpierw mieszkanie”, ale wysoka cena tego podejścia uniemożliwia jego wykorzystanie przez państwa o ograniczonych budżetach. W warunkach niskiego poziomu dochodów państwa wiele organizacji pozarządowych i komunalnych tworzy systemy pomocy osobom bezdomnym, korzystając z własnych zasobów. Aby ustalić, jak taka pomoc jest organizowana, przeprowadziliśmy badania fenomenologiczne w każdym z badanych w tej pracy krajów. Szczególną uwagę poświęciliśmy pomocy dostarczanej przez kraje rozwinięte w formie narzuconej współpracy lub w ramach międzynarodowego wolontariatu. W badaniach uwzględniliśmy wpływ lokalnych tradycji kulturowych na wybór metod i praktyk społecznych. Zwróciliśmy uwagę na rozwój społeczny i ekonomiczny oparty na zasobach poszczególnych środowisk lokalnych. Obserwacje te doprowadziły nas do wniosku, że lokalne zasoby mogą być istotnym czynnikiem rozwoju organicznych metod radzenia sobie z bezdomnością.

Keywords: favela; asset-based community development; international assistance; voluntarism

Słowa kluczowe: favela; rozwój społeczności oparty na zasobach; pomoc międzynarodowa; wolontariat

DOI 10.53301/lw/204972

Received: 04.04.2025

Accepted: 12.05.2025

Published: 31.03.2026

Corresponding author:

Jaroslav Richard Romaniuk
Jack, Joseph and Morton Mandel School of Applied
Social Sciences Case Western Reserve,
University Cleveland, Ohio, 11235 Bellflower Rd, 44106,
Cleveland, United States
e-mail: jrr3@case.edu

Introduction

The most frequently applied method of helping people who are unhoused or at risk of homelessness is the provision of public housing that offers subsidized lodging. However, there are always limits and conditions on when and how people might receive such accommodation. Individuals who are unable to obtain housing support, after utilizing and eventually exhausting all their potentially available resources, find themselves living on the street. Then they have to search for accessible shelter where they are able to find relative safety, rest, and food. The simplest way is to find shelter in slums among people with similar needs and resources. However, this exposes them to significant health concerns due to poor sanitization and overcrowding [1]. Our experiences from studying the case of India (Part I) prompt several questions: What does “ending homelessness” really mean for the survival of slums? Should slums be demolished, as they will not be needed? Unfortunately, as we learned from such an approach in India, physical destruction of slums simply pushes more people into living on the street. Part I of our work concludes with a vision of communities taking care of their poor. In this second part, models of cooperation aimed at creating sustainable social change that may lead to the elimination of homelessness are examined.

Goals and methods

It is generally accepted that improvements in public programs designed to help the unhoused increase program expenditures. Although advocates for change say that improvements will lower long-term costs of services for unhoused people [2], short-term expenditures will increase. This seems to be true in the United States, where both housing and the work of professionals are becoming very expensive. The transfer of ideas and knowledge on how to eradicate homelessness from the United States to other countries such as India seems impracticable due to the high cost of the methods described as evidence-based practices in the American context. As noted in Part I, the best methods to eliminate homelessness are based on the application of the Housing First model, together with simultaneous health promotion and efforts to address social disparities [3].

In the search for the best model of affordable housing for countries like India, we decided to study how other low-income countries with limited resources and a large unhoused populations address homelessness. Primary and secondary sources were used to investigate the best methods to eliminate homelessness, and field visits enabled direct observation of professional practice in the field of homelessness in some of the countries of our interest. The methodological approach in essence followed the principles of phenomenological research to answer basic questions about the status quo of homelessness in each of the environments studied.

During the research process, we noticed that each topic that we stumbled on, relevant to our goal, is studied within its own specialty. Very seldom did we find discussion concerning overlapping topics. The topics listed below address specific aspects of the problem, issues related to the existence of slums, their origins, and potential eradication:

- Research and science of architecture and functional city [4–6].
- International cooperation and assistance [7].
- Asset-Based Community Development [8, 9].
- Practicality of international volunteerism [10].

Differences in the cultures of the people studying the problem and those directly experiencing it, such as the cultural dimensions of individualism versus collectivism [11].

Our investigation of the best approaches to eradicating homelessness drew on all of the above areas of research, as they provide a foundation for community development in an age of globalization.

During our visits to different countries, we interviewed individuals directly involved in community development and working toward improving standards of living for their most vulnerable populations. We were able to collect information from numerous communities either through our membership in these communities (India for Lanjewar and Poland for Romaniuk) or via international volunteering (India, Brazil, Tanzania, Zimbabwe, Mexico, and Guatemala). International volunteering as a method of research allows for an in vivo study of community life and observation of organic communal responses to homelessness. A common conclusion from the authors' research and experiences is that societal responses to homelessness depend on the level of poverty, community assets, state and regional policies, and local traditions for assisting the vulnerable. One key lesson is that communities tend to self-organize to compensate for the lack of governmental responses to homelessness. Among the robust responses to homelessness is the creation of slums – a phenomenon on which we place special emphasis.

There are no reliable measures of homelessness, especially in low-income countries with large populations. For the purpose of this article, we drew data from the World Population Review (worldpopulationreview.com). Measures of homelessness as a human condition depend on many factors, such as climate change, ongoing conflicts, post-colonial histories, population size, and national income level. Our selection of countries to visit and discuss depended on how the world media presents their problems and successes. The article by Daniil Filipenco: “Homelessness statistics in the world: causes and facts” published on Dec 13, 2023, in Development Aid (<https://www.developmentaid.org/>) is one of our resources we utilized.

Before presenting our thoughts concerning the role of slums in contemporary discussions of homelessness, it is important to reiterate a point made in Part I of our work: the best approach to ending homelessness is investment in supportive public housing. In the long term, this approach is cheaper than the cost of homelessness [12]. It is also more cost-effective to treat the root causes of homelessness – such as substance abuse, domestic violence, and mental illness – to prevent many people from becoming unhoused [13, 14]. Many interventions aimed at assessing people experiencing homelessness are unsuccessful because of stigma and social exclusion [15]. Efforts to change the public image of homelessness might therefore be the most important approach. In practice, however, policies addressing homelessness are often dic-

tated by public demands to make unhoused people disappear from the public space.

There are, however, situations in which an entire country is devastated by an earthquake, war, or pandemic, and there are not enough resources to help millions of unhoused people without a livelihood. In such situations, only organized international aid can provide relief. The 2010 earthquake in Haiti exemplifies this situation, as does the response of the United States Agency for International Development (USAID). What is most interesting about the assistance provided by USAID is that it was not simply an offer of money, food, and shelter; it also included assistance to community organizations with the aim of eliminating the impoverished country's social problems and promoting long-term development [16].

International discourse

Our knowledge concerning how different countries prevent and work toward ending homelessness is based on our encounters with professionals from various countries and visits to their places of work. We found out that, to understand people's approach to homelessness and proposed models of assistance, it is necessary to know the history of that country, its traditions, and the social capital of its activists. What works in one country may not be acceptable in another, as the integration of models into the broader social system might be different.

In most countries, national, regional, and local authorities develop national strategies to eradicate homelessness in an attempt to help the unhoused. In some countries, such as Mexico and Guatemala, there are no unified national programs. In Guatemala, 50% of the population lives below the poverty line, and homelessness is a considerable social problem. In emergency situations, the government may offer shelters and humanitarian aid, but the main responsibility for helping the unhoused is left to local communities.

It is not easy to evaluate each national and regional program and its effectiveness. Each of these programs exists within a distinct historical, cultural, and economic context. It is easier to observe how local communities respond to the unhoused people living among them and how those individuals attempt to improve their living conditions. We need to ask questions such as the following: Are there government offices where people in need may apply for benefits, or are unhoused people left alone with their own resources? Our goal was to study how national policies and practices are translated into shared knowledge on how to assist the unhoused. We spoke with both those who helped people who were unhoused and those who received the help. The most interesting were community organizations, ranging from very small initiatives in Tanzania to much larger ones with offices located in neighboring countries (e.g., MORE in Zimbabwe). Each of these organizations uses a different model of assistance, specific to the nature of the community and the culture in which the program was developed.

In Poland, during the Soviet-style economy, the government constructed housing identified by many Western publicists as "vertical slums" [4]. Similar public housing in France and Great Britain (built in the Brutalist style) was

demolished after public outcry that living conditions in these buildings were substandard. In contrast, in Poland, many examples of such housing are still functioning, and are considered very livable and even desirable. This is possible because, perhaps surprisingly, they were built according to Le Corbusier's ideal of a functional city, with tall and compact buildings, significant green space between them, and all necessary institutions and services located nearby. Housing was relatively low-cost and available to many people. Today, in a capitalist economy, the cost of new housing is much higher, and housing is often built with far less space between buildings for green areas or essential services. However, new apartments have significantly more living space than those built in countries with Soviet-style economies, so the old apartments are now used mostly for single or double occupancy.

Affordable housing in Poland is the responsibility of the national government and local municipalities which offer different forms of social housing [17]. In general, there are many governmental and non-governmental (NGO) services for the homeless in Poland [18]. The recent inflow of millions of refugees from Ukraine following the February 2022 Russian invasion tested these services. Many individuals invited refugees into their homes [19] as the first emotional response to the consequences of war. This response reflected a general lack of trust in governmental solutions to the struggles of individuals, stemming from decades of communist rule. As a result, communities tried to respond in an organic way. We visited two programs assisting the homeless organized by religious (Roman Catholic) entities. We spoke with Adriana Porowska from the Camillian Mission of Social Welfare and Janusz Sukiennik from the Caritas program *Damy Radę* ("We can do it"). The goals of both programs are to address addiction and mental health conditions through recovery, education and vocational skills, sustainable employment and independence, and the restoration of full participation in society. One of Janusz Sukiennik's model approaches is the application of so-called training housing, where individuals live before obtaining permanent housing. It is a very successful method but difficult to implement on a large scale within homeless populations. Both of the above-mentioned non-profit organizations were founded by social workers and professionals whose mission is to assist people who are unhoused. Adriana Porowska and Janusz Sukiennik are community leaders and advocates for social change. Janusz Sukiennik organized a conference to which he invited faculty from our school, specialists in recovery, and Julia Wygnańska, a local specialist on the Housing First model, to discuss international approaches to homelessness. The tension between the "recovery first and housing second" approach and the "Housing First" model [20] remains present in many discussions [21].

Brazil is known for favelas, large areas composed of many small, often self-built structures or shacks, constructed by people who cannot afford living in cities. In Rio de Janeiro, 25% of the local population lives in favelas. According to the 2022 Brazilian Census, 8.1% of the Brazilian population resides in favelas. There are many descriptions of the favela phenomenon. Some portray them as places filled with gangs, crime, and narcotics, while others see them as communities in which residents

self-organize and assemble all necessary services to improve their living conditions [22]. One of the authors of this article worked as a volunteer in one of Rio de Janeiro's favelas and participated in a community-based, self-organized system of care. It is worth noting that the favela community cooperates with an international volunteer organization to recruit English teachers who can provide free instruction to local activists. Volunteers also take part in creating gardens and landscaping the favela's public places.

We heard the story of two brothers from a favela: one was killed by a local gang, and his younger brother, Alan Duarte, went on to create a center for the favela's youth where they could learn self-defense and boxing. The name of his NGO organization is Abraço Campeão (Embrace Champions) and it is based in Complexo do Alemão in Rio de Janeiro. The organization was featured in the documentary *The Good Fight*, which received an award at the Tribeca Film Festival. Alan Duarte is very proud of his community achievements and a great example of the power of leadership.

In Moshi, Tanzania, when Romaniuk requested to visit impoverished areas, he was taken to a part of the city that was visibly poor and was told that he would not be safe walking there alone. This is a common belief about places where people live in desperate conditions. Romaniuk also visited equally poor areas with single-room houses, where sleeping places were on the ground, built from clay and palm leaves. These were safe areas, and often families of single women with children lived there. Neighbors helped with food and childcare, and nonprofit community organizations offered support. Deogratus Peter, the executive director of one of such organizations, Naishi Foundation, (<https://www.naishifoundation.org/>), remarked that he is grateful for the support he receives from organizations arranging help from international volunteers. Peter also developed a tourist office that organizes safaris in the Serengeti and Ngorongoro, and trips to Kilimanjaro and other parks around Moshi. He maintains connections with the Department of Social Work at Mwenge Catholic University, and he works on developing collaborations with the Mandel School of Applied Social Sciences at Case Western Reserve University, where the authors of this article studied and work. Together with volunteers, he is responsible for helping the local community with their food and health needs, and for organizing financial support through his tourist initiatives. His engagement with local and international academic social workers contributes to the sustainability of these projects and helps ensure they receive the recognition they deserve.

Whether in Brazil or Tanzania, poor neighborhoods may be either places where crime and drugs prevail or well-organized communities supported by local leaders [22]. Those who have seen examples of well-organized poor neighborhoods believe that homelessness in low-income countries cannot be directly compared to homelessness in developed countries, as community assets, financial policies, resources, and needs in these two different kinds of places are so dissimilar in both type and scale. It should also be noted that the cultures of many countries with large homeless populations can often be described as collectivist, meaning that society places greater value

on the fate of the group than on the individual. As a result, relationships tend to form more easily among people living in similar conditions in these countries, compared with those in more "developed" countries with more individualistic cultures, where unhoused individuals often wander through the streets of city centers [11, 22, 23].

Providing even limited assistance in low-income countries has a greater impact than applying the same effort in developed countries. For example, Romaniuk volunteered with a private organization in Zimbabwe named MORE – Community Foundation, which serves as a community builder in Zimbabwe and South Africa. According to its representative, Norman Mutirwara, the foundation operates according to four pillars of its mission: education, support for local businesses, improvement of living standards, and interaction between humans and animals (nature). Romaniuk observed the foundation's collaboration with local communities and how its nature-based approach affected their environment. Many community organizations like MORE – private, nonprofit groups in countries of the so-called "Global South", rely on international support obtained through international voluntarism and tourism. Such voluntarism is sometimes criticized as being driven by a "savior complex", but when the community itself requests this support in the form of organic work, then such interaction results in a greater sense of appreciation and partnership rather than feelings of superiority [10]. Writing about nature-based solutions, Santos [24] likewise emphasized the importance of voluntarism and community-based tourism in partnering with favela-type communities in Brazil.

In all of the countries the authors visited in an effort to recognize local efforts to reduce homelessness, they observed the self-organization of communities using their own assets and social capital. These organizations often reached out to international communities to access their resources and/or to learn from experiences in other settings. A good example is provided by communities in Quetzaltenango (Xela), Guatemala (entremundos.org), which requested support in many areas such as education, health, human rights, and development. Regarding homelessness in Guatemala, social workers are most concerned about unhoused children or those at risk of becoming homeless. Public health workers likewise appreciated international cooperation in responding to this need [25, 26].

Brazil and India

In the article "Slums, Favelas, and Urban Villages", Ren [22] described three similar concepts of housing utilized by people who cannot afford to live in large cities in India, Brazil, and China. Unhoused individuals tend to gather together to protect themselves and form communities; these communities eventually become phenomena of their own. Clusters of their occupants may make up a large proportion of a city's residents; their settlements are glaring evidence of the ineffectiveness of urban governance [27]. Politicians are often compelled to deal with their existence because of the high value of the land they occupy and the voting rights held by their residents.

The approach of society and its leaders to slums varies by country. In India, there is a tradition of demolishing

slums with promises to relocate their inhabitants. Such processes are always traumatic and often worse than anticipated; relocations, if they occur at all, are to places far away from areas known to the inhabitants of the slum, from their jobs, and from the social capital they have already developed. Rather than eliminating homelessness, the demolition of slums frequently leads to an increase in the number of people living on the streets [27]. In China, where the government uses its power differently, urban villages (the equivalent of slums) are demolished without any organized relocation of their residents.

The most interesting is the case of Brazil. The self-organization and self-reliance of favelas, combined with the potential of the community's political power (through voting) and their capacity to negotiate with governing bodies, has created a markedly different approach to the existence of these communities. Instead of demolition (which unfortunately may still happen), the main approach is incremental upgrading of their living areas. The city may help favelas through access to electricity, water, and sanitation, thereby creating pathways for their fuller integration into the urban system. In this respect, favelas are a great example of community development through their assets and social capital, which translates into better negotiation capabilities [8, 9]. Governing bodies, instead of destroying people's efforts to create their living space, could use their strengths to address the shortage of affordable housing for all city residents.

It needs to be acknowledged that researchers from other countries also advocate for the maintenance and upgrading of slums. Among them are Banerjee [28], Harrison and Sharma [29] in India, and Asiamah [30] in Ghana. In his work, Asiamah described different slum policies in Africa and grouped them into the following categories: benign neglect, forced eviction, resettlement programs, and slum upgrading. Asiamah [30] provided the example that during the COVID-19 pandemic, the government of Ghana decided to demolish slums to combat the spread of the virus, resulting in more than 1,000 slum dwellers becoming homeless. He discussed optimal policies concerning slums in Ghana with policymakers and residents both outside and inside slums. Proponents of demolition argued that the main reasons were to limit space for criminal activity, beautify the city, and improve public health conditions. As a result of community discussions, Asiamah advocates for the incremental upgrading of slums rather than their clearance, and calls for more discussion on this issue.

There are obvious problems regarding who will pay for upgrading slums and concerns about the viability of upgrading as a solution. Few people wish to live in small, rough housing [31]. However, asking residents of the slums to pay for it would create another barrier to a path out of poverty. Society must therefore examine how much homelessness costs everyone and how harnessing the energy and motivation of people to leave homelessness will help lower its costs.

Mexico City

Mexico City is one of the largest urban agglomerations in the world. According to the World Population Review,

in 2024 the population of Greater Mexico City reached 22.5 million, with less than half of this population located in Mexico City proper. Mexico City has a different socio-political organization than the surrounding areas of its agglomeration, which belong to the State of Mexico and Hidalgo. This difference in socio-political organization is important to understand the conditions of people experiencing homelessness. While the situation of unhoused individuals in Mexico City is considered difficult, with only minimal support from government [32], conditions in the surrounding municipalities of Greater Mexico City are dramatically worse.

The author of this article met with Mateo Rivera and other members of the nonprofit organization Mi Valedor, which supports people who live on streets. Mexico City has a significant amount of substandard housing where poor people may live, but the real slums are located outside the city limits, within Greater Mexico City (called "the state part of the city"). Social workers and advocates from Mi Valedor explained that all they can do is to help those on the street with basic needs such as food, clothes, health care, and income generation. They provide a safe, friendly space for them during daytime, offering a sense of community and belonging. The model of help presented by Mateo Rivera from Mi Valedor is a form of harm reduction that does not offer housing but rather supports people on a path toward self-sufficiency in achieving income and housing. The process might take months or years, but the organization avoids creating barriers or limits for their support. Interestingly, Arturo Soto, the director of Mi Valedor and, also, an artist, says that one of the tools for building community is developing creativity and appreciation for local art among the unhoused.

There was limited discussion concerning slums. People who experienced homelessness in "the state part of the city" presented a vision of a lawless area with millions of people living in dire conditions. Residents of this region, some of whom were born and raised there, said that they had never received any help from the government, not even from the police when they were looking for safety. They stated that people living in "the state part of the city" come from different walks of life: those affected by poverty, immigrants, the homeless, and individuals with different physical and mental disabilities or social exclusion. All of them have significant barriers and limitations in changing their life conditions. Those who are able to leave their environment and reach organizations like Mi Valedor have a chance to change their lives, even at the cost of sleeping on the streets of Mexico City. This is why people say that those experiencing homelessness in Mexico City are relatively privileged compared with other unhoused people in the greater metropolitan area.

Voluntarism

As mentioned earlier, international volunteers represent one way in which the international community can support unhoused populations in low-income countries. The best example is the work of the United Nations (UN) toward Sustainable Development Goals (SDGs) (sdgs.un.org/goals). Any work toward SDGs, especially toward Goals 1 (end poverty in all its forms everywhere) and 11 (make cities and human settlements inclusive, safe, resilient, and sus-

tainable), helps with worldwide problems of homelessness. There are many ways to motivate people to gain interest in international travel, including professional engagement, appreciation of the beauty of the world and nature, a genuine mission to help the disadvantaged, or opportunities for self-discovery and self-development [33, 34]. The UN also advocates that international tourism can be helpful in achieving SDGs when integrated with the goals of the host community. In essence, any motivation that brings people to other countries with the aim of improving lives brings us closer to achieving UN Sustainable Development Goals and eventually to the eradication of homelessness.

Through his travels to several countries as both a volunteer and tourist, Romaniuk learned about the assets of each community he visited while observing how those assets are utilized to benefit communities. Tourists have different attitudes; some are annoyed by local gifts made in China, while others are genuinely interested in the lives of local residents. Tourist-volunteers may be involved in their host countries through activities such as gardening or by applying their professional skills and knowledge as doctors or teachers. However, it has to be stated that in low-income countries and impoverished communities, money spent there is of great significance. Communities usually know what they need and how to achieve what is needed. They know their conditions and how to improve them, but often lack the financial means to do so. Money is not something shameful but simply an important element of the transactional relationship between tourists and residents of a host country. In Tanzania, for example, hosts share with tourists the beauty of Kilimanjaro, serve local coffee, and guide visitors on safaris. It is the responsibility of tourists to pay for these experiences. We observed how the money received was then utilized to support local residents, orphans, the sick, and the homeless. If tourists feel that there is something problematic about this transaction [33], then they should pay more.

Voluntarism and community building based on strengths and assets are well-established forces that can improve living standards and are present in all of the countries discussed here. They exist in developing countries, in poor neighborhoods, and in favelas among populations excluded from formal city systems. Interestingly, people from low-income communities may reach out to the academic world to present their problems to researchers, educators, and students. Similarly, academics engage with poor neighborhoods to learn about their self-management methods [35]. Mateo Rivera helps the homeless in Mexico City, but he is also an academic working on his doctoral dissertation. With the UN Sustainable Development Goals in mind, the role of the academic is to establish what works and what does not for the people they seek to support. To do so, academics must observe these communities, experience them firsthand (even if only briefly), and try to understand the specific cultural and social contexts that differ from their own daily lives.

Conclusion

The material presented here suggests that improving living standards in highly populated, low-income countries requires research into, and understanding of, the forces that give rise to slums, both horizontal and vertical, favelas, and one-room houses built from clay. Every community

and country has different land and banking policies that need to be recognized. Cultural, political, and social solidarity traditions are also of great importance when discussing pathways toward ending homelessness. When focusing on poverty and the perceived impossibility of ending homelessness, we often forget about the strengths of the people living in slums. Immersed in an environment of slums, each impoverished community has its own assets, resources, and motivation to move beyond homelessness. Some of these resources are of interest to people from other countries. Professionals, volunteers, and tourists are willing to support communities that build upon such assets, and they can create an international movement to protect what is valuable in the world. People living in slums can work where they are located. They want education, vocational training, and good health, using the services of their own communities. We suggest that the best way to achieve affordable housing is through community development based on local assets and through the creation of pathways to reintegration with the rest of society. Rather than focusing on how much ending homelessness costs, societies need to explore how much could be saved by ending homelessness and relieving human suffering. To ensure human rights, the UN Sustainable Development Goals should be prioritized as guiding objectives in countries around the world.

References

1. Ezeh A, Oyebode O, Satterthwaite D, et al. The history, geography, and sociology of slums and the health problems of people who live in slums. *The Lancet*, 2017; 389(10068): 547–558. doi: 10.1016/S0140-6736(16)31650-6
2. Keenan C, Miller S, Hanratty J, et al. Accommodation-based interventions for individuals experiencing, or at risk of experiencing, homelessness. *Campbell Systematic Reviews*, 2021; 17(2): e1165. doi: 10.1002/cl2.1165. Update in: *Campbell Syst Rev*. 2020; 16(3): e1103. doi: 10.1002/cl2.1103
3. Nuti SV, Velasquez D, Adams LD, O'Connell JJ. Precision permanent supportive housing: A data-driven approach to addressing chronic homelessness. *Health Affairs Forefront*, 2024. doi: 10.1377/forefront.20240625.514007
4. Graham S. Luxified skies: How vertical urban housing became an elite preserve. *City*, 2015; 19(5): 618–645. doi: 10.1080/13604813.2015.1071113
5. Dzwierzynska J, Prokopska A. Urban planning by Le Corbusier according to praxeological knowledge. *IOP Conference Series: Earth and Environmental Science*, 2017; 95: 052007. doi: 10.1088/1755-1315/95/5/052007
6. Urban F. Postmodernism and socialist mass housing in Poland. *Planning Perspectives*, 2019; 35(1): 27–60. doi: 10.1080/02665433.2019.1672208
7. Fentahun G. Foreign aid in the post-colonial Africa: Means for building democracy or ensuring Western domination? *Cogent Social Sciences*, 2023; 9(1): 2241257. doi: 10.1080/23311886.2023.2241257
8. Appau PK, Asibey MO, Grant R. Enabling asset-based community development solutions: Pro-poor urban climate resilience in Kumasi, Ghana. *Cities*, 2024; 145, 104723. doi: 10.1016/j.cities.2023.104723
9. Nel H. Asset-based community-driven development (ABCD) as an approach and model to address the challenges of Africa. *Community Development Practice in Africa: Putting Theory into Practice*, 2024; 67–83. doi: 10.1108/979-8-88730-720-620251008

10. Haddock M, Devereux P. Measuring the contribution of volunteering to the sustainable development goals: Challenges and opportunities. *Voluntaris*, 2016; 4(1): 68–100. doi: 10.5771/2196-3886-2016-1-68
11. Binder CC. Redistribution and the individualism–collectivism dimension of culture. *Social Indicators Research*, 2019; 142, 1175–1192. doi: 10.1007/s11205-018-1964-6
12. Parsell C, Sharma N, Kuskoff E. Ending homelessness through permanent supportive housing: A cost offset update. Life Course Centre, Australia. 2023. doi: 10.14264/31b3388
13. Romaniuk JR. Leczenie uzależnień. Materiały na zamówienie. Klinika Leczenia Uzależnień „Odwrócenie”, Węgorzewo 2024
14. Zhao E. The key factors contributing to the persistence of homelessness. *International Int J Sustain Dev World Ecol*, 2023; 30(1): 1–5. doi: 10.1080/13504509.2022.2120109
15. Speak S, Tipple G. Perceptions, persecution and pity: The limitations of interventions for homelessness in developing countries. *Int J Urban Reg Res*, 2006; 30(1): 172–188. doi: 10.1111/j.1468-2427.2006.00647.x
16. Weisenfeld PE. Successes and challenges of the Haiti earthquake response: The experience of USAID. *Emory Int Law Rev*, 2011; 25(3): 1097–1116
17. Cieśla A. Local housing strategies in Poland as a tool to develop social and affordable housing: barriers for development. *Res Pap Econ Finance*, 2023; 7(2): 81–93. doi: 10.18559/ref.2023.2.6
18. Sygulska K. Wybrane obszary problemu bezdomności w Polsce – przegląd literatury. *Praca Socjalna*, 2023; 38(2): 127–148. doi: 10.5604/01.3001.0053.684519
19. Romaniuk JR, Mamzer H, Farkas KJ. The transformation of social work in Ukraine before and during the war. *Society Register*, 2024; 8(1): 7–26. doi: 10.14746/sr.2024.8.1.01
20. Wygnańska J. Person first, fidelity second. *Eur J Homelessness*, 2020; 14(2): 301–312
21. Farkas KJ, Romaniuk JR, Baranowski M. Beyond housing first: Rethinking neoliberal policies impacting homelessness. *J Public Health Policy*, 2024; doi: 10.1057/s41271-024-00537-7
22. Ren X. Slums, Favelas, and Urban Villages. *Curr Hist*, 2020; 119(813): 15–21
23. Odo OM, Onyishi IE, Onah IL. A psychological analysis of the impact of the drift from collectivism value orientation to individualism in Africa. *Pract Psychol*, 2022, 12(1). <https://journals.aphriapub.com/index.php/PP/article/view/2059>
24. Santos LB. Nature-based solutions in urban periphery communities in Brazil. *Societal Impacts*, 2024; 3: 100050. doi: 10.1016/j.socimp.2024.100050
25. Karabanow J. Creating a culture of hope: Lessons from street children agencies in Canada and Guatemala. *Int Soc Work*, 2003; 46(3): 369–386
26. Oldfield J, Stevenson A, Ortiz E. Promoting resilience in street connected young people in Guatemala: The role of psychological and educational protective factors. *J Community Psychol*, 2020; 48(2): 590–604
27. Ghosh, S. Understanding homelessness in neoliberal city: A study from Delhi. *J Asian Afr Stud*, 2020; 55(2): 285–297. doi: 10.1177/0021909619875775
28. Banerjee P. Abandon the slum? Toward an alternative recognition of urban informal dwelling. *J Urban Hist*, 2023; 49(3): 600–614. doi: 10.1177/00961442221127311
29. Harrison ME, Sharma M. Urbanisation and urban villages: An overview of slum communities in India. In: Chatterjee U, Bandyopadhyay N, Setiawati MD, Sarkar S, eds. *Urban Commons, Future Smart Cities and Sustainability*. Springer Geography. Cham: Springer; 2023; p. 89–106. doi: 10.1007/978-3-031-24767-5_5
30. Asiamah P. Slums development in Ghana, debates on eradication and upgrading: A necessary highlight on Abinkyi. *J Polit Sci Int Relat*, 2023; 6(1): 1–8. doi: 10.11648/j.jpsir.20230601.11
31. Jones BG. 'Bankable Slums': the global politics of slum upgrading. *Third World Q*, 2012; 33(5): 769–789. doi: 10.1080/01436597.2012.679027
32. Liera C, Dickin S, Rishworth A, et al. Human Rights, COVID-19, and barriers to safe water and sanitation among people experiencing homelessness in Mexico City. *Front Water*, 2023; 5: 1054182. doi: 10.3389/frwa.2023.1054182
33. Sin HL. Volunteer tourism – “Involve me and I will learn”? *Ann Tour Res*, 2009; 36(3): 480–501. doi: 10.1016/j.annals.2009.03.001
34. Allum C. Insights on international volunteering: Perspectives from the Global South. *Can J Dev Stud*, 2021; 43(2): 298–300. doi: 10.1080/02255189.2021.1912717
35. Chupp M, Hirsch J, Malone M. Introduction: Integrating asset-based community development and community-based research for social change: A beginning. *Gateways Int J Community Res Engag*, 2023; 16(2): 1–12. doi: 10.5130/ijcre.v16i2.8968



PHARMACOLOGICAL STRATEGIES FOR REDUCING METABOLIC COMPLICATIONS INDUCED BY SECOND-GENERATION ANTIPSYCHOTICS IN PATIENTS WITH SCHIZOPHRENIA

Farmakologiczne strategie redukcji powikłań metabolicznych wywołanych przez leki przeciwpsychotyczne drugiej generacji u pacjentów ze schizofrenią



Hanna Weronika Wołodkiewicz, Weronika Caban

Medical University of Warsaw, Poland

Hanna Weronika Wołodkiewicz –  0009-0000-5166-6855

Weronika Caban –  0009-0002-1530-4392

Abstract

The aim of this paper was to review the current literature on pharmacological treatments for metabolic complications induced by second-generation antipsychotics in patients with schizophrenia and to compare their efficacy. Data from meta-analyses, randomized studies, cohort studies and systematic reviews were analysed, focusing on the use of metformin, topiramate, GLP-1 agonists, SGLT2 inhibitors, opioid receptor antagonists, bupropion, and aripiprazole. The largest body of evidence for efficacy was found for metformin, especially in patients after the first episode of the disease. Topiramate and bupropion also cause weight loss, although their use may be associated with some adverse effects. GLP-1 agonists have a beneficial effect on body weight and metabolic parameters, but require continuous therapy. SGLT2 inhibitors and aripiprazole improve the metabolic profile, but clinical data remain limited. Samidorfan is effective only in combination with olanzapine. Pharmacological strategies to reduce metabolic complications due to second-generation antipsychotics are promising but require further long-term studies and evaluation of combination therapy. Personalized treatment and close patient monitoring remain crucial to improve the safety and efficacy of antipsychotic therapy.

Streszczenie

Celem pracy jest przegląd najnowszej literatury dotyczącej farmakologicznych metod leczenia powikłań metabolicznych indukowanych przez leki przeciwpsychotyczne drugiej generacji u pacjentów ze schizofrenią oraz porównanie ich skuteczności. Analizie poddano dane pochodzące z metaanaliz, badań randomizowanych, kohortowych oraz przeglądów systematycznych, koncentrujące się na stosowaniu: metforminy, topiramatu, agonistów GLP-1, inhibitorów SGLT2, antagonistów receptorów opioidowych, bupropionu oraz aripiprazolu. Najwięcej dowodów potwierdzających skuteczność dotyczy metforminy, szczególnie u pacjentów po pierwszym epizodzie choroby. Topiramat oraz bupropion również powodują redukcję masy ciała, choć ich stosowanie może wiązać się z działaniami niepożądanymi. Agoniści GLP-1 wpływają korzystnie na masę ciała i parametry metaboliczne, jednak wymagają ciągłej terapii. Inhibitory SGLT2 oraz aripiprazol poprawiają profil metaboliczny, lecz liczba danych klinicznych jest ograniczona. Samidorfan wykazuje skuteczność jedynie w połączeniu z olanzapiną. Farmakologiczne strategie redukcji powikłań metabolicznych wywołanych przez leki przeciwpsychotyczne drugiej generacji są obiecujące, lecz wymagają dalszych badań długoterminowych oraz oceny terapii skojarzonej. Personalizacja leczenia i monitorowanie pacjentów pozostają kluczowe dla poprawy bezpieczeństwa i skuteczności terapii przeciwpsychotycznej.

Keywords: schizophrenia; antipsychotic drugs; metformin; aripiprazole; GLP-1

Słowa kluczowe: schizofrenia; leki przeciwpsychotyczne; metformina, aripiprazol; GLP-1

DOI 10.53301/lw/205115

Received: 09.04.2025

Accepted: 14.05.2025

Published: 31.03.2026

Corresponding author:

Hanna Weronika Wołodkiewicz
Medical University of Warsaw
e-mail: hwołodkiewicz@gmail.com

Introduction

Schizophrenia is a severe, chronic mental disorder that often requires long-term or lifelong pharmacotherapy. Second-generation antipsychotics (SGAs), which effectively alleviate psychotic symptoms and reduce the risk of relapse, are currently the cornerstone of therapy. However, these treatments have been linked to a substantial risk of adverse reactions, with metabolic complications such as weight gain, insulin resistance, dyslipidaemia, and elevated risk of type 2 diabetes mellitus (T2DM) having major clinical significance [1,2]. These disturbances markedly increase cardiovascular (CV) morbidity in schizophrenic patients, which is up to twofold higher compared to the general population, and contribute to reduced life expectancy [4]. Additionally, antipsychotic-induced weight gain adversely affects patients' quality of life and may negatively influence treatment adherence, potentially leading to premature discontinuation [5, 6].

The risk of metabolic complications depends on both the pharmacological profile of the antipsychotic and individual patient factors. Among drug-related aspects, receptor-binding affinity (particularly for histamine H₁, serotonergic 5-HT_{2C}, and dopaminergic D₂ receptors) is particularly critical, as these play a key role in appetite regulation, energy balance, and metabolic homeostasis [7].

It is crucial to emphasize that the metabolic adverse effects of antipsychotics differ qualitatively from typical obesity, exhibiting a more rapid onset, profounder impact on glucose metabolism, and distinct hormonal and neuroendocrine alterations induced by these agents [8]. Furthermore, schizophrenic patients may be more susceptible to antipsychotic-associated weight gain than individuals with other psychiatric diagnoses [9].

Due to the significant clinical risk, regular monitoring of metabolic parameters is recommended in individuals taking antipsychotics, with particular attention paid to individual risk factors [10]. If significant metabolic adverse effects occur, dose reduction or switching to a drug with a lower metabolic risk, such as aripiprazole, lurasidone, or ziprasidone, may be considered [9]. Although such a therapeutic strategy may benefit some patients [11], it carries a risk of poor psychotic symptom control and does not consistently yield clinically meaningful weight loss [12]. Non-pharmacological interventions, including dietary education and promotion of regular physical activity, are vital for both preventing and managing metabolic complications [8]. However, such interventions often produce only transient effects and may prove inadequate in cases of substantial antipsychotic-induced weight gain [13].

Despite the availability of multiple therapeutic strategies, the optimal management of metabolic adverse effects associated with antipsychotic therapy remains a major clinical challenge.

Aim

This paper summarises the current knowledge on pharmacological treatments for antipsychotic-induced me-

tabolic complications in schizophrenic patients, while comparing their efficacy.

Results

Metformin

Metformin is an antihyperglycaemic agent that inhibits gluconeogenesis and lipid synthesis, leading to improved blood glucose control and reducing triglyceride and low-density lipoprotein (LDL) cholesterol. Its appetite-suppressing effect may be related to increased production of glucagon-like peptide-1 (GLP-1), which promotes satiety, stimulates insulin release, and inhibits glucagon secretion. Metformin's multifaceted actions also reduce glucose absorption from the gastrointestinal tract [15]. These mechanisms reduce insulin resistance and enhance peripheral glucose metabolism, thereby promoting weight loss [14]. Its efficacy in reducing body weight in SGA-treated patients has been extensively investigated and confirmed in many meta-analyses and cohort studies across diverse patient populations, establishing it as a primary adjunctive agent for mitigating antipsychotic metabolic adverse effects [14]. In their meta-analysis of 12 randomised, double-blind, controlled trials, De Silva et al. showed that metformin yielded a mean weight loss of 3.27 kg and a BMI reduction of 1.13 kg/m² ($p < 0.001$) in 743 patients. It additionally induced a mean reduction in the insulin resistance index (IRI) of 1.49 ($p < 0.001$). Metformin was effective in both paediatric and adult populations; however, greater efficacy was observed in first-episode patients (-5.94 kg) compared with those receiving chronic treatment (-2.06 kg) [16]. The association between weight loss and gender varies across studies (Lee et al.; Hakami et al.). However, metformin was more frequently combined with antipsychotic treatment in women, possibly due to the higher prevalence of coexisting indications, such as polycystic ovary syndrome (PCOS), which is often linked to obesity. Metformin has also proven effective in patients on clozapine. Since clozapine reduces GLP-1 levels by disrupting its pathway in the small intestinal epithelium, metformin may be particularly beneficial in these patients. In their meta-analysis of 487 patients on clozapine, Siskind et al. demonstrated reductions in body weight (3.12 kg) and BMI (1.18 kg/m²) with metformin versus placebo. Additionally, patients benefited from improvements in fasting glucose, triglycerides, and waist circumference, which are key components of metabolic syndrome [15]. These studies confirm metformin as an appropriate treatment for weight loss in patients on SGAs, with the added benefit of alleviating metabolic syndrome and thereby reducing CV risk [16].

Topiramate

Topiramate promotes satiety and suppresses appetite, likely by inhibiting carbonic anhydrase; it also reduces lipogenesis and boosts lipoprotein lipase activity [17]. Nickel et al. conducted a 10-week study in women treated with olanzapine. The study group receiving topiramate 250 mg/day showed an average weight loss of

4.4 kg compared with 1.2 kg weight gain in controls. Similar results were reported by Afshar et al. for patients receiving clozapine. The dose-dependent weight loss was highlighted by Ko et al., who stratified patients into two study groups: topiramate 100 mg/day or 200 mg/day, and a control group. Weight loss of 1.68 kg, 5.35 kg, and 0.3 kg was observed across the groups, respectively. Patients receiving topiramate 200 mg/day were more likely to achieve $\geq 5\%$ weight loss. Paraesthesia, with dose-dependent incidence of 60% in the 200 mg/day group vs 25% in the 100 mg/day group, was the most common adverse reaction. Paraesthesia also occurred in the placebo group, but at a significantly lower rate (10%). Additionally, worse control of schizophrenic symptoms was observed in the study groups compared to the placebo group, although this effect has not been reported elsewhere. Therefore, when prescribing topiramate to patients on SGAs, potential adverse effects should be considered, particularly at higher doses, and the symptoms of the underlying disease should be closely monitored. Nevertheless, topiramate still shows promising effects in terms of weight management [14, 17].

GLP-1 receptor agonists

GLP-1, an incretin hormone secreted by intestinal L cells in response to nutrients, stimulates insulin release, inhibits glucagon secretion, and lowers blood glucose levels. GLP-1 receptor agonists (GLP-1RAs) slow gastric emptying, thereby prolonging satiety and reducing appetite, while their glucose-dependent mechanism minimises the risk of hypoglycaemia. GLP-1RAs are commonly used to treat T2DM and often promote weight loss. Studies in patients receiving SGAs have also confirmed this effect [18, 19]. Subcutaneous liraglutide or exenatide treatment resulted in mean weight reductions of 3–5.3 kg. Exenatide proved particularly beneficial for patients on olanzapine and/or clozapine, yielding a mean weight loss of 5.29 kg, significantly greater than in those receiving other antipsychotics [18]. GLP-1 receptor agonists also exert multifaceted benefits on lipid metabolism, fasting glucose, glycated haemoglobin, and waist circumference, which are key components of metabolic syndrome and CV risk factors. However, 12 months after treatment discontinuation, patients returned to baseline metabolic parameters, while partial weight loss achieved with liraglutide was maintained [19]. Gastrointestinal (GI) symptoms, including nausea, vomiting, and diarrhoea were the most common adverse effects; however, these were mild, and the dropout rate was similar in both the study and control group. Potential effects of GLP-1RAs on the risk of suicidal behaviours have also been ruled out. Semaglutide offers greater convenience with once-weekly dosing; however, its use for this indication is still under investigation [19].

SGLT2

Sodium–glucose cotransporter 2 (SGLT2) inhibitors represent another class of medications that may improve body weight in SGA-treated patients. These agents act by inhibiting glucose reabsorption in the proximal renal tubules, leading to glucosuria and a consequent negative caloric balance. Due to their

capacity to reduce blood glucose and glycated haemoglobin (HbA1c), as well as their low risk of hypoglycaemia, T2DM is the primary indication for this class of drugs. In clinical trials, SGLT2 inhibitors induced an average weight loss of 1–3 kg. SGLT2 inhibitors also mitigate CV risk by lowering blood pressure via natriuresis. Additionally, they provide renal benefits by reducing intraglomerular pressure and hyperfiltration. Ertugliflozin is characterized by a favourable safety profile comparable to placebo, while significantly reducing metabolic syndrome components, including body weight. SGLT2 inhibitors are routinely used as second-line therapy alongside metformin, showing clear benefits for weight reduction [20]. However, clinical data regarding their use in patients treated with clozapine or olanzapine remain limited.

Opioid receptor antagonists

Endogenous opioids interact with three main receptor types, i.e. mu (MOR), delta (DOR), and kappa (KOR), which regulate appetite, satiety, energy expenditure, and overall metabolic functions. Given the involvement of the opioid system in these processes, antagonists targeting these receptors could mitigate antipsychotic-induced metabolic disturbances. Three substances are being investigated for this indication: naltrexone, samidorphan, and LY255582. Naltrexone is mainly indicated for alcohol use disorder and opioid dependence, whereas its combination with bupropion is approved for long-term management of overweight and obesity. Preclinical and clinical studies on the effect of naltrexone on weight loss when used concomitantly with antipsychotics are inconclusive. In animal studies, it demonstrated weight-lowering effects despite having no effect on food intake [21]. In a pilot study involving 24 women with schizoaffective disorder or schizophrenia, naltrexone treatment led to weight loss after 8 weeks, but only in those without T2DM [22]. Another study involving patients receiving olanzapine for the same indications showed no weight loss after 12 weeks, but a reduction in fat mass was observed in the naltrexone group [23].

Samidorphan, used in conjunction with olanzapine, uniquely promotes weight loss in this combination while counteracting metabolic adverse effects of olanzapine. In preclinical studies, it consistently reduced olanzapine-associated metabolic adverse effects. The drug primarily acts by reducing body weight and fat mass, with studies also showing appetite suppression. Clinical trials have confirmed these conclusions, additionally demonstrating reduced waist circumference and a positive impact on cholesterol and glucose metabolism. The combination of samidorphan and olanzapine, under the trade name Lybalvi, was approved by the FDA in 2021 for bipolar disorder [21].

In preclinical trials, LY255582 reduced fat mass, total body weight, and appetite. These metabolic effects are based on improved glucose tolerance and lipid profile, which may be related to changes in carbohydrate metabolism. Despite promising results, further studies are needed to confirm the clinical efficacy of LY255582. Differences in effects among individual opioid recep-

tor antagonists likely arise from their varying affinities for specific receptor subtypes, resulting in distinct levels of appetite modulation and impacts on lipid metabolism. Further comparative studies of these substances will help determine their efficacy and optimize therapeutic strategies [21].

Bupropion

Bupropion is a norepinephrine–dopamine reuptake inhibitor with minimal serotonergic activity. It is widely used as an antidepressant and has also been employed as a weight-reducing agent, either as monotherapy or in combination with naltrexone. Its role in supporting smoking cessation is an additional clinically relevant benefit in psychiatric populations. A randomized, double-blind study evaluating the effect of bupropion on body weight included 26 patients receiving SGAs. The study group received bupropion at 150–300 mg/day combined with olanzapine (10–20 mg/day) or risperidone (2–4 mg/day). At treatment week 8, a statistically significant reduction in body weight was seen in the bupropion group, without comparable effect in the placebo group. Given bupropion's dopaminergic and noradrenergic action, psychotic manifestations were systematically monitored post-intervention, with no aggravation in the bupropion group [24].

In a prospective, open-label trial, standard-dose bupropion was given to 8 patients on olanzapine for at least 26 months, with a mean weight gain of 13.3 kg. At 24 weeks, patients achieved a mean weight loss of 3.4 kg, accompanied by improved lipid profiles [25]. Bupropion is also combined with naltrexone, which is also used to treat obesity in the general population. Reports on its efficacy for weight loss in psychiatric patients using SGAs remain ambiguous. Tham et al. demonstrated a significant body weight reduction of 10.9% relative to baseline ($p < 0.001$), accompanied by a decrease in waist circumference at 52 weeks of treatment. However, these findings lack consistent replication, as many other studies failed to demonstrate comparable effects [26]. Although the cited studies showed no significant rise in adverse reactions with bupropion therapy, isolated seizures have been reported in patients concomitantly taking olanzapine and bupropion, both known to lower the seizure threshold. Olanzapine carries an estimated cumulative seizure risk of about 10% after 3.8 years of treatment compared to only 0.4% for bupropion at standard therapeutic doses. To date, there is insufficient evidence to reliably assess the cumulative or synergistic seizure risk associated with the combined use of these two medications. However, caution is warranted in patients with seizure history or other predisposing risk factors; alternative weight-loss pharmacotherapies should be considered in these cases. Although available data on bupropion appear promising, further studies in broader patient populations are needed to confirm its efficacy and better estimate the risk of seizures [27].

Aripiprazole

Aripiprazole acts as a partial agonist at dopamine D₂ and serotonin 5-HT_{1A} receptors and an antagonist at

serotonin 5-HT_{2A} and 5-HT_{2C} receptors. Unlike other SGAs, aripiprazole is not associated with weight gain and may even promote weight loss. In one study, adjunctive treatment with aripiprazole in patients receiving clozapine resulted in a mean weight reduction of 5.1 kg after 34 weeks of therapy. A similar metabolic effect was observed in patients whose primary antipsychotic was olanzapine. However, adjunctive aripiprazole therapy was associated with significant adverse reactions in some patients, including sinus tachycardia, worsened psychotic symptoms, and new-onset auditory hallucinations. These complications were more common in patients receiving combined aripiprazole/clozapine treatment. These adverse effects likely stem from enhanced dopaminergic neurotransmission and increased sympathetic activation due to aripiprazole's partial agonism at D₂ and 5-HT_{1A} receptors, coupled with its antagonism at 5-HT_{2A/2C} receptors. Additional symptoms (nausea, increased anxiety, and ataxia) also occurred more frequently in patients receiving adjunctive aripiprazole. Other than that, no significant differences in Positive and Negative Syndrome Scale (PANSS) scores emerged between the study and control group [28]. In addition to reducing body weight, aripiprazole improves lipid profiles, significantly lowering total cholesterol, LDL cholesterol, and triglycerides while raising HDL cholesterol. Aripiprazole plus olanzapine/clozapine enhances metabolic parameters and mitigates CV risk in these patients [29]. A meta-analysis of 55 randomized controlled trials ($n=4,457$ patients) showed significant mean weight loss of 5.08 kg ($p < 0.00001$) and BMI reduction, without worsening psychotic symptoms. However, the authors stressed the need to validate these findings through higher-quality studies registered in official clinical trial databases [30].

Discussion

Weight gain and metabolic disturbances from SGAs significantly elevate CVD risk and impair patients' quality of life. A 2023 meta-analysis [9] recommends prioritizing lifestyle modifications, supplemented by pharmacological options like metformin, topiramate, or bupropion, to counteract metabolic disturbances from these treatments. If these measures prove ineffective, SGA switching should be considered. The studies presented in this paper assessed the efficacy of both the above-mentioned and other therapies investigated for this indication.

Many studies, including meta-analyses, confirm the efficacy of metformin in mitigating weight gain among SGA-treated patients, especially those in early disease stages. Therefore, metformin constitutes a reasonable primary treatment option for this patient group; however, further long-term studies are needed to assess whether these therapeutic effects persist over time. This may be a particularly suitable option for diabetic patients, given metformin's established effects in reducing insulin resistance, fasting serum glucose and triglyceride levels, and waist circumference. Nevertheless, more comprehensive studies are needed in this patient population.

Topiramate, an antiepileptic agent, also exhibits weight-reducing properties; however, its use is associated with adverse effects such as paraesthesia, hypersalivation, and psychomotor slowing, particularly at higher doses. Due to reports of potential exacerbation of schizophrenia symptoms, its use warrants particular caution and close monitoring of the patient's clinical status.

GLP-1 receptor agonists effectively reduce body weight and, importantly, exert beneficial effects on metabolic syndrome manifestations. This is reflected by improved lipid metabolism, lower fasting serum glucose, reduced glycated haemoglobin levels, and reduced waist circumference. The need for daily subcutaneous injections is a major limitation of liraglutide therapy. Furthermore, the return to baseline metabolic parameters 12 months after treatment cessation highlights the need for continuous administration to maintain therapeutic outcomes. This class of medications is predominantly associated with GI adverse effects, which are generally mild.

A major advantage of SGLT2 inhibitors is their reduced CV risk and beneficial effects on the kidneys. Clinical trials have demonstrated their efficacy in the general population; however, data regarding patients receiving clozapine or olanzapine remain limited. The available data indicate that this drug class warrants consideration in patients with elevated CV risk.

Although opioid receptor antagonists have shown potential anti-weight gain effects, research in this area is inconclusive. Samidorfan has proven efficacy only in combination with olanzapine and was approved by the FDA in 2021. Studies assessing other members of this group (e.g., LY255582, currently in preclinical development), which may expand therapeutic options, are also underway.

Bupropion also supports weight loss, especially when combined with naltrexone. However, research on its efficacy in patients taking SGAs is inconclusive. Although bupropion does not compromise psychotic symptom control, the concomitant reduction in seizure threshold with olanzapine raises safety concerns. Special attention should be paid to aripiprazole, which, unlike most other SGAs, does not cause weight gain and may, in some cases, lead to weight loss. Studies have shown that adding aripiprazole to clozapine or olanzapine can result in significant weight loss and improved lipid profiles. Aripiprazole monotherapy may also be considered. From a practical standpoint, systematic monitoring strategies for patients on antipsychotics are essential, alongside education for psychiatrists regarding the metabolic sequelae of these agents and strategies for their mitigation. This necessitates the development of clear guidelines and protocols for managing these complications. Incorporating pharmacological preventive strategies into routine treatment protocols could enhance the overall health of psychiatric patients, mitigate their CV risk, and lower the risk of treatment discontinuation.

The cost-benefit balance of pharmacotherapy for complications of antipsychotics is another important aspect to consider. Increasing the number of medications taken may contribute to poor adherence. Additional medications may cause other adverse reactions and promote polypharmacy. Therefore, it is crucial to identify situations when antipsychotic therapy is essential and treatment-related complications persist, posing sustained harm to the patient.

However, this review should be interpreted in light of several important limitations. First, most studies are short-term, making it difficult to assess the long-term efficacy and safety of the analysed therapies. Consequently, the observed weight loss effects may prove transient, with their persistence following treatment cessation remaining uncertain. In particular, data on the persistence of favourable metabolic outcomes after discontinuation of pharmacotherapies such as metformin, GLP-1 receptor agonists, or SGLT2 inhibitors are missing. Second, there is a lack of analyses assessing the efficacy of combination approaches, such as metformin and GLP-1 agonists, which could potentially improve treatment outcomes. Furthermore, the methodological heterogeneity across studies (different patient populations, drug dosages, and follow-up durations) precludes direct comparisons of results. Third, the mechanisms underlying individual treatment responses remain unclear. Many studies fail to account for the key factors influencing treatment efficacy, such as comorbidities (e.g., insulin resistance, polycystic ovary syndrome) or prior antipsychotic exposure. Metformin appears to be particularly effective in first-episode patients; however, the mechanisms underlying this observation have not yet been fully elucidated. Fourth, several pharmacological agents discussed are associated with clinically relevant adverse effects despite their proven efficacy. For instance, topiramate may induce psychomotor slowing and exacerbate psychotic symptoms despite its efficacy in inducing weight loss. GLP-1 receptor agonists may cause GI adverse effects, whereas the combination of bupropion and olanzapine may elevate the risk of reduced seizure threshold. Furthermore, robust data on the long-term safety of some interventions in psychiatric populations remain scarce.

Although existing evidence supports the efficacy of several pharmacological strategies for weight reduction in patients with psychiatric disorders, further long-term, comparative studies are needed to comprehensively delineate their efficacy, safety profiles, and potential drug-drug interactions.

In summary, current pharmacological interventions offer promising approaches for reducing weight gain induced by SGAs; however, the evidence often remains limited and reflects short-term outcomes. Future research should therefore prioritize evaluating long-term efficacy and safety, alongside developing personalised treatment strategies tailored to patients' individual clinical profiles.

References

1. De Hert M, Detraux J, van Winkel R, Yu W, Correll CU. Metabolic and cardiovascular adverse effects associated with antipsychotic drugs. *Nat Rev Endocrinol*, 2011; 8(2): 114–126. doi: 10.1038/nrendo.2011.156. PMID: 22009159
2. De Hert M, Dekker JM, Wood D, et al. Cardiovascular disease and diabetes in people with severe mental illness position statement from the European Psychiatric Association (EPA), supported by the European Association for the Study of Diabetes (EASD) and the European Society of Cardiology (ESC). *Eur Psychiatry*, 2009; 24(6): 412–424. doi: 10.1016/j.eurpsy.2009.01.005
3. Ratliff JC, Palmese LB, Reutenauer EL, et al. Obese schizophrenia spectrum patients have significantly higher 10-year general cardiovascular risk and vascular ages than obese individuals without severe mental illness. *Psychosomatics*, 2013; 54(1): 67–73. doi: 10.1016/j.psych.2012.03.001
4. Roberts LW, Louie AK, Guerrero APS, et al. Premature mortality among people with mental illness: advocacy in academic psychiatry. *Acad Psychiatry*, 2017 Aug; 41(4): 441–446. doi: 10.1007/s40596-017-0738-9
5. Speyer H, Westergaard C, Albert N, et al. Reversibility of antipsychotic-induced weight gain: a systematic review and meta-analysis. *Front Endocrinol (Lausanne)*, 2021; 12: 577919. doi: 10.3389/fendo.2021.577919
6. Weiden PJ, Mackell JA, McDonnell DD. Obesity as a risk factor for antipsychotic noncompliance. *Schizophr Res*, 2004; 66(1): 51–57. doi: 10.1016/s0920-9964(02)00498-x.
7. Reynolds GP, Kirk SL. Metabolic side effects of antipsychotic drug treatment—pharmacological mechanisms. *Pharmacol Ther*, 2010; 125(1): 169–179. doi: 10.1016/j.pharmthera.2009.10.010
8. Manu P, Dima L, Shulman M, et al. Weight gain and obesity in schizophrenia: epidemiology, pathobiology, and management. *Acta Psychiatr Scand*, 2015; 132(2): 97–108. doi: 10.1111/acps.12445
9. Campforts B, Drukker M, Crins J, et al. Association between antipsychotic medication and clinically relevant weight change: meta-analysis. *BJPsych Open*, 2023; 9(1): e18. doi: 10.1192/bjo.2022.619
10. Tschoner A, Engl J, Laimer M, et al. Metabolic side effects of antipsychotic medication. *Int J Clin Pract*, 2007; 61(8): 1356–1370. doi: 10.1111/j.1742-1241.2007.01416.x
11. Newcomer JW, Weiden PJ, Buchanan RW. Switching antipsychotic medications to reduce adverse event burden in schizophrenia: establishing evidence-based practice. *J Clin Psychiatry*, 2013; 74(11): 1108–1120. doi: 10.4088/JCP.12028ah1
12. Siskind D, Gallagher E, Winckel K, et al. Does switching antipsychotics ameliorate weight gain in patients with severe mental illness? A systematic review and meta-analysis. *Schizophr Bull*, 2021; 47(4): 948–958. doi: 10.1093/schbul/sbaa191
13. Hall KD, Kahan S. Maintenance of lost weight and long-term management of obesity. *Med Clin North Am*, 2018; 102(1): 183–197. doi: 10.1016/j.mcna.2017.08.012
14. Fiedorowicz JG, Miller DD, Bishop JR, et al. Systematic Review and meta-analysis of pharmacological interventions for weight gain from antipsychotics and mood stabilizers. *Curr Psychiatry Rev*, 2012; 8(1): 25–36. doi: 10.2174/157340012798994867
15. Siskind DJ, Leung J, Russell AW, et al. Metformin for clozapine associated obesity: a systematic review and meta-analysis. *PLoS One*, 2016; 11(6): e0156208. doi: 10.1371/journal.pone.0156208
16. de Silva VA, Suraweera C, Ratnatunga SS, et al. Metformin in prevention and treatment of antipsychotic induced weight gain: a systematic review and meta-analysis. *BMC Psychiatry*, 2016; 16: 341. doi: 10.1186/s12888-016-1049-5.
17. Ellinger LK, Ipema HJ, Stachnik JM. Efficacy of metformin and topiramate in prevention and treatment of second-generation antipsychotic-induced weight gain. *Ann Pharmacother*, 2010; 44(4): 668–679. doi: 10.1345/aph.1M550
18. Menon T, Lee S, Gong XY, et al. A systematic review on the efficacy of GLP-1 receptor agonists in mitigating psychotropic drug-related weight gain. *CNS Spectr*, 2024; 1–7. doi: 10.1017/S1092852924000531
19. Vasiliu O. Therapeutic management of atypical antipsychotic related metabolic dysfunctions using GLP 1 receptor agonists: A systematic review. *Exp Ther Med*, 2023; 26(1): 355. doi: 10.3892/etm.2023.12054
20. Vasiliu O. Impact of SGLT2 inhibitors on metabolic status in patients with psychiatric disorders undergoing treatment with second generation antipsychotics (Review). *Exp Ther Med*, 2023; 25(3): 125. doi: 10.3892/etm.2023.11824
21. McIntyre RS, Citrome L, Cummings H, et al. Opioid antagonism mitigates antipsychotic-associated weight gain: focus on olanzapine. *CNS Spectr*, 2023; 28(3): 288–299. doi: 10.1017/S1092852922000116
22. Tek C, Ratliff J, Reutenauer E, et al. A randomized, double-blind, placebo-controlled pilot study of naltrexone to counteract antipsychotic-associated weight gain: proof of concept. *J Clin Psychopharmacol*, 2014; 34(5): 608–612. doi: 10.1097/JCP.0000000000000192
23. Taveira TH, Wu WC, Tschibelu E, et al. The effect of naltrexone on body fat mass in olanzapine-treated schizophrenic or schizoaffective patients: a randomized double-blind placebo-controlled pilot study. *J Psychopharmacol*, 2014; 28(4): 395–400. doi: 10.1177/0269881113509904
24. Weizman S, Shelef A, Bloemhof Bris E, Stryjer R. A double-blind, placebo-controlled trial of bupropion add-on to olanzapine or risperidone in overweight individuals with schizophrenia. *J Clin Psychopharmacol*, 2021; 41(6): 629–631. doi: 10.1097/JCP.0000000000001481
25. Gadde KM, Zhang W, Foust MS. Bupropion treatment of olanzapine-associated weight gain: an open-label, prospective trial. *J Clin Psychopharmacol*, 2006; 26(4): 409–413. doi: 10.1097/01.jcp.0000227354.54074.5d
26. Lee K, Abraham S, Cleaver R. A systematic review of licensed weight-loss medications in treating antipsychotic-induced weight gain and obesity in schizophrenia and psychosis. *Gen Hosp Psychiatry*, 2022; 78: 58–67. doi: 10.1016/j.genhosppsy.2022.07.006
27. Schmitz A, Botner B, Hund M. Bupropion with clozapine: case reports of seizure after coadministration. *J Pharm Pract*, 2021; 34(3): 497–502. doi: 10.1177/0897190020904280
28. Choi YJ. Efficacy of adjunctive treatments added to olanzapine or clozapine for weight control in patients with schizophrenia: a systematic review and meta-analysis. *Scientific World Journal*, 2015; 2015: 970730. doi: 10.1155/2015/970730

-
29. Gupta B, Chee KS, Neo LQ, et al. Effect of aripiprazole as an adjunct to atypical antipsychotics on weight and metabolic profile: a 12-week open-label trial. *Ther Adv Psychopharmacol*, 2021; 11: 20451253211046765. doi: 10.1177/20451253211046765.
30. Zheng W, Zheng YJ, Li XB, et al. Efficacy and safety of adjunctive aripiprazole in schizophrenia: meta-analysis of randomized controlled trials. *J Clin Psychopharmacol*, 2016; 36(6): 628–636. doi: 10.1097/JCP.0000000000000579



THE IMPACT OF BRUXISM ON THE HEALTH OF STRESS-EXPOSED INDIVIDUALS – SOLDIERS: DIAGNOSIS, TREATMENT, AND PREVENTION

Wpływ bruksizmu na zdrowie osób szczególnie narażonych na stres – żołnierzy: diagnostyka, leczenie i profilaktyka



Adam Łuczak, Adrianna Gliszczyńska, Konstancja Nosowicz, Patrycja Różak, Nina Szczepańska, Agata Tuczyńska

Military Specialist Medical Clinic in Poznań, Szyling Branch, Poland

Adam Łuczak – [ORCID ID 0009-0004-1848-5437](#)
 Adrianna Gliszczyńska – [ORCID ID 0009-0009-1721-5327](#)
 Konstancja Nosowicz – [ORCID ID 0009-0000-5080-1509](#)
 Patrycja Różak – [ORCID ID 0009-0004-2643-8817](#)
 Nina Szczepańska – [ORCID ID 0009-0003-7448-4466](#)
 Agata Tuczyńska – [ORCID ID 0000-0003-1648-6765](#)

Abstract

The article reviews the literature on the impact of bruxism on the health of soldiers – a professional group particularly exposed to chronic stress. The mechanisms underlying bruxism, its health consequences, including temporomandibular joint disorders, and the correlation between stress levels and the severity of bruxism are discussed. Current diagnostic methods (such as polysomnography and surface electromyography) are reviewed, along with various therapeutic strategies: pharmacological, behavioural, physiotherapeutic, and occlusal splints. The paper underscores the value of an interdisciplinary approach and prevention to mitigate the effects of bruxism in this occupational group.

Streszczenie

Artykuł przedstawia przegląd literatury dotyczącej wpływu bruksizmu na zdrowie żołnierzy – grupy zawodowej szczególnie narażonej na chroniczny stres. Omówiono mechanizmy powstawania bruksizmu, jego konsekwencje zdrowotne, w tym zaburzenia stawu skroniowo-żuchwowego, oraz zależności pomiędzy poziomem stresu a nasileniem objawów tego zaburzenia. Zaprezentowano również aktualne metody diagnostyczne (m.in. polisomnografię, elektromiografię powierzchniową) oraz różnorodne strategie terapeutyczne, w tym farmakologiczne, behawioralne, fizjoterapeutyczne oraz stosowanie szyn okluzyjnych. Podkreślono znaczenie interdyscyplinarnego podejścia i profilaktyki w minimalizowaniu skutków bruksizmu w tej grupie zawodowej.

Keywords: bruxism; dentistry; health; soldiers; stress

Słowa kluczowe: bruksizm; stomatologia; zdrowie; żołnierze; stres

DOI 10.53301/lw/206064

Received: 08.05.2025

Accepted: 09.06.2025

Published: 31.03.2026

Corresponding author:

Adam Łuczak
 Military Specialist Medical Clinic in Poznań,
 Szyling Branch
 e-mail: adamluczak2k@gmail.com

Introduction

Military service requires not only exceptional physical fitness but also considerable psychological resilience. Soldiers are routinely exposed to intense stressors, necessitating constant readiness to perform in challenging, unpredictable environments. Intensive training, combat operations, extended separations from family, and constant responsibility for one's own life and that of comrades are the primary sources of chronic stress in this occupational group.

The impact of stress on human health has been extensively investigated and documented in the scientific literature. Chronic stress disrupts the functioning of the nervous, endocrine, and immune systems, thereby contributing to the development of multiple disorders, including cardiovascular diseases (CVDs), depressive disorders, and immune dysfunction. One less apparent yet clinically significant consequence of stress is its effect on oral health. This issue holds particular significance for military personnel, who are particularly exposed to chronic psychological stress.

Stress may contribute to a wide range of oral health issues, including bruxism, periodontal disease, xerostomia, and recurrent aphthous ulcers. The mechanisms underlying these effects are multifactorial and involve endocrine responses, such as elevated cortisol levels, immune system dysregulation, reduced salivary flow, as well as changes in daily hygiene habits. Individuals experiencing stress may neglect proper oral hygiene, increase the use of harmful substances such as tobacco and alcohol, and adopt unhealthy dietary patterns to compensate for the experienced stress.

Unfortunately, neglecting oral health can have serious consequences. Periodontal disease and dental caries not only compromise aesthetics and overall quality of life, but also induce pain and functional limitations, including masticatory and phonatory difficulties. For soldiers, who are required to sustain peak physical readiness and operational efficacy across all settings, such oral health issues may give rise to substantial limitations and impaired performance.

Bruxism, defined as involuntary grinding or clenching of teeth, is a multifactorial condition in which psychological stress plays a significant aetiological role. Military personnel exposed to sustained psychophysical stress are therefore at particularly high risk of developing this disorder.

Aim

This paper discusses the impact of stress on developing bruxism, with particular emphasis on soldiers—a population exposed to chronic stress. Diagnostic and therapeutic methods are discussed. Research confirming this relationship is also presented, along with effective strategies for preventing and counteracting the negative effects of stress. The aim of this paper was to highlight the importance of oral health in the context of soldiers' operational readiness and overall performance, as well as to encourage the implementation of targeted interventions to re-

duce stress-related health consequences in this professional group.

Health issues associated with bruxism

Bruxism, particularly its chronic form, may lead to multiple health complications affecting both the stomatognathic system and overall systemic health. Persistent, uncontrolled clenching or grinding of the teeth results in overload of the masticatory muscles, which may subsequently lead to structural and functional disturbances.

In their literature review, Manfredini et al. [1] identified temporomandibular disorders (TMDs) as one of the most common complications of bruxism, affecting up to 85% of patients with chronic forms. TMD manifests clinically with mandibular pain, muscle stiffness, limited mandibular range of motion, and joint clicking upon movement.

In their systematic analysis, Melo et al. [2] demonstrated that sleep bruxists were 2.3 times more likely to develop TMDs compared with non-bruxists (OR = 2.3; 95% CI: 1.6–3.4).

Bruxism is linked to a variety of health issues, including tension-type headaches that affect up to 65% of patients with active disease [3]. Additionally, enamel wear and microcracking can increase, causing tooth sensitivity and gradual loss of dental hard tissues. Nocturnal masticatory activity may also disrupt sleep, often coexisting with conditions like obstructive sleep apnoea [3].

Sleep bruxism is the most common form and manifests as involuntary teeth clenching or grinding during sleep. It most commonly arises during transitional sleep phases, referred to as microarousals, triggered by brief central nervous system activation in response to internal or external stimuli. This activation triggers masticatory hyperactivity, leading to symptoms like headaches, tension in facial and neck muscles, tooth sensitivity, and damage to enamel and other dental hard tissues.

From a general medical viewpoint, bruxism has a negative impact on patients' quality of life through sleep disruption, compromised mental health, and reduced social functioning. The accompanied chronic pain and stress can further intensify symptoms, forming a vicious cycle between bruxism and stress.

Stress as a major risk factor

Although bruxism may stem from multiple causes, such as malocclusion and anatomical abnormalities, recent research highlights psychological stress as a primary trigger. Chronic emotional tension from everyday stressors can trigger unconscious masticatory hyperactivity during both wakefulness and sleep. Teeth grinding can thus serve as a maladaptive mechanism for releasing tension, where the body (lacking other ways to manage built-up emotional stress) triggers an involuntary motor response. Furthermore, chronic stress can impair CNS function, especially in dopaminergic pathways that regulate motor and emotional processing. Dysregulation of these systems may impair control over masticatory mus-

cle activity, thereby contributing to the development of sleep bruxism.

Sleep bruxism, manifested as involuntary teeth clenching or grinding during sleep, is one of the most prevalent forms of this disorder [4]. Studies have shown that individuals experiencing bruxism (whether awake or asleep) report markedly higher perceived stress on the Perceived Stress Scale (PSS), elevated dental anxiety as measured with the Dental Anxiety Scale (DAS), and increased gag reflex sensitivity per the Gag Assessment Scale (GAS), relative to non-bruxers [4].

The mean perceived stress level among bruxers was 14.6, which was significantly higher than that observed in the non-bruxism group (mean score of 9.3). Similarly, the severity of dental anxiety was greater among bruxers, with a mean DAS of 11.2 vs 7.5 in non-bruxers. Sensitivity to the gag reflex was also elevated among bruxers, with a mean GAS of 10.8 vs 7.4 in controls. No significant differences were found between groups in terms of the need for control, as assessed by the Desirability of Control (DC) scale, indicating that this psychological trait does not substantially contribute to bruxism [4]. Figure 1 shows perceived stress, dental anxiety, and sensitive gag reflex in bruxers vs non-bruxers.

Stress and bruxism among military personnel

In their study involving 251 Fort Hood soldiers (USA), Chen et al. found significant correlations between bruxism and three psychosocial factors: perceived stress (Perceived Stress Scale [PSS]: $r = 0.250$; $p < 0.001$), sleep quality (Pittsburgh Sleep Quality Index [PSQI]: $r = 0.325$;

$p < 0.001$), and dental anxiety (Dental Anxiety Scale [DAS]: $r = 0.144$; $p < 0.01$). Bruxers demonstrated higher mean perceived stress scores (M = 14.83 vs. 10.66), poorer sleep quality (PSQI: M = 7.02 vs. 4.29), and greater levels of dental anxiety (DAS: M = 6.65 vs. 5.82) compared with non-bruxers.

Impact on oral health

Research shows that psychological stress and associated parafunctional habits, like awake and sleep bruxism, markedly compromise stomatognathic health. Painful TMDs are one of the major consequences of bruxism.

A study involving more than 1,600 participants compared data from patients diagnosed with TMDs ($n = 733$) with those from a non-TMD control group from the general population ($n = 890$). It was found that TMD patients were significantly more likely to report symptoms of bruxism, both during wakefulness (33.9% vs. 11.2%) and sleep (49.4% vs. 23.5%), compared with healthy controls [6].

Importantly, statistical analysis revealed that both awake (OR = 1.7) and sleep bruxism (OR = 1.8) were associated with an increased risk of TM pain. The highest risk, however, was observed in individuals exhibiting both forms of bruxism concurrently, in whom the likelihood of developing TMDs increased nearly eightfold (OR = 7.7). These findings indicate that stress, serving as both trigger and aggravator of parafunctional habits, not only promotes bruxism onset, but also yields severe clinical outcomes, such as chronic pain of facial muscles and TMJ, as well as chewing dysfunctions. Thus, stress-reduction strategies

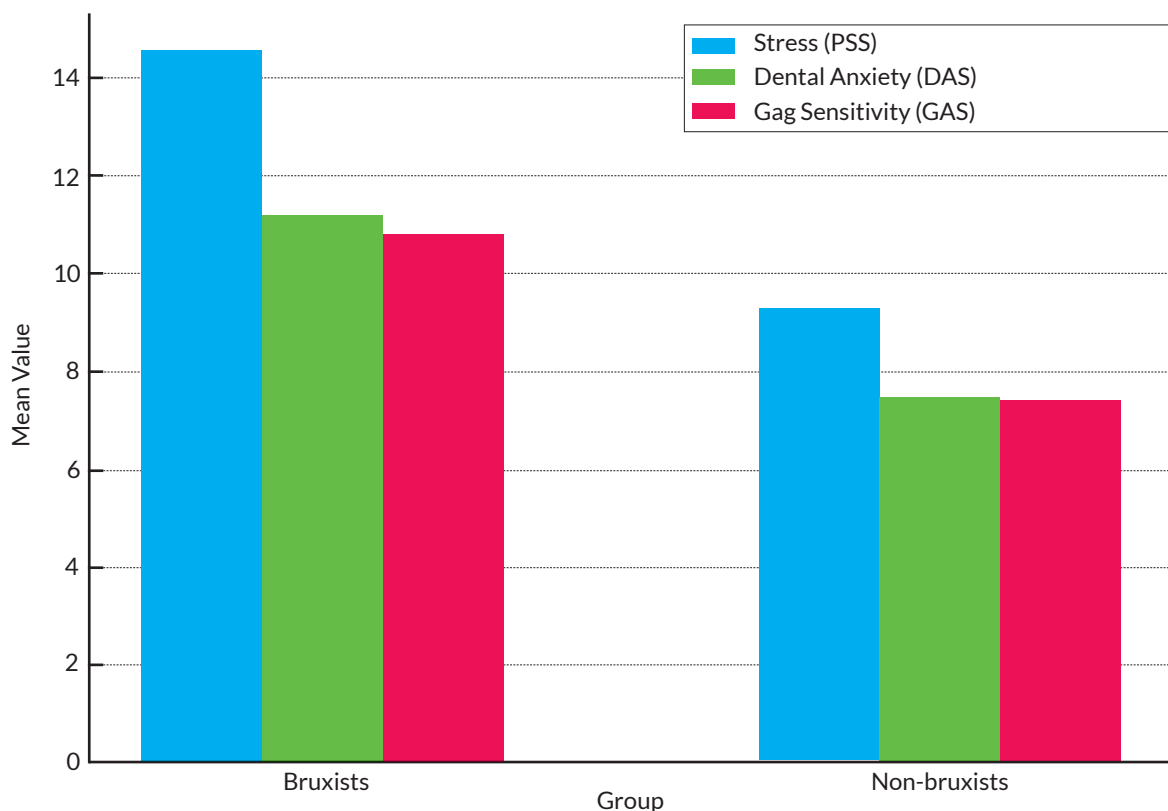


Figure 1. Comparison of variables depending on the presence of bruxism

should be included as a key element in preventing and treating bruxism and its complications [6].

Figure 2 presents a comparison of the prevalence of awake and sleep bruxism between controls and TMD patients. It may be seen that both awake and sleep bruxism are markedly more common in the latter group, reinforcing the idea that bruxism acts as either a risk factor for or outcome of stress-induced muscle hyperactivity and tension.

Diagnosis of bruxism

Precise diagnosis of sleep and awake bruxism is crucial for implementing effective therapeutic interventions. The multifactorial aetiology of this disorder, especially the prominent role of psychological stress, requires a comprehensive and differentiated diagnostic approach.

Lobbezoo et al. proposed a three-tier diagnostic framework for bruxism: “possible” (based on patient history or self-report), “probable” (history plus clinical examination), and “definite” (EMG or polysomnography). This classification system facilitates standardization of diagnostic criteria and improves diagnostic accuracy.

Polysomnography (PSG) remains one of the most precise diagnostic modalities for sleep bruxism, allowing objective measurement of masticatory muscle activity during sleep. Kato et al. [8] demonstrated that individuals with confirmed sleep bruxism experienced an average of approximately 4.1 tooth-clenching episodes per hour of sleep, with rhythmic masticatory muscle activity observed in 86% of patients.

In clinical practice, surface electromyography (sEMG) serves more commonly as a less expensive and more easily available alternative. According to Castroflorio et al. [9], EMG was able to detect patterns of masticatory activity typical of bruxism in approximately 75% of pa-

tients. The use of EMG as a diagnostic adjunct is particularly valuable in outpatient settings. In the case of awake bruxism, diagnosis is primarily based on patient self-report and clinical history taking. Manfredini et al. [3] reported that daytime teeth clenching is reported by about 20–30% of the general population; however, confirming awake bruxism requires systematic observation and often an interdisciplinary input.

Despite diagnostic advances, researchers emphasize the lack of clear, universally accepted diagnostic criteria. A systematic review by Manfredini et al. [1] points to substantial variability in sleep bruxism classification in the available literature, resulting in reported prevalence differences of up to 40%.

In summary, accurate diagnosis of bruxism requires a multimodal approach, integrating history taking, clinical examination, and specialised assessment of masticatory muscle activity. Such a comprehensive strategy may prove vital for effective management, particularly in patients with bruxism closely linked to elevated stress levels.

Pharmacotherapy

Pharmacotherapy is an adjunctive option for bruxism, especially in cases with severe symptoms and stress- or anxiety-related aetiology. Clinical trials to date have primarily targeted sleep bruxism management, using agents such as dopamine agonists, benzodiazepines, antidepressants, anxiolytics, and muscle relaxants.

In their systematic review, Lobbezoo et al. [7] evaluated the efficacy of several pharmacological agents. Clonazepam, given at 0.5–1.0 mg before bedtime, significantly reduced bruxism episode frequency in about 69% of treated patients. Buspirone, an anxiolytic, also showed beneficial effects, especially in patients with high stress levels, by decreasing the intensity of parafunctional activity.

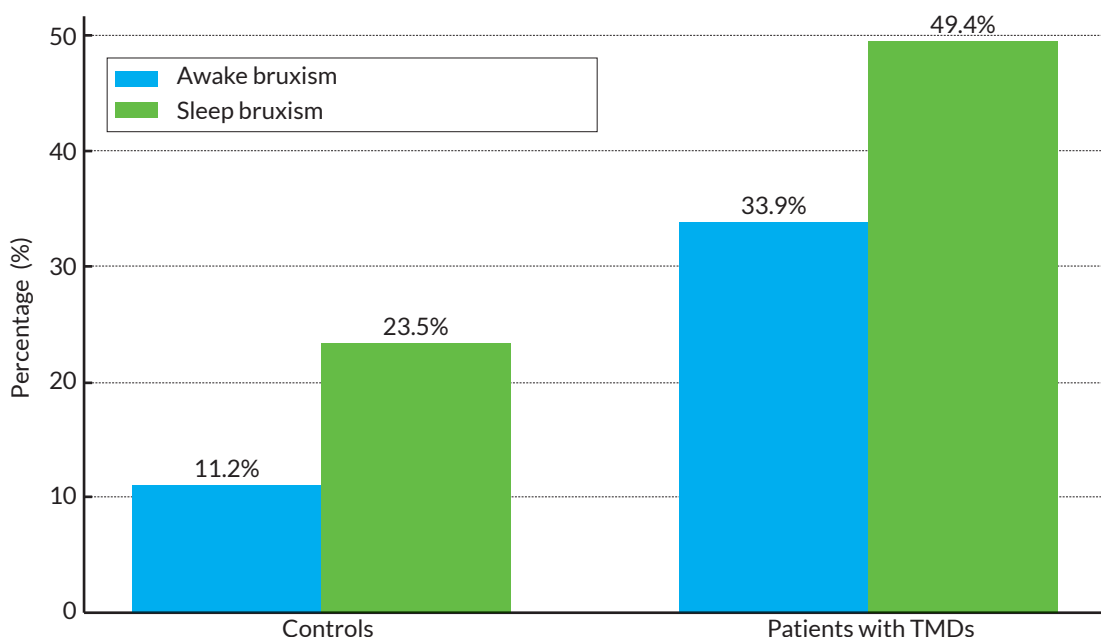


Figure 2. The prevalence of bruxism in healthy individuals and patients with temporomandibular joint disorders (TMDs)

In their review of randomized controlled trials, Macedo et al. [10] reported that dopamine agonists (including pergolide) were the most effective pharmacological agents for sleep bruxism, reducing masseter muscle activity by up to 30–40%. However, the authors cautioned against routine use due to potential adverse reactions and stressed the need for treatment individualization.

Huynh et al. [11] evaluated the effectiveness of pharmacological treatments for sleep bruxism and found that therapeutic outcomes varied depending on the underlying aetiology of the disorder. Patients with stress-induced bruxism achieved symptom reduction of about 25–35% with anxiolytics (like diazepam).

Although pharmacological interventions may provide meaningful benefits, they are generally not recommended as first-line therapy for bruxism, but short-term adjunctive treatment, particularly in psychogenic cases. Furthermore, the potential risks of adverse effects, tolerance, and dependence, especially with prolonged benzodiazepine use, must be carefully considered.

Botulinum toxin

In recent years, botulinum toxin type A (BTX-A) has gained increasing attention as an effective therapeutic option for bruxism, particularly in patients with severe symptoms. A systematic review of 68 publications [12] identified four randomized trials that met the inclusion criteria and compared the efficacy of BTX-A against placebo (saline) or standard treatments like occlusal splints, pharmacotherapy, and cognitive-behavioural therapy (CBT).

The research showed that BTX-A injections significantly decreased bruxism episode frequency, as evidenced by patient reports and objective measures (EMG). Additionally, a marked reduction in muscle pain associated with masticatory hyperactivity was observed. Furthermore, a reduction in maximum occlusal force was achieved, thereby decreasing the risk of dental damage and TMJ overload. The treatment demonstrated superior efficacy compared with controls, including both placebo and conventional modalities, with the most pronounced therapeutic effects observed in patients with severe bruxism.

Based on the available evidence, the authors of the review concluded that BTX-A injections are generally safe and well tolerated. They may prove useful in routine clinical practice, especially for patients with severe or refractory bruxism unresponsive to other therapies.

Behavioural therapy

Behavioural therapy, including biofeedback and CBT, is one of the recommended non-invasive methods for alleviating bruxism, especially one linked to chronic stress and emotional tension. Available evidence shows that behavioural interventions significantly reduce teeth grinding and clenching episodes, along with associated masticatory muscle pain.

In their systematic review, Vieira et al. [13] demonstrated that biofeedback reduced the frequency of parafunction-

al episodes by up to 40% in awake bruxists compared to controls. Similarly, Ilovar et al. [14] reported that biofeedback therapy significantly decreased masseter muscle activity during both sleep and wakefulness in adult patients, with clinically meaningful improvement observed in 73% of participants.

Orthlieb et al. [15] reported that raising patient awareness of parafunctional habits like bruxism, followed by their modification via CBT, yields significant symptom relief, especially in “reactive” bruxism triggered by emotional stressors. It is important to note that the effectiveness of behavioural interventions largely depends on patient engagement and therapeutic consistency. Moreover, unlike pharmacological treatments or invasive methods, these therapies are not associated with adverse effects, which makes them an attractive long-term option, especially for those with chronic stress-induced bruxism.

Physiotherapy

Physiotherapy has gained popularity as an adjunctive treatment for bruxism, especially in patients with significant muscle tension and TMJ dysfunction. Physiotherapeutic interventions typically include manual therapy, deep tissue massage, stretching exercises, and postural improvement.

El-Gendy et al. [16] found that deep stripping massage significantly improved sleep quality (drop in PSQI by 5.8) and increased maximum mouth opening of an average by 0.87 cm at treatment week 6 in sleep bruxists.

A systematic review by Amorim et al. [17] evaluated the efficacy of physiotherapeutic interventions in bruxism. The analysis revealed that masticatory muscle massage, therapeutic exercises, and relaxation techniques significantly reduced pain (based on visual analogue scale, VAS), and enhanced masticatory function. The authors further highlighted the potential of physiotherapeutic interventions to alleviate bruxism, including masticatory hyperactivity. However, they pointed to considerable heterogeneity among the included studies and limited quality of some of the available evidence.

Considering the above, physiotherapy constitutes an effective element of bruxism management, particularly among patients whose symptoms are aggravated by stress and increased masticatory muscle tension. Physiotherapeutic interventions can be effectively combined with other therapeutic modalities, including occlusal splints and relaxation techniques, thereby increasing the efficacy of comprehensive treatment.

Occlusal splints

Occlusal splints, also known as stabilization or relaxation splints, represent one of the most common therapeutic interventions for both stress-related and idiopathic bruxism. Their primary objectives are to protect dental structures from excessive wear, reduce masticatory hyperactivity, and alleviate pain due to stomatognathic overload. However, the available evidence suggests variable clinical efficacy. In their systematic review, Macedo et al. [10] reported that, despite the widespread use of

occlusal splints for sleep bruxism, there is no conclusive evidence supporting their superiority over placebo or no therapeutic intervention. However, more recent analyses, including a review by Ainoosahy et al. [18], suggest a higher level of clinical efficacy for occlusal splints. Their review compared different types of splints and showed a reduction in the frequency of bruxism episodes by approximately 36–47% within the first 6–8 weeks of treatment.

In another study, Vrbanović and Alajbeg reported that occlusal splint therapy reduced masticatory muscle pain in 68% of bruxists vs 42% controls, confirming the beneficial effect of occlusal splint therapy on stress-induced somatic symptoms of bruxism.

However, Hardy and Bonsor [20] note that the long-term efficacy of occlusal splints remains uncertain, with their effectiveness potentially limited in cases where severe psychological stress or anxiety disorders are the dominant aetiology; in such cases, supplementation with behavioural or pharmacological therapy is recommended.

In summary, occlusal splints may represent a valuable therapeutic option for managing bruxism, particularly for short-term relief of physical symptoms. However, splint therapy should be used as part of a combined treatment approach in cases where stress constitutes a key aetiological factor.

Bruxism prevention and lifestyle modification

Contemporary research increasingly underscores the crucial role of psychological and behavioural factors in the pathogenesis of bruxism, thereby redirecting therapeutic focus toward non-pharmacological modalities, including lifestyle modifications. Many systematic reviews and meta-analyses demonstrated significant correlations between psychosocial stress, sleep disorders, and excessive consumption of stimulants, such as caffeine, alcohol, and tobacco, and both the occurrence and severity of awake and sleep bruxism [21–23].

Observational and clinical studies indicate that interventions targeting stress reduction, enhanced sleep hygiene, and elimination of maladaptive habits substantially reduce the incidence and intensity of bruxism episodes, particularly in mild to moderate cases [22]. Regularly performed relaxation techniques, including breathing exercises, meditation, and progressive muscle relaxation, have demonstrated efficacy in mitigating daytime jaw clenching and subjective masticatory muscle tension within several weeks of treatment onset [23].

Furthermore, the authors emphasize that chronic bruxism frequently co-occurs with chronic emotional stress, adjustment disorders, and sleep deprivation; the implementation of cognitive-behavioural strategies combined with efforts to reduce exposure to stressful environmental stimuli may lead to significant clinical improvement, including a marked reduction in the frequency of bruxism episodes [23]. Although lifestyle modifications are rarely sufficient as monotherapy for advanced bruxism, they constitute a vital element of adjunctive therapy, particu-

larly when integrated with dental, physiotherapeutic, or pharmacological interventions, as well as help prevent symptom recurrence.

Conclusions

Bruxism, which is a parafunctional disorder with complex, multifactorial aetiology, represents an escalating health concern, particularly among occupational cohorts exposed to chronic stress, such as military personnel. A growing body of evidence underscores the central role of chronic psychological stress in the development and persistence of both sleep and awake bruxism. In the investigated populations of military and non-military adults, statistically significant correlations were identified between stress levels, dental anxiety, increased sensitivity to dental stimuli, and bruxism. Moreover, risk analyses have linked coexisting sleep and awake bruxism with an approximately 8 times higher risk of TMDs.

An interdisciplinary therapeutic approach appears essential for effective management of bruxism. Pharmacological interventions, including benzodiazepines, dopamine agonists, and anxiolytics, have demonstrated moderate efficacy, particularly in cases with psychogenic aetiology. BTX-A injections have demonstrated efficacy in mitigating masticatory muscle hyperactivity and the frequency of bruxism episodes, particularly among patients exhibiting severe or treatment-resistant symptoms. Behavioural interventions, including biofeedback and cognitive-behavioural therapy, also play an important role in bruxism management by mitigating clinical symptoms and targeting underlying stress- and anxiety-related mechanisms. Physiotherapy, encompassing manual therapy techniques, masticatory muscle massage, and relaxation exercises, contributes to improved mandibular mobility and sleep quality. Occlusal splints, which effectively alleviate somatic symptoms, are used as an adjunct in comprehensive treatment, alongside lifestyle modifications and stress-reduction strategies.

For military personnel, effective diagnosis, treatment, and prevention of bruxism should be closely integrated with both general healthcare and psychological support systems. Regular mental health screening, stress management training, and targeted preventive programmes may substantially reduce the incidence of bruxism and its complications. This literature review highlights that a comprehensive understanding of the interplay between chronic stress and bruxism forms the cornerstone of effective therapeutic strategies, which aim to enhance the quality of life and psychophysical resilience among armed forces personnel.

References

1. Manfredini D, Winocur E, Guarda-Nardini L, et al. Epidemiology of bruxism in adults: a systematic review of the literature. *J Orofac Pain*, 2013 Spring; 27(2): 99–110. doi: 10.11607/jop.921.
2. Melo G, Duarte J, Pauletto P, et al. Bruxism: An umbrella review of systematic reviews. *J Oral Rehabil*, 2019 Jul; 46(7): 666–690. doi: 10.1111/joor.12801.

3. Manfredini D, Ahlberg J, Winocur E, Lobbezoo F. Management of sleep bruxism in adults: a qualitative systematic literature review. *J Oral Rehabil*, 2015 Nov; 42(11): 862–874. doi: 10.1111/joor.12322
4. Winocur E, Uziel N, Lisha T, et al. Self-reported bruxism – associations with perceived stress, motivation for control, dental anxiety and gagging. *J Oral Rehabil*, 2011 Jan; 38(1): 3–11. doi: 10.1111/j.1365-2842.2010.02118.x
5. Chen GC. Psychosocial risk factors and bruxism in the United States military population. [dissertation]. Bethesda (MD): Uniformed Services University of the Health Sciences, 2019
6. Sierwald I, John MT, Schierz O, et al. Association of temporomandibular disorder pain with awake and sleep bruxism in adults. *J Orofac Orthop*, 2015 Jul; 76(4): 305–317. doi: 10.1007/s00056-015-0293-5
7. Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: an international consensus. *J Oral Rehabil*, 2013 Jan; 40(1): 2–4. doi: 10.1111/joor.12011
8. Kato T, Rompré P, Montplaisir JY, et al. Sleep bruxism: an oromotor activity secondary to micro-arousal. *J Dent Res*, 2001 Oct; 80(10): 1940–1944. doi: 10.1177/00220345010800101501
9. Castroflorio T, Bargellini A, Rossini G, et al. Risk factors related to sleep bruxism in children: A systematic literature review. *Arch Oral Biol*, 2015 Nov; 60(11): 1618–1624. doi: 10.1016/j.archoralbio.2015.08.014
10. Macedo CR, Silva AB, Machado MA, et al. Occlusal splints for treating sleep bruxism (tooth grinding). *Cochrane Database Syst Rev*, 2007 Oct 17; 2007(4): CD005514. doi: 10.1002/14651858.CD005514.pub2
11. Huynh NT, Rompré PH, Montplaisir JY, et al. Comparison of various treatments for sleep bruxism using determinants of number needed to treat and effect size. *Int J Prosthodont*, 2006 Sep–Oct; 19(5): 435–441
12. Fernández-Núñez T, Amghar-Maach S, Gay-Escoda C. Efficacy of botulinum toxin in the treatment of bruxism: Systematic review. *Med Oral Patol Oral Cir Bucal*, 2019 Jul 1; 24(4): e416–e424. doi: 10.4317/medoral.22923
13. Vieira MA, Oliveira-Souza AIS, Hahn G, Bähr L, et al. Effectiveness of biofeedback in individuals with awake bruxism compared to other types of treatment: a systematic review. *Int J Environ Res Public Health*, 2023 Jan 14; 20(2): 1558. doi: 10.3390/ijerph20021558
14. Ilovar S, Zolger D, Castrillon E, et al. Biofeedback for treatment of awake and sleep bruxism in adults: systematic review protocol. *Syst Rev*, 2014 May 2; 3: 42. doi: 10.1186/2046-4053-3-42
15. Orthlieb JD, Tran NY, Camoin A, Mantout B. Propositions for a cognitive behavioral approach to bruxism management. *Int J Stomatol Occlusion Med*, 2013 Mar; 6(1): 6–15. doi: 10.1007/s12548-012-0072-5
16. El-Gendy MH, Ibrahim MM, Helmy ES, et al. Effect of manual physical therapy on sleep quality and jaw mobility in patients with bruxism: A biopsychosocial randomized controlled trial. *Front Neurol*, 2022 Dec 8; 13: 1041928. doi: 10.3389/fneur.2022.1041928
17. Amorim CSM, Espirito Santo AS, Sommer M, Marques AP. Effect of physical therapy in bruxism treatment: a systematic review. *J Manipulative Physiol Ther*, 2018 Jun; 41(5): 389–404. doi: 10.1016/j.jmpt.2017.10.014
18. Ainoosah S, Farghal AE, Alzemei MS, et al. Comparative analysis of different types of occlusal splints for the management of sleep bruxism: a systematic review. *BMC Oral Health*, 2024 Jan 5; 24(1): 29. doi: 10.1186/s12903-023-03782-6
19. Vrbanović E, Alajbeg IZ. Long-term effectiveness of occlusal splint therapy compared to placebo in patients with chronic temporomandibular disorders. *Acta Stomatol Croat*, 2019 Sep; 53(3): 195–206. doi: 10.15644/asc53/3/1
20. Hardy RS, Bonsor SJ. The efficacy of occlusal splints in the treatment of bruxism: A systematic review. *J Dent*, 2021 May; 108: 103621. doi: 10.1016/j.jdent.2021.103621
21. Bertazzo-Silveira E, Kruger CM, Porto De Toledo I, et al. Association between sleep bruxism and alcohol, caffeine, tobacco, and drug abuse: A systematic review. *J Am Dent Assoc*, 2016 Nov; 147(11): 859–866.e4. doi: 10.1016/j.adaj.2016.06.014
22. Polmann H, Réus JC, Massignan C, et al. Association between sleep bruxism and stress symptoms in adults: A systematic review and meta-analysis. *J Oral Rehabil*, 2021 May; 48(5): 621–631. doi: 10.1111/joor.13142
23. Minakuchi H, Fujisawa M, Abe Y, et al. Managements of sleep bruxism in adult: A systematic review. *Jpn Dent Sci Rev*, 2022 Nov; 58: 124–136. doi: 10.1016/j.jdsr.2022.02.004



TETANUS – WHAT A SURGEON SHOULD KNOW ABOUT *CLOSTRIDIUM TETANI*

Tężec – co chirurg powinien wiedzieć o zakażeniu *Clostridium tetani*



Paweł Radkowski^{1,2}, Maciej Szewczyk³, Tomasz Jędrzejak⁴, Łukasz Banasiak⁵, Julia Oszytko⁶

1. Department of Anesthesiology and Intensive Care, School of Medicine, Collegium Medicum, University of Warmia and Mazury in Olsztyn, Poland
2. Department of Anesthesiology and Intensive Care, Voivodeship Clinical Hospital in Olsztyn, Poland
3. Department of Internal Medicine, Independent Public Multispecialty Healthcare Center in Stargard, Poland
4. Department of General Surgery, SPZOZ Hospital in Działdowo, Poland
5. Department of Plastic Surgery, Voivodeship Specialist Hospital in Olsztyn, Poland
6. Faculty of Medicine, University of Opole, Poland

Paweł Radkowski –  0000-0002-9437-9458

Maciej Szewczyk –  0009-0009-0818-4848

Tomasz Jędrzejak –  0009-0006-6457-3651

Łukasz Banasiak –  0000-0001-5533-4057

Julia Oszytko –  0009-0008-8917-8943

Abstract

Tetanus is an infectious disease caused by the spore-forming bacterium *Clostridium tetani*, whose spores are commonly found in soil. Owing to the widespread implementation of vaccination programs, the incidence of tetanus has significantly declined; however, given the frequency of traumatic injuries and the environmental ubiquity of the pathogen, continued vigilance remains essential. The pathogenesis of the disease is primarily mediated by potent neurotoxins produced by the bacterium, particularly tetanospasmin. The hallmark clinical manifestations include generalized muscle spasms, which may progress to severe complications such as respiratory failure and death. Post-exposure management requires careful assessment of an individual's risk of developing tetanus in order to guide appropriate therapeutic strategies. In confirmed cases, treatment typically involves antimicrobial therapy, administration of tetanus-specific immunoglobulin, and supportive symptomatic care. The differential diagnosis should include conditions such as strychnine poisoning, viral encephalitis, oropharyngeal disorders, and rabies.

Streszczenie

Tężec jest chorobą zakaźną wywoływaną przez bakterię przetrwalnikującą *Clostridium tetani*, której przetrwalniki powszechnie występują w glebie. Dzięki szeroko zakrojonym programom szczepień ochronnych zapadalność na tężec znacznie się zmniejszyła, jednak ze względu na częste występowanie urazów oraz obecność drobnoustroju w środowisku, konieczne jest zachowanie czujności. Patogeneza choroby opiera się głównie na działaniu silnych neurotoksyn produkowanych przez bakterię, zwłaszcza tetanospazminy. Charakterystycznymi objawami klinicznymi są uogólnione skurcze mięśni, które mogą prowadzić do poważnych powikłań, takich jak niewydolność oddechowca, a nawet zgon. Postępowanie poekspozycyjne wymaga dokładnej oceny ryzyka rozwoju tężca, co umożliwi wdrożenie odpowiedniej strategii terapeutycznej. W przypadku potwierdzonego zakażenia leczenie zazwyczaj obejmuje antybiotykoterapię, podanie swoistej immunoglobuliny przeciw tężcowej oraz leczenie objawowe. W diagnostyce różnicowej należy uwzględnić zatrucie strychniną, wirusowe zapalenie mózgu, schorzenia jamy ustnej i gardła, a także wściekliznę.

Keywords: treatment; management; prophylaxis; *Clostridium tetani*

Słowa kluczowe: postępowanie; leczenie; profilaktyka; *Clostridium tetani*

DOI 10.53301/lw/206976

Received: 18.05.2025

Accepted: 11.06.2025

Published: 31.03.2026

Corresponding author:

Maciej Szewczyk

Department of Internal Medicine,
Independent Public Multispecialty

Healthcare Center in Stargard

e-mail: maciej95szewczyk@gmail.com

Introduction

Tetanus is an infectious disease caused by *Clostridium tetani*, a Gram-positive, anaerobic bacillus that naturally occurs in soil and in the gastrointestinal tract of mammals. The microorganism exists in both a vegetative form and as spores. *C. tetani* spores are highly resistant to adverse environmental conditions, allowing them to survive in soil and animal excreta for many years. Their main biological function is to ensure the survival of the strain in unfavorable conditions. Destruction of the spore form is possible through exposure to a temperature of 121°C for 15 minutes under a pressure of 1 atmosphere (autoclaving conditions).

Clostridium tetani produces two main toxins: tetanospasmin and tetanolysin. Tetanospasmin is a potent neurotoxin responsible for the characteristic neurological manifestations of tetanus. It acts by blocking the release of inhibitory neurotransmitters in the central nervous system, leading to uncontrolled muscle contractions. Tetanolysin, on the other hand, exhibits hemolytic properties, damaging the cell membranes of erythrocytes and potentially leading to hemolysis and secondary tissue necrosis [1, 2].

Epidemiology

Globally, tetanus remains a disease of epidemiological significance (particularly in its neonatal form in developing countries). According to the World Health Organization, 15,000 cases were reported in 2018 [3]. Before the introduction of routine vaccination in Poland in 1954, approximately 400 cases were reported annually. Between 2000 and 2017, the number of cases in Poland did not exceed 20 per year. In 2019, 17 cases were recorded, followed by two cases in 2020, and 13 in 2023, based on data from the National Institute of Public Health – National Institute of Hygiene in Poland. Notably, no cases of neonatal tetanus have been reported in Poland since 1984 [4].

In recent years, reported cases of tetanus in Poland have predominantly occurred in individuals over 60 years of age, with a tendency to present outside the spring and summer seasons, which may be related to an increase in gardening activities. A common feature in many reported cases is the absence of information regarding the patient's vaccination status, which may indicate a low level of awareness about recommended booster vaccinations. Consequently, it appears necessary to strengthen educational efforts to raise public awareness about the importance of tetanus booster vaccinations in adults [5]. In the aforementioned patients – older adults over 60 years of age who have not received booster vaccinations over an extended period – tetanus tends to follow a particularly severe course, often leading to death.

In recent years, due to anti-vaccination movements, public trust in vaccines has been declining. The number of exemptions from mandatory vaccinations has increased dramatically. Over the past five years, the number of such exemptions in Poland nearly doubled, rising from 48.6 thousand in 2019 to 87.3 thousand in 2023 [6, 7].

Pathogenesis

Tetanus infection most commonly occurs through the entry of *Clostridium tetani* spores into the body following contamination of damaged skin with soil, dust, organic fertilizers, or contaminated objects. Particularly dangerous are situations involving tissue injury caused by non-sterile instruments, such as cutting the umbilical cord under non-aseptic conditions. In such cases, neonatal tetanus (*tetanus neonatorum*) may develop.

The incubation period typically ranges from 4 to 21 days, although in some cases it may be significantly longer – even over 50 days. *C. tetani* spores can remain dormant in the body, encapsulated by connective tissue, and become activated under favorable conditions – such as tissue necrosis, hypoxia, impaired blood supply, or the presence of an anaerobic environment.

This bacterium preferentially colonizes wounds contaminated with organic material and coexisting aerobic flora (e.g., pyogenic bacteria), which consume oxygen and thereby create conditions favorable for the growth of anaerobes. The risk of infection is particularly high in inadequately treated wounds – especially those older than 24 hours, containing necrotic tissue, or resulting from highly penetrating mechanisms such as puncture, laceration, gunshot, or crush injuries.

However, infection may also develop following seemingly minor injuries, such as puncture wounds caused by nails. Cases of tetanus have also been reported after procedures performed under non-sterile conditions, including abortions, tattooing, intravenous injections in individuals with substance use disorders, as well as in patients with chronic wounds, pressure ulcers, and other ulcers, as well as following dental extractions and in the course of middle ear infections.

Tetanospasmin, the primary neurotoxin produced by *C. tetani*, is extremely potent; in a non-immunized individual, the lethal dose is only 2.5×10^{-9} mg [1, 8–10].

After *Clostridium tetani* spores transition into the active vegetative form within a wound, the bacteria multiply intensively and synthesize toxins responsible for the clinical manifestations of tetanus [1]. Once produced at the site of infection, tetanospasmin spreads via the lymphatic vessels into the systemic circulation and subsequently shows affinity for structures of the nervous system. The toxin penetrates the anterior horns of the spinal cord and the motor nuclei of cranial nerves, where it binds irreversibly to neurons.

Tetanospasmin exerts its pathogenic effect by irreversibly inhibiting the release of inhibitory neurotransmitters, including glycine and gamma-aminobutyric acid (GABA) by blocking the synaptic mechanism responsible for their release. This results in a loss of physiological inhibition of motor neurons, leading to increased muscle tone and painful tonic-clonic contractions of striated skeletal muscles. Characteristic clinical symptoms include trismus (lockjaw), spasms of the facial, neck, and trunk muscles, and, less commonly, involvement of the limb muscles [11].

Prognosis

Untreated tetanus may lead to severe complications, including respiratory failure of either obstructive or central origin, acute myocardial infarction, paralytic ileus, and in approximately 90% of cases – death. The highest mortality rates are observed in two age groups: children with incomplete vaccination and consequently low levels of specific immunity, and elderly individuals (over 65 years of age), in whom significantly reduced concentrations of circulating anti-tetanus antibodies are found.

The clinical presentation – particularly the sequence in which increased muscle tone develops in different muscle groups – depends on the route through which tetanospasmin spreads within the nervous system. This process is determined by the location of the wound and the distance the neurotoxin must travel to reach the central nervous system and peripheral nerve endings.

The severity of the clinical course is strongly correlated with the amount of toxin produced by a given strain of *Clostridium tetani*. The most severe forms of the disease are observed in patients with deep and extensive wounds, in women during the postpartum period, and in individuals who use intravenous drugs [11].

Recovery from tetanus does not confer immunity. Immunity is acquired through vaccination and is age-dependent; in individuals over 60 years of age, it may be as low as 30% [9, 10]. The major risk factors for tetanus infection are presented in Table 1.

Clinical symptoms

In adults, tetanus most often develops after minor skin injuries, such as abrasions or small cuts, while in children the most common route of pathogen entry is chronic otitis media. The initial phase of the disease is the prodromal phase, preceding the onset of full-blown tetanus. During this phase, nonspecific symptoms may occur, such as anxiety, general malaise, increased muscle tension, excessive sweating, headache, insomnia, as well as pain and paresthesias surrounding the wound.

In the advanced stage of the disease, particularly in more severe cases, the dominant clinical symptom is generalized tonic spasms of the skeletal muscles, which may be accompanied by respiratory arrest and cyanosis, while full consciousness is preserved, resembling the clinical picture of botulinum toxin poisoning. One of the pathognomonic symptoms of tetanus is the tonic spasm of the jaw muscles, leading to an inability to open the mouth, known as trismus (lockjaw), which sometimes results in the characteristic “snout-like” positioning of the lips.

Persistent increased tension of the facial mimic muscles gives the face an immobile, mask-like expression, sometimes creating the appearance of an ironic smile, referred to as risus sardonicus (sardonic smile) [12–14].

In severe, fully developed cases of tetanus, repeated episodes of generalized tonic spasms of the skeletal muscles are observed, lasting from a few seconds up to several minutes. Their frequency can exceed a dozen episodes per hour. These spasms, resulting from uncontrolled excitation of motor neurons, lead to a significant increase in muscle metabolism, which in turn causes hyperthermia and symptoms related to the autonomic nervous system, such as tachycardia, hypertension, excessive salivation, and sweating. The spasms primarily affect the muscles of the neck, back, trunk, abdomen, and lower limbs, producing the characteristic arched posture and stiffening of the body known as opisthotonus. Involvement of the upper limbs is rare and usually does not dominate the clinical picture of the disease [12–14].

Depending on the severity of symptoms, the clinical course of tetanus can be classified into the following degrees of severity – mild form: trismus (lockjaw), sardonic smile, occasional and mild muscle spasms, possibly slight stiffness; moderate form: dysphagia, marked stiffness, more frequent periodic muscle spasms, increased respiratory rate (>30 breaths/min); severe form: respiratory failure, generalized muscle spasms, tachycardia, blood pressure fluctuations, heart rate >120/min, increased respiratory rate (>40 breaths/min), episodes of apnea; very severe form (sometimes mentioned): all symptoms of the severe form, plus pronounced autonomic nervous system

Table 1. Tetanus – risk factors for disease development

Risk factors for tetanus development	
<ul style="list-style-type: none"> • Injury while working with soil • Obesity (poorer response to vaccination) • Intravenous drug use • Lack of up-to-date tetanus vaccination 	
High risk of tetanus infection	Low risk of tetanus infection
Crush wounds	Well-vascularized, small superficial wounds occurring in a home environment, without contain necrotic tissue
Deep puncture wounds	
Gunshot wounds	
Wounds containing foreign bodies	
Wounds heavily contaminated with soil, feces, or saliva	
Wounds not treated within 24 hours	
Wounds with associated shock	
Burns or frostbite	

Table 2. Clinical forms of tetanus [1, 8, 9, 12, 14]

Forms of tetanus	Course
Generalized	Most common form, occurring in about 80% of cases. Spasms appear in a descending pattern, starting with increased tension of the masseter muscles, difficulty chewing, progressing to trismus and sardonic smile (increased tension of the orbicularis oris muscle), followed by stiffness of the neck, difficulty swallowing, and stiffness of the chest muscles.
Localized	Rare form of tetanus, characterized by muscle stiffness localized around the wound (site of infection). In cases of partial immunity to the tetanus toxin, it may resolve spontaneously. Most commonly, it represents a prodromal phase of generalized tetanus.
Neonatal	Generalized form occurring in newborns of non-immunized mothers. Common in developing countries due to infection of the umbilical stump. Presents with poor feeding, facial grimacing, and severe spastic contractions that can be triggered by touch.
Cephalic	Rare form of tetanus, sometimes described as a specific type of localized tetanus, resulting from head injury. Presents as cranial nerve palsy (most commonly affecting the facial nerve, cranial nerve VII) and often weakness of the facial muscles (due to damage to the lower motor neuron).

disturbances, including severe hypertension, tachycardia or hypotension, and bradycardia [13, 14].

Depending on the clinical course, tetanus can present in four: generalized, localized, neonatal, and cephalic, which are presented in Table 2 [1, 8, 9, 12, 14].

Post-exposure prophylaxis – management

The procedure for managing suspected tetanus cases has been developed and adopted by the Epidemiological Committee of the Sanitary-Epidemiological Council under the Chief Sanitary Inspector. Management of wounds at risk of tetanus infection includes:

I. Wound care (cleaning and surgical management) – prevents the multiplication of *Clostridium tetani* in the wound, thereby inhibiting the production of tetanus neurotoxin.

II. Administration of antibiotics – to inhibit the growth of *C. tetani*. The most effective antibiotic is metronidazole, administered intravenously at a dose of 500 mg every 6 hours or 1,000 mg every 12 hours for 7–10 days. Older textbooks also note the effectiveness of penicillin, which is active in vitro against *C. tetani*, but as an antagonist of GABAergic transmission it may worsen prognosis. In case of metronidazole allergy, doxycycline (100 mg every 12 hours) is the drug of choice, or alternatively a macrolide or clindamycin, for 7–10 days. However, antibacterial therapy plays a relatively minor role.

III. Active immunization – involves administration of tetanus toxoid vaccine, either monovalent (tetanus only) or polyvalent (combined with pertussis and diphtheria). The primary vaccination in children consists of 4 DTP doses over two years, which provides immunity for approximately 5 years. Booster vaccinations are given to maintain basic immunity at ages 6, 14, and 19 years.

IV. Active-passive immunization – administration of human tetanus immunoglobulin (HTIG) in addition to the vaccine. Prophylactically, a dose of 250 IU is recommended. If more than 24 hours have passed since injury or if the wound is complicated, a dose of 500 IU should be administered. Higher doses are also recommended for obese individuals. For therapeutic purposes, the dose should be 3,000–6,000 IU, given as a single intramuscular injection

(without allergy testing; dosage may be modified according to manufacturer recommendations), combined with appropriate supportive treatment. In exceptional cases where HTIG is unavailable, equine antitoxin may be administered intramuscularly at 40,000–100,000 IU (after allergy testing). The antitoxin neutralizes free neurotoxin that has not yet bound to receptors, which shortens disease duration and alleviates symptoms [14, 15].

It is important to remember that:

- a single dose of vaccine given to an unvaccinated person does not provide immunity;
- the level and duration of immunity depend on the number of vaccinations received;
- having had tetanus does not confer immunity and does not protect against reinfection;
- routine use of antibiotics is not recommended in tetanus prophylaxis; however, wound observation and appropriate antibiotic therapy should be prescribed if signs of infection occur;
- tetanus prophylaxis should always be undertaken [14–16].

Table 3 may help facilitate decision-making regarding management.

In surgical practice, physicians should know when to apply post-exposure prophylaxis in patients presenting with wounds. The risk of developing tetanus is low in fresh, minimally contaminated wounds without necrotic tissue. The risk is high in heavily contaminated wounds, puncture wounds, lacerations, crush injuries, gunshot wounds, wounds treated after more than 24 hours, or wounds with necrotic tissue present within the injury [15, 17].

Differential diagnosis

Tetanus should be differentiated from other causes of increased muscle tone and spasms. In distinguishing tetanus from strychnine poisoning, normal muscle tone during the period without spasms supports strychnine poisoning, which is not seen in tetanus. When differentiating from viral encephalitis, which can also present with increased muscle tone and seizures, tetanus is indicated by complete preservation of consciousness [1]. Unlike encephalitis or meningitis, headache is not a feature in tetanus [13].

Table 3. Tetanus post-injury prophylaxis

Vaccination history	Risk of developing tetanus	
	Low risk	High risk
Unvaccinated person Uncertain vaccination history Incomplete vaccination	Unassociated tetanus vaccination according to the basic schedule (3 doses)	Unassociated tetanus vaccine according to the basic schedule + specific immunoglobulin (toxoid)
Last vaccine dose administered > 10 years ago	One booster dose	One booster dose + specific immunoglobulin (toxoid)
Last vaccine dose administered 5–10 years ago	One booster dose	One booster dose
Last vaccine dose administered < 5 years ago	Immunoprophylaxis not required	Immunoprophylaxis not required; in very high-risk cases, administration of one booster dose should be considered

Trismus is not unique to tetanus and requires exclusion of peritonsillar abscess, cellulitis of the floor of the mouth, odontogenic periostitis, and serum sickness, in which jaw joint pain with trismus may sometimes occur. Swallowing disorders may be of central origin, so this cause should be considered in the differential diagnosis, as along with rabies [1].

Tetanus should also be differentiated from tetany, overdose of psychoactive substances, or acute dystonic reaction, which may result from the use of haloperidol or promethazine. In acute dystonic reactions, neck muscle stiffness occurs with accompanying head twisting to the side, a feature not observed in tetanus [18].

Tetanus is diagnosed clinically, as there are no laboratory tests routinely available in practice to confirm the disease. A useful diagnostic tool is the spatula test: touching the posterior pharyngeal wall with a spatula normally triggers a gag reflex, whereas in tetanus it elicits a masseter muscle spasm, causing the patient to bite down on the spatula [1, 13]. This test has 100% specificity and 94% sensitivity [1].

Cerebrospinal fluid in tetanus usually shows no abnormalities [10].

Tetanus treatment

In suspected symptomatic tetanus, rapid action is essential. Whenever possible, it is important to ask the patient about their vaccination status, take a detailed history, attempt to identify the entry point of infection, place the patient in a dark and quiet room (preferably in the Intensive Care Unit), and draw blood for biochemical and toxicological tests (including tests for strychnine, neuroleptic drugs, phenothiazine derivatives, narcotics, and other tests guided by clinical suspicion) [14].

To quickly neutralize circulating toxin, it is crucial to administer human tetanus immunoglobulin (HTIG) at 3,000–6,000 IU intramuscularly as soon as possible, without allergy testing, or if unavailable, equine antitoxin at 40,000–100,000 IU intramuscularly and/or intravenously after an allergy test. Dosage should be adjusted according to the manufacturer's recommendations [14]. Surgical removal of necrotic tissue and thorough wound debridement to eliminate *Clostridium tetani* spores from the wound is also very important [14].

To eliminate the bacteria, antibiotic therapy should be initiated:

- Metronidazole IV, 500 mg every 6 hours or 1,000 mg every 12 hours for 7–10 days. In case of intolerance or allergy to metronidazole:
- Doxycycline (100 mg every 12 hours). For intravenous administration, proper preparation of the solution is essential: the doxycycline solution should be protected from light; the contents of the ampule should be diluted with sterile water for injection to 10 ml, then the stock solution should be further diluted in 100 to 1,000 ml of 0.9% sodium chloride or 5% glucose solution (yielding a concentration of 0.1 mg to 1 mg doxycycline per 1 ml). The solution should be prepared immediately before use and administered as an infusion over 1–4 hours;
- Alternatively, a macrolide or clindamycin may be considered (antibiotic treatment for 7–10 days) [12, 19].

It is crucial to maintain control over muscle spasms and excessive sympathetic nervous system activity. Frequent, strong spasms triggered by external stimuli – such as touch, pain, bright light, or sounds – can cause apnea and rhabdomyolysis [13]. To achieve sedation, reduce muscle tension, and prevent spasms, intravenous benzodiazepines may be administered (e.g., diazepam 10–40 mg every 1–8 hours or midazolam 5–15 mg/hour, depending on need). Based on available literature, midazolam may be the preferred choice; however, current reports are based on case series and individual clinical cases in which combinations of different drugs were used [1, 14, 20]. During benzodiazepine therapy, clinicians must monitor for excessive sedation, respiratory depression, or even coma. Antispasmodic treatment for tetanus should be continued for a prolonged period. After treatment ends, it is important to gradually reduce doses to avoid withdrawal syndrome [14].

Equally important is ensuring airway patency and proper ventilation. If airway obstruction persists, intubation and mechanical ventilation should be performed [14]. Early elective tracheostomy is recommended in moderate or severe cases to prevent aspiration and stridor [13].

In cases of very severe muscle spasms persisting despite sedation and/or mechanical ventilation disturbances caused by muscle contractions, intrathecal baclofen (administered at a dose of 1,000 µg every 24 hours or 40–200 µg as a single dose, followed by continuous infusion)

Table 4. Tetanus treatment

Tetanus treatment scheme		
Symptomatic treatment	Control of muscle spasms, securing the airway, maintaining proper blood pressure	Sedatives, muscle relaxants, mechanical ventilation, beta-blockers, morphine, magnesium sulfate
Specific treatment	Antibiotic therapy	Metronidazole, tetracycline
	Specific immunoglobulin	
Surgical treatment	Removal of microorganisms through proper wound cleansing	

can be considered. Alternatively, neuromuscular blockade using agents such as pancuronium or vecuronium may be employed, as these have been shown to be effective in the treatment of severe tetanus [9, 14]. When using neuromuscular blockade, it is important to maintain intravenous benzodiazepine therapy at the same doses to prevent autonomic nervous system hyperreactivity. The duration of neuromuscular blockade itself should be kept as short as possible [13].

It should also be remembered that the effect of muscle relaxants is influenced by factors such as acid-base balance disturbances (acidosis increases the effect of non-depolarizing skeletal muscle relaxants, while alkalosis decreases it; the opposite effect occurs with depolarizing muscle relaxants), and liver or kidney dysfunction. Therefore, in cases of renal failure or hepatic dysfunction, atracurium or cisatracurium appear to be the preferred drugs. However, in renal failure, clinicians should be aware of the possible accumulation of laudanosine, a metabolite with potential epileptogenic and hypotensive effects [21, 22].

For severe symptoms related to the sympathetic nervous system (tachycardia, excessive secretions, blood pressure spikes, excessive sweating, urinary retention), the following medications may be used:

- Magnesium sulfate i.v. (40 mg/kg over 30 minutes, followed by 2 g/h continuous infusion); literature shows it has a beneficial effect on controlling muscle spasms and sympathetic instability, reducing the need for ventilatory support and decreasing mortality; during magnesium sulfate treatment, patellar reflex should be periodically assessed; if absent, the dose should be reduced.
- Labetalol i.v. (0.25–1 mg/min)
- Morphine i.v. (0.5–1 mg/kg body weight per hour as continuous infusion) [1, 11, 14, 20].

If these measures are ineffective, epidural blockade may be considered [13, 14]. In cases where labetalol alone is insufficient to lower blood pressure, clonidine has been reported to improve outcomes [20].

For bradycardia, cardiac pacing should be applied; in hypotension, administration of crystalloids is recommended. During treatment, enteral nutrition via feeding tube should also be ensured – preferably with a high-calorie diet – alongside thrombosis prophylaxis and pressure sore prevention [1, 14].

For severe or moderate tetanus cases, treatment should take place in the Intensive Care Unit [14].

After resolution of muscle spasms, rehabilitation should begin, including physiotherapy and psychotherapy. Additionally, tetanus vaccination should be planned and started. Complete primary vaccination is recommended for unvaccinated patients, while previously vaccinated individuals should receive two doses spaced more than four weeks apart. The vaccine should be administered at a site different from where HTIG was given [14]. The therapeutic approach is summarized in Table 4.

Summary and conclusions

Tetanus is an acute infectious disease characterized by tonic spasms of the skeletal muscles. The spasm of the masseter muscles is referred to as trismus. Every patient with a wound requires tetanus prophylaxis, which can be either nonspecific or specific. Nonspecific prophylaxis involves cleaning and surgical debridement of the wound. Specific prophylaxis includes active-passive immunization, guided by an assessment of the patient's risk of developing tetanus. Despite widespread vaccination in many countries, tetanus remains present worldwide. There is a continuing need for further public education to raise awareness about the importance of booster tetanus vaccinations in adults. This disease leads to serious complications that can be life-threatening, so treatment requires both specific immunoglobulin therapy and supportive care, including intensive therapy. The availability of a highly immunogenic vaccine and effective, safe immunoglobulin allows for prevention even after exposure to tetanus spores. The most important factor is proper patient assessment for appropriate management. Key factors include the patient's tetanus vaccination history, immune status, and the nature of the wound.

References

- Bae C, Bourget D. Tetanus. 2023 May 31. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-.
- Garrigues L, Do TD, Bideaux C, et al. Insights into *Clostridium tetani*: From genome to bioreactors. *Biotechnol Adv*, 2022; 54: 107781. doi: 10.1016/j.biotechadv.2021.107781
- World Health Organization. Tetanus. https://www.who.int/health-topics/tetanus#tab=tab_1 (access 10.06.2025)
- Zieliński A. Tężec. In: Baumann-Popczyk A, Sadkowska-Todys M, Zieliński A, red. *Choroby zakaźne i pasożytnicze – epidemiologia i profilaktyka* (Wyd. VII). Alfa-Medica Press, 2014: 404–407
- Rumik A, Paradowska-Stankiewicz I. Tetanus in Poland in 2020–2022. *Przegl Epidemiol*. 2024; 78(4): 439–446. doi:10.32394/pe/195960

6. Zgliczyński WS. Szczepienia ochronne w Polsce. *Infos. Biuro Analiz Sejmowych*. 2021 Czerwiec 24; 6(287)
7. Jaka jest liczba uchyleń dotyczących szczepień obowiązkowych? Ostatnia aktualizacja: 30 września 2024, <https://szczepienia.pzh.gov.pl/faq/jaka-jest-liczba-uchylen-szczepien-obowiazkowych/>
8. Callison C, Nguyen H. Tetanus prophylaxis. [Updated 2023 Jan 23]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. <https://www.ncbi.nlm.nih.gov/books/NBK559008/>
9. George EK, De Jesus O, Tobin EH, et al. Tetanus (*Clostridium tetani* infection) [Updated 2024 Feb 26]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. <https://www.ncbi.nlm.nih.gov/books/NBK482484/>
10. Fields B, Guerin CS, Justice SB. Don't be a stiff: a review article on the management of tetanus. *Adv Emerg Nurs J*, 2021; 43(1): 10–20. doi: 10.1097/TME.0000000000000333
11. Yen LM, Thwaites CL. Tetanus. *Lancet*, 2019; 393 (10181): 1657–1668. doi: 10.1016/S0140-6736 (18) 33131-3
12. Brook I. Current concepts in the management of *Clostridium tetani* infection. *Expert Rev Anti Infect Ther*, 2008; 6(3): 327–336. doi: 10.1586/14787210.6.3.327.
13. Karnad DR, Gupta V. Intensive care management of severe tetanus. *Indian J Crit Care Med*, 2021; 25(Suppl 2): S155–S160. doi:10.5005/jp-journals-10071-23829
14. Flisiak R, Mrukowicz J. Tężec, Data aktualizacji: 13 sierpnia 2021. https://www.mp.pl/interna/chapter/B16.II.18.3.2?fbclid=IwZXh0bgNhZW0CMTEAAAR31kuJ8jToG5wWdd2haVH4o9CmxvejYb-wlGjWom2DB3IGlaK5NI3_KRSw_aem_sRdAn9eQfVzoNty5LVcxkQ (access: 15.12.2025)
15. Komunikat Głównego Inspektora Sanitarnego z dnia 28 października 2022 r. w sprawie Programu Szczepień Ochronnych na rok 2023. *Dz. U. Ministra Zdrowia*, poz. 113
16. Załęski A, Kuchar E. Post-exposure prophylaxis in cases of injury – rules of conduction. *Lek Wojsk*, 2022; 100(1): 7–11. doi:10.53301/lw/145192
17. Tetanus. *Epidemiology and Prevention of Vaccine-Preventable Diseases*. (www.cdc.gov/vaccines/pubs/pink-book/downloads/tetanus.pdf)
18. Tężec. *Interna Szczeklika 2023*. Podręcznik chorób wewnętrznych. Gajewski P, Szczeklika A, red. *Medycyna Praktyczna*, Kraków 2023; p. 2460–2462
19. Doxycyclinum TZF, 20 mg/ml, roztwór do infuzji Doxycyclinum, ChPL
20. Rodrigo C, Fernando D, Rajapakse S. Pharmacological management of tetanus: an evidence-based review. *Crit Care*, 2014; 18(2): 217. doi:10.1186/cc13797
21. Radkowski P, Szewczyk M, Czajka A, et al. The influence of acid-base balance on anesthetic muscle relaxants: a comprehensive review on clinical applications and mechanisms. *Med Sci Monit*, 2024; 30: e944510. doi:10.12659/MSM.944510
22. Radkowski P, Krupiniewicz KJ, Suchcicki M, et al. Navigating anesthesia: muscle relaxants and reversal agents in patients with renal impairment. *Med Sci Monit*, 2024; 30: e945141. doi:10.12659/MSM.945141



WHEN MYOCARDIAL INFARCTION IS NOT THE ACTUAL DIAGNOSIS – DIAGNOSTIC CHALLENGES IN ACUTE SETTINGS

Kiedy zawał serca to nie zawał – diagnostyczne wyzwania w stanach nagłych



Hubert Kowal^{1,2}, Marta Mielniczuk³, Jarosław Kowal^{4,3}, Paweł Krześciński³

1. Faculty of Medicine (student), Medical University of Lodz, Poland
2. Military Institute of Medicine – National Research Institute, Department of Cardiology and Internal Medicine (student), Poland
3. Military Institute of Medicine – National Research Institute, Department of Cardiology and Internal Medicine, Poland
4. Military Institute of Medicine – National Research Institute, Deputy Director for the Medical Support Region of the Armed Forces, Poland

Hubert Kowal –  0009-0006-9031-4768

Marta Mielniczuk –  0000-0002-3531-2042

Jarosław Kowal –  0009-0007-8196-2779

Paweł Krześciński –  0000-0003-1909-0993

Abstract

Aortic dissection is an acute state in which a tear in the intimal layer of the aortic wall allows blood to enter in between the intimal and medial layers. Risk factors include hypertension, smoking, dyslipidaemia, inflammatory diseases of the aorta and genetic connective tissue disorders. Severe, tearing chest pain is the main symptom of aortic dissection. The dissection may progress along the aorta, leading to several complications, such as myocardial infarction, acute aortic regurgitation, shock, stroke, acute kidney injury, abdominal organ ischaemia. Due to the high mortality rate, aortic dissection requires urgent diagnosis and rapid initiation of treatment. This article describes a case of a 78-year-old male admitted to the hospital with chest pain and neurological symptoms. Initially, the clinical presentation suggested a complicated myocardial infarction accompanied by cardiogenic shock; however, further diagnostic workup revealed an aortic dissection. The patient was transferred to the Cardiothoracic Surgery Operating Room, but he did not survive the surgery. The clinical presentation of aortic dissection can vary, with nonspecific symptoms due to possible multiple complications. Rapid, multidisciplinary diagnostic evaluation and immediate therapeutic intervention are essential, as the mortality rate increases rapidly over time.

Streszczenie

Rozwarstwienie aorty jest ostrym stanem, w którym w wyniku uszkodzenia błony wewnętrznej w ścianie aorty dochodzi do napływu krwi pomiędzy błoną wewnętrzną a środkową. Do czynników ryzyka rozwarstwienia należą: nadciśnienie tętnicze, palenie papierosów, dyslipidemia, choroby zapalne aorty i choroby genetyczne tkanki łącznej. Głównym objawem występującym w przypadku rozwarstwienia aorty piersiowej jest silny, rozdzierający ból w klatce piersiowej. Rozwarstwienie może postępować wzdłuż aorty, powodując liczne powikłania, m.in. zawał serca, ostrą niedomykalność aortalną, wstrząs, udar mózgu, ostre uszkodzenie nerek, niedokrwienie narządów jamy brzusznej. Ze względu na dużą śmiertelność jest to stan wymagający pilnej diagnostyki oraz jak najszybszego wdrożenia odpowiedniego postępowania leczniczego. W pracy przedstawiono opis przypadku 78-letniego mężczyzny przyjętego do szpitala z powodu bólu w klatce piersiowej i objawów neurologicznych. Obraz kliniczny przy przyjęciu w pierwszej kolejności wskazywał na powikłany zawał serca z towarzyszącym wstrząsem kardiogenym, natomiast w trakcie diagnostyki rozpoznano rozwarstwienie aorty. Pacjent po wstępnej stabilizacji stanu klinicznego trafił na Blok Operacyjny Kliniki Kardiologii, gdzie zmarł w trakcie operacji. Obraz kliniczny rozwarstwienia aorty może być różnicowany, objawy mogą być nieswoiste ze względu na możliwe liczne powikłania. Wymagana jest jak najszybsza wieloprofilowa diagnostyka i niezwłoczne wdrożenie leczenia, ze względu na dużą śmiertelność, która dynamicznie wzrasta w czasie.

Keywords: aortic dissection; myocardial infarction; shock; stroke; complicated dissection

Słowa kluczowe: rozwarstwienie aorty; zawał serca; wstrząs; udar; powikłane rozwarstwienie

DOI 10.53301/lw/207195

Received: 02.05.2025

Accepted: 16.06.2025

Published: 31.03.2026

Corresponding author:

Hubert Kowal

Faculty of Medicine,

Medical University of Lodz

e-mail: hubert.kowal222@gmail.com

Introduction

Aortic dissection (AD) is an acute aortic syndrome defined by an intimal tear that permits blood to enter the space between the intima and media, thereby forming a false lumen. AD is classified as type A (involving the ascending aorta) or type B (not involving the ascending aorta) according to the Stanford classification, as well as types I, II, IIIa, and IIIb (depending on the location of the dissection site) according to the DeBakey classification [1]. Hypertension (HT) is the most important and most common risk factor [2]. Cigarette smoking, dyslipidaemia, inflammatory aortic diseases, and genetic connective tissue disorders, such as Marfan syndrome, Ehlers–Danlos syndrome type 4, and Loeys–Dietz syndrome [3, 4], also play a significant role. The pooled incidence of all types of ADs is approximately 4.8 cases per 100,000 individuals per year, with the overall mortality rate reaching about 50% [5, 6]. Severe, excruciating chest pain unresponsive to nitrates that may radiate to the interscapular or lumbar regions, depending on the location of the lesion, is the main symptom of AD. Physical examination may reveal hyper- or hypotension, a murmur consistent with aortic regurgitation, and a pulse deficit in one limb. Complications of AD include aortic valve insufficiency, myocardial ischaemia or infarction, heart failure, shock, pericardial tamponade, limb or abdominal ischaemia, and neurological events such as stroke or transient cerebral ischaemia [3, 6].

According to the 2024 recommendations of the European Society of Cardiology, the diagnostic pathway depends on the risk of aortic dissection as assessed by the Aortic Dissection Detection Risk Score (ADD-RS). High-risk haemodynamically stable patients (ADD-RS \geq 2) should have computed tomography (CT) of the aorta performed as soon as possible. This modality demonstrates the highest sensitivity (100%) and specificity (98%) among the available imaging techniques and is therefore the preferred diagnostic tool [7, 8]. In haemodynamically unstable patients who cannot be transported for CT, transthoracic and/or transoesophageal echocardiography represents an alternative diagnostic approach. In low-risk patients (ADD-RS < 2), an electrocardiogram, point-of-care ultrasound (POCUS), chest radiography, or D-dimer testing should be performed. If any raises suspicion of AD, CT should follow [4]. ICU admission for continuous monitoring is recommended. Surgical intervention is indicated for acute type A aortic dissection and complicated type B dissections, where open repair or thoracic endovascular aortic repair (TEVAR) may be considered based on arterial anatomy. TEVAR may also be considered in uncomplicated type B dissections with favourable anatomy and an estimated life expectancy of at least five years. In other cases, conservative management and outpatient follow-up are recommended.

Each case of aortic dissection presents a major diagnostic and therapeutic challenge requiring specialist, interdisciplinary care. This paper reports a case of AD with a particularly insidious and unfavourable clinical course.

Case report

A 78-year-old man with unknown medical history was transported by emergency medical team (EMT) to the

emergency department (ED) with suspected ST-elevation myocardial infarction (STEMI). According to the EMT, the patient had been experiencing chest and epigastric pain accompanied by left-sided muscle weakness for several hours. An electrocardiogram performed by the EMT showed a junctional rhythm of 50 bpm, ST-segment elevation in leads II, III, and aVF, and ST-segment depression in lead V2. On admission, the patient was in serious condition, drowsy and confused, precluding medical history taking. Physical examination revealed left-sided weakness and pupillary asymmetry, with the right pupil dilated relative to the left. The patient was haemodynamically unstable (BP 80/50 mmHg), stabilised with a continuous dopamine infusion. Electrocardiographic findings suggestive of inferior STEMI prompted urgent transfer to the catheterization laboratory for coronary angiography. Due to haemodynamic instability from a junctional rhythm at 30 bpm, a temporary transvenous pacing lead was inserted in the first place, achieving capture at 80 bpm. Due to angiographic suspicion of cardiac tamponade, transthoracic echocardiography performed in the catheterization laboratory showed pericardial effusion with a maximum separation of 18 mm but no signs of tamponade. Coronary angiography demonstrated marked aortic dilatation with suspected dissection, and the procedure was therefore terminated. Emergency aortic CT confirmed a dissection involving the entire thoracic aorta (Fig. 1) and most likely the entire abdominal aorta, with possible extension into the iliac arteries. The dissection extended to the brachiocephalic trunk, the left common carotid artery (LCCA), the right common carotid artery, the left subclavian artery, and the left renal artery, with significant narrowing of the true lumen of the ascending aorta. A false lumen was identified in the LCCA, likely thrombosed, causing significant stenosis. The pericardial sac contained blood (approximately 15 mm), with bloody fluid measuring about 30 mm in both pleural cavities. The patient underwent cardiac surgical consultation and was scheduled for urgent surgical intervention. Upon admission to ICU, he remained in a serious general condition, drowsy and confused, with limited verbal and logical communication. Circulatory failure persisted despite continuous dobutamine infusion (blood pressure 90/55 mmHg, heart rate 80 bpm with ventricular pacing), along with respiratory failure requiring passive oxygen therapy via an oxygen mask. Pupillary asymmetry and left-sided muscle weakness persisted. Cardiac auscultation revealed a soft diastolic murmur. To stabilise haemodynamics, norepinephrine was added to the dobutamine infusion, and intravenous fluid therapy was initiated. Laboratory workup showed metabolic acidosis, elevated lactates (3.3 mmol/L), increased myocardial necrosis markers (hs-TnT 88 ng/L), impaired renal function (creatinine 1.5 mg/dL), markedly raised D-dimers (62.9 μ g/mL), mild normocytic anaemia (haemoglobin 11.6 g/dL), and thrombocytopenia ($100 \times 10^3/\mu$ L). POCUS demonstrated segmental wall motion abnormalities, including akinesia of the proximal half of the inferior and inferolateral walls, with a reduced left ventricular ejection fraction of 45%. The ascending aorta was markedly dilated from the bulb, with a visible dissection membrane. The aortic valve was tricuspid with a dilated annulus and at least moderate regurgitation. Pericardial effusion was also noted, measuring up to 15 mm anterior to the right ventricle (with a visible fibrin layer) and up to 12 mm posterior to the left

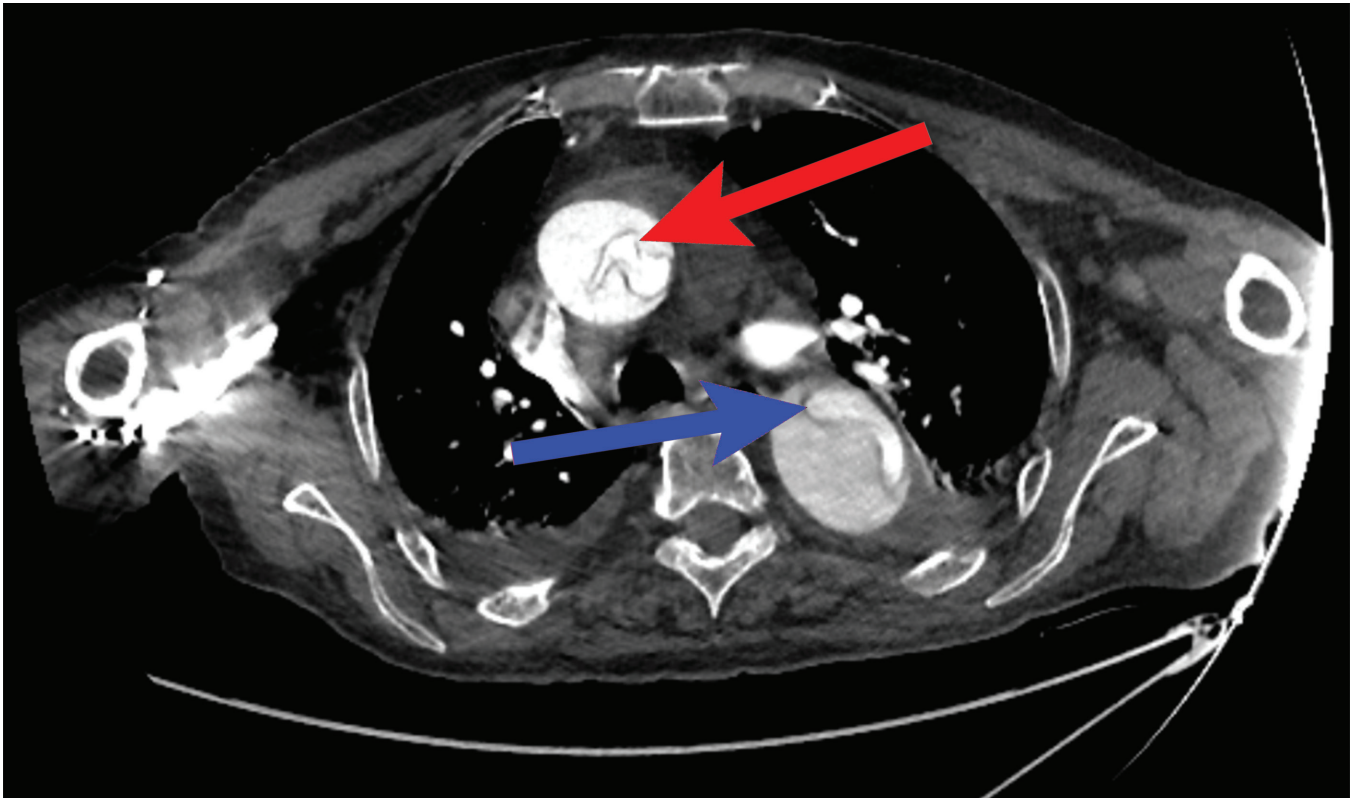


Figure 1. Evident dissection membrane in the ascending aorta (red arrow) and in the thoracic aorta (blue arrow)

ventricular posterior wall. The patient, in a critical condition, was transferred to the operating theatre of the Department of Cardiac Surgery for surgical intervention. Under cardiopulmonary bypass, median sternotomy was performed, relieving pericardial tamponade via a small incision, which provoked massive pericardial haemorrhage. Aortic rupture was palpated, with repeated unsuccessful attempts to place a transverse aortic clamp. Due to failure to establish effective cardiopulmonary bypass and control bleeding from the multidirectionally ruptured aorta, the patient died intraoperatively.

Discussion

Acute aortic dissection is a sudden, rapidly progressive emergency with high mortality, requiring prompt diagnosis and intervention. Acute aortic dissection often presents a diagnostic challenge, as its nonspecific symptoms can mimic other conditions, especially with organ involvement. This makes the presented case particularly noteworthy, as the patient exhibited multiple complications and features mimicking complicated MI rather than aortic dissection, thereby shaping the initial diagnostic and therapeutic strategy. Severe chest pain (type A in 79% and type B in 63% of cases), which may radiate to the interscapular and lumbar regions, is the primary symptom of AD [9]. Abdominal aorta involvement can cause abdominal pain, as seen in the presented case. Notably, abdominal pain may be the sole symptom of aortic dissection (AD) in 4.6% of patients, with such atypical presentations linked to delayed diagnosis and worse prognosis [10]. Chest pain is linked to many conditions and is a common cause of hospital admission, with atraumatic cases accounting for about 8% of ED visits [11]. AD is rarely the

initially suspected cause of such symptoms, accounting for only 0.1% of cases (1 in 980) [11]. In the present patient, in addition to pain, the electrocardiogram demonstrated ST-segment elevation in leads II, III, and aVF, plus impaired automaticity typical of inferior wall MI. The clinical presentation strongly suggested STEMI, prompting initiation of targeted diagnostic and therapeutic measures. The patient was urgently transferred to the catheterization laboratory for coronary angiography. However, significant aortic dilatation and suspected pericardial effusion noted early in the examination prompted discontinuation of further assessment.

Acute aortic regurgitation, occurring in 40–75% of cases, which may lead to acute heart failure or even shock, is one of the most common cardiovascular complications of AD. STEMI secondary to aortic dissection is diagnosed in only 0.51% of cases [12]. Shock is a severe complication of AD, affecting 15.1–23.3% of patients and linked to worse short-term prognosis and higher in-hospital mortality [13, 14]. The described patient developed cardiogenic shock, potentially stemming from MI, acute aortic regurgitation, or progressive pericardial effusion. AD may also be complicated by hypovolemic shock, particularly in cases of aortic rupture with haemorrhage into the pleural cavities, mediastinum, or peritoneal cavity. Additionally, the patient presented with neurological symptoms suggestive of stroke and laboratory evidence of acute renal failure. These findings may have been secondary to shock or dissection involving the renal and cranial arteries, as demonstrated on CT.

The diagnostic challenges associated with AD have been well illustrated in a case report by Lasa-Berasain et al. [15].

The authors described a 60-year-old patient with abdominal AD managed with endovascular repair, who suddenly developed neurological symptoms suggestive of acute stroke, including extension of the left upper limb, drooping of the left corner of the mouth, and loss of consciousness. The patient subsequently experienced cardiac arrest; after successful resuscitation, he remained haemodynamically unstable and was therefore transferred to a referral hospital for further management. On admission to ED, physical examination revealed weak peripheral pulses, prolonged capillary refill time, and an early diastolic murmur over the aortic valve. The patient was hypotensive, with a blood pressure of 88/67 mmHg. Electrocardiography showed ST-segment elevation in the lateral leads, accompanied by ST-segment depression and tall T waves in the anterior leads. POCUS revealed, among other findings, a dissection membrane in the ascending aorta, along with aortic regurgitation. An urgent CT scan subsequently confirmed type A aortic dissection. In the authors' case, closely mirroring ours, the clinical presentation mimicked MI. Aortic dissection was suspected only after POCUS demonstrated the dissection membrane.

Chenkin [16] described a 69-year-old man presenting to ED with sudden-onset severe stabbing retrosternal pain that began 2 hours earlier during physical exertion. The pain was accompanied by dyspnoea and profuse sweating. Physical examination was largely unremarkable, except for markedly elevated blood pressure (185/101 mmHg). Electrocardiography revealed ST-segment elevation in leads aVR, V1, and V2, along with diffuse ST-segment depression in multiple other leads. Due to suspected acute coronary syndrome, the patient was scheduled for transfer to the catheterization laboratory. However, US performed in the emergency department revealed a dissection membrane in the ascending aorta along with severe aortic regurgitation. Pharmacological treatment was initiated, and an urgent CT scan was performed, which confirmed acute type A aortic dissection. During transport to the operating room, the patient developed cardiac arrest and died despite resuscitation efforts. Myocardial infarction masked AAD, posing a major diagnostic challenge due to their divergent treatment strategies.

Conclusions

As shown by our case report and other cited cases, acute aortic dissection produces a highly heterogeneous clinical picture, necessitating prompt multidisciplinary diagnostic evaluation for accurate diagnosis. In certain cases, subtly expressed symptoms or findings noted only on physical examination may prove crucial in raising suspicion of AD. Prompt and accurate diagnosis of AD is critical, as mortality rises by about 2% per hour of delay in initiating appropriate treatment [17]. Multi-organ complications can progressively alter the patient's clinical presentation and condition, leading to fatal outcomes despite advanced diagnosis and therapy.

References

1. Sherkw WM, Khaja MS, Williams DM. Anatomy, pathology, and classification of aortic dissection. *Tech Vasc Interv Radiol*, 2021; 24(2): 100746. doi: 10.1016/j.tvir.2021.100746
2. Hibino M, Otaki Y, Kobeissi E, et al. Blood pressure, hypertension, and the risk of aortic dissection incidence and mortality: results from the J-SCH study, the UK Biobank study, and a meta-analysis of cohort studies. *Circulation*, 2022; 145(9): 633–644. doi: 10.1161/CIRCULATIONAHA.121.056546
3. Sayed A, Munir M, Bahbah EI. Aortic dissection: a review of the pathophysiology, management and prospective advances. *Curr Cardiol Rev*, 2021; 17(4): e230421186875. doi: 10.2174/1573403X16666201014142930
4. Mazzolai L, Teixido-Tura G, Lanzi S, et al. ESC Scientific Document Group. 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases. *Eur Heart J*, 2024; 45(36): 3538–3700. doi: 10.1093/eurheartj/ehae179
5. Gouveia E Melo R, Mourão M, Caldeira D, et al. A systematic review and meta-analysis of the incidence of acute aortic dissections in population-based studies. *J Vasc Surg*, 2022; 75(2): 709–720. doi: 10.1016/j.jvs.2021.08.080
6. Booth K; (on behalf of UK-AS, the UK Aortic Society). Acute aortic dissection (AAD) – a lethal disease: the epidemiology, pathophysiology and natural history. *Br J Cardiol*, 2023; 30(1): 9. doi: 10.5837/bjc.2023.009
7. Goldstein SA, Evangelista A, Abbara S, et al. Multimodality imaging of diseases of the thoracic aorta in adults: from the American Society of Echocardiography and the European Association of Cardiovascular Imaging: endorsed by the Society of Cardiovascular Computed Tomography and Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*, 2015; 28(2): 119–82. doi: 10.1016/j.echo.2014.11.015
8. Shiga T, Wajima Z, Apfel CC, et al. Diagnostic accuracy of transesophageal echocardiography, helical computed tomography, and magnetic resonance imaging for suspected thoracic aortic dissection: systematic review and meta-analysis. *Arch Intern Med*, 2006; 166(13): 1350–1356. doi: 10.1001/archinte.166.13.1350
9. Evangelista A, Isselbacher EM, Bossone E, et al. Insights From the International Registry of Acute Aortic Dissection: A 20-year experience of collaborative clinical research. *Circulation*, 2018; 137(17): 1846–1860. doi: 10.1161/CIRCULATIONAHA.117.031264
10. Upchurch GR Jr, Nienaber C, Fattori R, et al. Acute aortic dissection presenting with primarily abdominal pain: a rare manifestation of a deadly disease. *Ann Vasc Surg*, 2005; 19(3): 367–373. doi: 10.1007/s10016-004-0171-x
11. Alter SM, Eskin B, Allegra JR. Diagnosis of aortic dissection in emergency department patients is rare. *West J Emerg Med*, 2015; 16(5): 629–631. doi: 10.5811/westjem.2015.6.25752
12. Zhu QY, Tai S, Tang L, et al. STEMI could be the primary presentation of acute aortic dissection. *Am J Emerg Med*, 2017; 35(11): 1713–1717. doi: 10.1016/j.ajem.2017.05.010
13. Bossone E, Pyeritz RE, Braverman AC, et al. Shock complicating type A acute aortic dissection: Clinical correlates, management, and outcomes. *Am Heart J*, 2016; 176: 93–99. doi: 10.1016/j.ahj.2016.02.019

-
14. Lin CY, Kao MC, Lee HF, et al. Analysis of outcomes and prognostic factor in acute type A aortic dissection complicated with preoperative shock: A single-center study. *PLoS One*, 2024; 19(4): e0302669. doi: 10.1371/journal.pone.0302669
 15. Lasa-Berasain P, Salas PR, Azparren EE, Sanz ER. Myocardial infarction and haemorrhagic stroke as a rare presentation of acute aortic dissection: a fatal case report. *Eur Heart J Case Rep*, 2023; 7(11): ytad529. doi: 10.1093/ehjcr/ytad529
 16. Chenkin J. Diagnosis of aortic dissection presenting as ST-elevation myocardial infarction using point-of-care ultrasound. *J Emerg Med*, 2017; 53(6): 880–884. doi: 10.1016/j.jemermed.2017.08.012
 17. Chukwu M, Ehsan P, Aburumman RN, et al. Acute stanford type A aortic dissection: a review of risk factors and outcomes. *Cureus*, 2023; 15(3): e36301. doi: 10.7759/cureus.36301



ATRIAL FIBRILLATION – WHAT ARE THE BENEFITS OF M-HEALTH?

Migotanie przedsionków –
co daje nam m-health?



Dawid Sidorczuk, Agata Galas, Paweł Krzesiński

Military Institute of Medicine – National Research Institute, Department of Cardiology and Internal Diseases, Warsaw, Poland

Dawid Sidorczuk – 0009-0009-9412-2511

Agata Galas – 0000-0002-8683-3913

Paweł Krzesiński – 0000-0003-1909-0993

Abstract

The development of mobile technologies has led to a growing popularity of health-related applications (m-health/mobile health). Some of these devices enable users to register single-lead electrocardiograms, which can facilitate diagnosis and help prevent complications associated with the most common arrhythmias, such as atrial fibrillation. This study reviews the performance of devices based on oscillometry and plethysmography, as well as those capable of generating ECG recordings that may serve as a basis for atrial fibrillation diagnosis. Given the increasing prevalence of modern technologies across diverse population groups (including healthy individuals, physically active people, and patients with diagnosed cardiovascular disease), it is essential to consider the limitations associated with their use.

Streszczenie

Rozwój technologii mobilnych spowodował wzrost popularności aplikacji związanych ze zdrowiem (m-health/mobile health). Część z tych urządzeń pozwala na rejestrację jednodowodzeniowego elektrokardiogramu, co może ułatwiać diagnostykę i zapobieganie powikłaniom najczęstszych arytmii, np. migotania przedsionków. W pracy omówiono działanie urządzeń opartych na oscylometrii i pletyzmografii oraz takich, które pozwalają na uzyskanie zapisu elektrokardiogramu i mogą stanowić podstawę rozpoznania migotania przedsionków. Ze względu na rosnącą powszechność wykorzystania nowoczesnych technologii w różnych grupach populacyjnych (w tym osób zdrowych, aktywnych fizycznie oraz pacjentów z rozpoznanymi schorzeniami układu krążenia), niezbędne jest uwzględnienie ograniczeń związanych z ich zastosowaniem.

Keywords: m-health; atrial fibrillation; smart devices

Słowa kluczowe: m-health; migotanie przedsionków; urządzenia „smart”

DOI 10.53301/lw/207196

Received: 07.06.2025

Accepted: 16.06.2025

Published: 31.03.2026

Corresponding author:

Dawid Sidorczuk

Military Institute of Medicine – National Research
Institute, Department of Cardiology and Internal
Diseases, 128 Szaserów Str., 04-141 Warsaw
e-mail: dawid.sidorczuk@gmail.com

Introduction

With the widespread adoption of smartphones and other mobile devices, the popularity of health-related applications (m-health/mobile health) has been steadily increasing. Some of these apps allow users to monitor parameters such as blood pressure or body weight, replacing traditional paper logs. Others enable the recording of heart rate or single-lead electrocardiograms (ECG), either as standalone tools or in combination with a dedicated device.

ECG monitoring via mobile devices can serve as a method for detecting asymptomatic arrhythmias – potential causes of severe yet preventable complications. A notable example is atrial fibrillation (AF), where complications can be prevented through anticoagulant therapy. Beyond clinical settings, these tools can also support individuals at increased risk of cardiac events due to lifestyle factors, pre-existing health conditions, or the type of sports they practise.

Aim of study

The aim of this study is to present the available methods of ECG monitoring using mobile devices and their efficacy in detecting the most common arrhythmia, which is AF.

Diagnosis of atrial fibrillation

AF has an estimated prevalence of 1–2% [1]. However, because many patients remain asymptomatic and therefore undiagnosed, the true prevalence of this condition is likely underestimated. According to the 2024 European Society of Cardiology (ESC) guidelines for the management of atrial fibrillation, the gold standard for diagnosing AF is an ECG recording, regardless of the number of leads [2]. This means that AF can also be diagnosed using single-lead recordings, such as those obtained from mobile devices. The guidelines additionally introduce the concept of device-detected subclinical AF, which – although insufficient for establishing a definitive diagnosis – serves as a prognostic marker for future AF [2].

Available technologies

The 2024 ESC guidelines divide the available rhythm-assessment tools into diagnostic and non-diagnostic categories [2]. The former include standard 12-lead ECG, Holter monitoring, telemetry, handheld devices, dedicated patches, biotextiles, and smart devices (watches and smartphones). These tools allow for the acquisition of single- or multi-lead ECG recordings. It is worth noting that the first three options are limited to outpatient or hospital settings. Tools classified as non-diagnostic are based solely on heart rhythm assessment using plethysmography, oscillometry, accelerometry, mechanocardiography, acoustic analysis, or pulse palpation. They do not allow for recording an ECG trace, and a diagnosis cannot be established on their basis. However, abnormalities detected by these devices may indicate an increased risk of arrhythmia and prompt further diagnostic evaluation.

Oscillometry

Oscillometric analysis is used in automatic blood pressure monitors. This method relies on detecting changes in the volume of the limb enclosed by the cuff, depending on the cuff-inflation pressure. In addition to determining systolic and diastolic blood pressure values, the measurement also provides the heart rate [3].

By measuring pulse intervals during gradual deflation of the cuff, the device can calculate both the mean value and the standard deviation of the intervals, which in turn allows the determination of an irregularity index. Based on this, the device can classify the rhythm as regular or irregular, resulting in a notification indicating a potential risk of arrhythmia [4].

Pucci et al. compared the effectiveness of AF screening using the Microlife WatchBP blood pressure monitor with standard 12-lead ECG [5]. The study population consisted of patients admitted to an internal medicine ward. The sensitivity of the blood pressure monitor for AF detection was 86%, while specificity reached 96%. The authors noted that false-negative results were associated with AF presenting with regular ventricular activity (QRS complexes). Among patients with newly diagnosed AF, the device demonstrated 100% effectiveness.

Photoplethysmography

Photoplethysmography (PPG) is a method based on assessing variations in blood volume within blood vessels by analysing changes in the parameters of a reflected light beam. This enables measurement of the periodic rise and fall in vascular filling, allowing determination of a time interval equivalent to the R-R interval on an ECG. A smartphone camera can be used to record the reflected light, with a fingertip placed over the lens. The application installed on the device then evaluates rhythm regularity and informs the user of potential abnormalities [6].

Chan et al. evaluated the effectiveness of AF detection using the Cardio Rhythm app on an iPhone 4S [7]. Each of the three PPG recordings performed was classified as either regular or irregular; if at least two out of three were determined to be irregular, the application identified AF. The sensitivity of this method was approximately 93%, with a specificity of nearly 98%. The authors noted potential difficulties in obtaining a correct recording, such as motion artifacts from finger movement and inadequate finger placement on the camera. The study also compared the above method with the performance of the AliveCor Heart Monitor, which provides a single-lead ECG recording. The specificity of both methods was comparable; however, the sensitivity of the automatic AliveCor algorithm was 71.4%, a lower value than reported in the earlier study by Lau et al. [8], where sensitivity reached 98%. The authors emphasised that the discrepancy between the two studies may be related to Chan et al. using a newer version of the application.

Handheld devices

Handheld devices allow the recording of a single-lead ECG. An example is the Beurer ME90 [9]. The tracing is

obtained by placing the index fingers on the sensor, which enables acquisition of lead I. The built-in algorithm performs real-time analysis of the recording and, through messages displayed on the screen, informs the user whether the tracing is normal (“OK”) or whether an arrhythmia (“Rhythm”), a pause (“Pause”), or an abnormal QRS morphology (“Wave”) has been detected. Additionally, it provides information on the heart rate. The device allows results to be displayed on a smartphone via Bluetooth and enables data export to a computer via USB (Universal Serial Bus).

A study conducted by Brito et al. evaluated the effectiveness of the Beurer ME90’s built-in algorithm in detecting atrial fibrillation and atrial flutter compared with a standard 12-lead ECG. An alternative device placement was also tested by positioning it parasternal on the left side to obtain a modified V4 lead. It was shown that for lead I recordings, sensitivity was 88.9% and specificity was 61.9%, with false-negative results occurring exclusively in cases of atrial flutter. The use of the modified V4 lead in combination with lead I increased specificity to 84.3% [10].

KardiaBand

KardiaBand is an optional accessory for the Apple Watch that enables single-lead ECG recording. It is a wristband that can replace the standard watch strap. It contains a sensor that records an ECG tracing when the user places a finger from the opposite hand on its surface. The band connects to the smartwatch via Bluetooth and can also be paired with an iPhone [11].

Bumgarner et al. compared the effectiveness of distinguishing sinus rhythm from AF in patients scheduled for electrical cardioversion [12]. Each participant underwent a standard ECG recording, followed by a KardiaBand recording. Each tracing was then evaluated by two physicians, who classified the rhythm into one of four categories: sinus rhythm, AF, atrial flutter, or unclassified. The interpretation generated by the device’s automatic algorithm was also compared with the physicians’ assessments.

Overall, the algorithm achieved a sensitivity of 93% and a specificity of 84%. However, a substantial proportion of KardiaBand tracings (57 out of 169) remained unclassified by the algorithm. According to the authors, this was primarily due to insufficient recording duration, low P-wave amplitude, or the presence of artifacts. A clinician-led review of these unclassified recordings provided further insight: AF was confirmed in all 14 AF cases, but 5 out of 25 sinus rhythm recordings were incorrectly classified as AF (80% specificity). When all KardiaBand recordings were interpreted by clinicians and compared with a standard ECG, the sensitivity for detecting AF increased to 99%, with a specificity of 83%.

The authors note that the demonstrated efficacy of the band in detecting AF is comparable to that of implantable loop recorders, and they highlight the strong correlation between KardiaBand tracings and standard 12-lead ECG recordings. They conclude that the KardiaBand is highly effective for AF detection; however,

final interpretation of the recordings should still be supported by clinician review.

Kardia Mobile

Kardia Mobile is a device designed to work with smartphones and tablets. It is shaped like a small plate with metal electrodes on both sides. To record an ECG, the user opens the dedicated Kardia app and selects the recording option. The tracing is then obtained by placing the index and middle fingers of both hands on the electrodes, producing a recording comparable to lead I of a standard ECG. The recording time is 30 seconds, after which the tracing is wirelessly transmitted to the connected smart device. The app’s automatic algorithm analyses the recording and generates one of the following messages: “interference,” “normal,” “AF,” or “unclassified” if the tracing is unsuitable for interpretation. Data export in PDF format is also available. The device is compatible with most Apple and Android systems [13, 14].

Kotowski et al. compared the effectiveness of AF detection using ECG recordings from the Kardia Mobile device with that of a standard 12-lead ECG [14]. The sensitivity for detecting AF was 92.8%, and the specificity was 100%. It is worth noting that the study did not assess the performance of the automatic algorithm, as the recordings were interpreted by physicians based on printed exports of the data.

Apple Watch

The Apple Watch Series 4 is a smartwatch equipped with two electrodes that enable single-lead ECG recording. One electrode is located beneath the watch face, and the other on its lateral surface. Recording is performed by resting the arm wearing the watch on a stable surface and touching the side electrode with a finger of the opposite hand. The resulting tracing is analysed by an algorithm, after which the user receives one of four feedback messages: sinus rhythm (if the heart rate is between 50–100 beats/min), AF (irregular rhythm 50–120 beats/min), low or high heart rate, or inconclusive (when the tracing cannot be reliably interpreted). Similar to the Kardia Mobile device, results can be presented on an iPhone screen and exported in PDF format. It is worth noting that when the heart rate is below 50 beats/min or above 120 beats/min, algorithmic interpretation may be impaired, resulting in an inconclusive outcome [15].

Shahid et al. conducted a meta-analysis of studies evaluating the effectiveness of the Apple Watch compared with a standard 12-lead ECG [16]. The review included 11 studies with a total of 4,241 participants with diverse clinical profiles. The device demonstrated high accuracy in detecting AF, with a sensitivity of 94.8% and a specificity of 95%. The authors emphasised that despite the considerable heterogeneity of the study populations, restricting the analysis to patients with AF reduced this variability, supporting the consistency of the available evidence. However, they also highlighted the need for improved methodology in future research to better assess, among other aspects, the clinical utility and economic implications of using this technology.

Mobile recorders and stroke risk

A diagnosis of AF significantly increases the risk of ischaemic stroke. In patients with AF, the risk is estimated to be approximately five times higher than in the general population [17]. To prevent thromboembolic events, the ESC guidelines recommend initiating anticoagulation therapy in all patients with elevated risk, whenever possible [2]. This principle is reflected in the “CARE” strategy for AF management, in which “A” stands for “Avoid” (Avoid stroke and thromboembolism).

The growing popularity of self-recording ECG devices raises the question of whether they can contribute to reducing this risk and enable the timely initiation of therapy using direct oral anticoagulants (DOACs) or vitamin K antagonists.

Feldman et al. attempted to estimate the proportion of individuals using ECG-enabled devices who could benefit from anticoagulant treatment if the diagnosis of AF were based on these devices [18]. It was shown that only 0.25% of the subjects would be candidates for initiating such treatment. This means that out of 400 device users, only one would derive measurable benefits in the prevention of ischaemic events.

A significant concern involves the complications of anticoagulant treatment, specifically the increased risk of bleeding. Sunaga et al. are currently conducting research on the feasibility of using DOAC therapy based on patient monitoring with the Apple Watch Series 4 [19]. The study includes patients who have undergone ablation and subsequently returned to sinus rhythm. The primary premise is to continue DOAC treatment in the event of arrhythmia and to discontinue therapy in the absence of AF episodes. The initial observation period lasts 30 days. If no AF episode occurs during this time, anticoagulation is discontinued on the 31st day. If an AF episode occurs during the first month or after cessation, therapy is to be continued or reinitiated, respectively. The primary endpoint is the percentage reduction in the number of days on DOAC therapy during a 360-day observation period. Secondary endpoints include mortality, stroke, thromboembolic events, bleeding episodes, and device malfunction. The study aims to determine whether personalised DOAC therapy guided by Apple Watch monitoring can reduce DOAC use compared with conventional anticoagulation therapy.

Summary and practical implications

The growing popularity and accessibility of smart devices offer new opportunities to leverage their technology for health monitoring. The development of devices and applications dedicated to heart rhythm analysis and short ECG recording enables their use in the diagnosis and

management of various conditions, including AF. A concise overview of the operating principles and effectiveness of individual methods, as reported across numerous studies, is presented in Table 1.

When considering the advantages of handheld or mobile ECG monitoring, ease of use, intuitive interfaces, and immediate, on-demand accessibility are particularly noteworthy. In the event of arrhythmia symptoms, users can instantly record a tracing and analyse the rhythm, unlike with a standard 12-lead ECG, which requires a visit to a medical facility. Collecting results, for example in the form of PDF files, enables subsequent medical consultation and, according to current guidelines, may support both the diagnosis of the disease and the initiation of treatment. However, it should be noted that the user decides when to activate the relevant function and perform the examination, which limits the detection of clinically silent arrhythmic episodes that do not prompt a recording.

Importantly, these devices may generate false-positive results, which can cause unnecessary anxiety among users and lead to redundant diagnostic testing. Another drawback is the difficulty in distinguishing between different arrhythmias, as some false-negative results may mask other rhythm disorders, such as atrial flutter. Furthermore, it is important to note the potential difficulties faced by elderly individuals when using such devices. This population represents the vast majority of patients affected by heart rhythm disorders, yet operating smartphone-based devices or applications can pose a significant challenge for them, often due to lower technological proficiency and difficulties in acquiring new digital skills. This may significantly limit the use of the methods discussed, which in turn would reduce their potential benefits.

In screening for AF, a significant challenge is the relatively high number of patients who must be screened to detect a single case, which entails potential costs related to the unit price of the device. The very need for active arrhythmia screening and for monitoring an asymptomatic individual – who, although considered healthy, carries certain risk factors – may not provide sufficient motivation for purchasing a smartwatch or smartphone.

Undoubtedly, however, the availability of this type of technology creates substantial opportunities for use in both the prevention and management of AF. The high sensitivity of these devices places them almost on par with the methods used to date. The ability to identify arrhythmia based on a single-lead ECG helps overcome the limitation of capturing arrhythmic episodes exclusively during standard ambulatory ECG. The continued advancement of this technology, together with improved accessibility and the reduction of current limitations and

Table 1. Sensitivity and specificity of selected mobile ECG monitoring methods [5, 7, 10, 12, 14, 16]

	Microlife WatchBP	Cardiio Rhythm	Beurer ME90	KardiaBand	Kardia Mobile	Apple Watch
Sensitivity	86%	93%	88.9%	93%	92.8%	94.8%
Specificity	96%	98%	61.9%	84%	100%	95%

drawbacks, will undoubtedly broaden the scope of benefits for patients with cardiac rhythm disorders in the future.

References

1. Balsam P. Migotanie przedsionków – definicja, patogenez, etiologia i klasyfikacja. *Choroby Serca i Naczyń*, 2019; 16(1): 63–65
2. Van Gelder IC, Rienstra M, Bunting KV, et al.; ESC Scientific Document Group. 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*, 2024; 45(36): 3314–3414. doi: 10.1093/eurheartj/ehae176. Erratum in: *Eur Heart J*, 2025; 46(41): 4349. doi: 10.1093/eurheartj/ehaf306
3. Lewis PS; British and Irish Hypertension Society's Blood Pressure Measurement Working Party. Oscillometric measurement of blood pressure: a simplified explanation. A technical note on behalf of the British and Irish Hypertension Society. *J Hum Hypertens*, 2019; 33(5): 349–351. doi: 10.1038/s41371-019-0196-9. Erratum in: *J Hum Hypertens*, 2020; 34(3): 259. doi: 10.1038/s41371-020-0316-6
4. Wiesel J, Fitzig L, Herschman Y, Messineo FC. Detection of atrial fibrillation using a modified microlife blood pressure monitor. *Am J Hypertens*, 2009; 22(8): 848–852. doi: 10.1038/ajh.2009.98
5. Pucci G, Santoni E, Bisogni V, et al. Screening for atrial fibrillation during automated blood pressure measurement among patients admitted to internal medicine ward. *Intern Emerg Med*, 2021; 16(6): 1605–1611. doi: 10.1007/s11739-021-02691-2
6. Proesmans T, Mortelmans C, Van Haelst R, et al. Mobile phone-based use of the photoplethysmography technique to detect atrial fibrillation in primary care: diagnostic accuracy study of the FibrCheck App. *JMIR Mhealth Uhealth*, 2019; 7(3): e12284. doi: 10.2196/12284
7. Chan PH, Wong CK, Poh YC, et al. Diagnostic performance of a smartphone-based photoplethysmographic application for atrial fibrillation screening in a primary care setting. *J Am Heart Assoc*, 2016; 5(7): e003428. doi: 10.1161/JAHA.116.003428
8. Lau JK, Lowres N, Neubeck L, et al. iPhone ECG application for community screening to detect silent atrial fibrillation: a novel technology to prevent stroke. *Int J Cardiol*, 2013; 165(1): 193–194. doi: 10.1016/j.ijcard.2013.01.220
9. <https://www.beurer.com/pl/p/45415/> (access: 5.02.2025)
10. Brito R, Mondouagne LP, Stettler C, et al. Automatic atrial fibrillation and flutter detection by a handheld ECG recorder, and utility of sequential finger and precordial recordings. *J Electrocardiol*, 2018; 51(6): 1135–1140. doi: 10.1016/j.jelectrocard.2018.10.093
11. Chon KH, McManus DD. Detection of atrial fibrillation using a smartwatch. *Nat Rev Cardiol*, 2018; 15(11): 657–658. doi: 10.1038/s41569-018-0057-1.
12. Bumgarner JM, Lambert CT, Hussein AA, et al. Smartwatch algorithm for automated detection of atrial fibrillation. *J Am Coll Cardiol*, 2018; 71(21): 2381–2388. doi: 10.1016/j.jacc.2018.03.003
13. <https://kardia-mobile.pl/dla-pacjenta/#jakzrobicbadanie> (access: 12.02.2025)
14. Koltowski L, Balsam P, Glowczynska R, et al. Kardia Mobile applicability in clinical practice: A comparison of Kardia Mobile and standard 12-lead electrocardiogram records in 100 consecutive patients of a tertiary cardiovascular care center. *Cardiol J*, 2021; 28(4): 543–548. doi: 10.5603/CJ.a2019.0001
15. Klier K, Koch L, Graf L, et al. Diagnostic accuracy of single-lead electrocardiograms using the Kardia Mobile app and the Apple Watch 4: validation study. *JMIR Cardio*, 2023; 7: e50701. doi: 10.2196/50701
16. Shahid S, Iqbal M, Saeed H, et al. Diagnostic accuracy of Apple Watch electrocardiogram for atrial fibrillation: a systematic review and meta-analysis. *JACC Adv*, 2025; 4(2): 101538. doi: 10.1016/j.jacadv.2024.101538
17. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham Study. *Stroke*. 1991; 22(8): 983–988. doi: 10.1161/01.str.22.8.983
18. Feldman K, Duncan RG, Nguyen A, et al. Will Apple devices' passive atrial fibrillation detection prevent strokes? Estimating the proportion of high-risk actionable patients with real-world user data. *J Am Med Inform Assoc*, 2022; 29(6): 1040–1049. doi: 10.1093/jamia/ocac009
19. Sunaga A, Tanaka N, Egami Y, et al.; OCVC²Arrhythmia Investigators. Novel anticoagulation therapy using apple watch after catheter ablation for atrial fibrillation-Up to AF trial: design and rationale. *J Arrhythm*, 2024; 41(1): e13194. doi: 10.1002/joa3.13194



THE EVOLVING ROLE OF ARTIFICIAL INTELLIGENCE IN ORTHODONTIC DIAGNOSIS AND TREATMENT PLANNING

Ewolucyjna rola sztucznej inteligencji w diagnostyce i planowaniu leczenia ortodontycznego



Konstancja Nosowicz¹, Agata Tuczyńska², Patrycja Różak¹, Adam Łuczak¹, Adrianna Gliszczyńska¹, Nina Szczepańska¹

1. *Military Specialist Outpatient Clinic, Independent Public Healthcare Centre in Poznań, Szylinga Branch, Poland*
2. *Department and Clinic of Maxillofacial Orthopaedics and Orthodontics, Poznan University of Medical Sciences, Poland*

Konstancja Nosowicz – 0009-0000-5080-1509
 Agata Tuczyńska – 0000-0003-1648-6765
 Patrycja Różak – 0009-0004-2643-8817
 Adam Łuczak – 0009-0004-1848-5437
 Adrianna Gliszczyńska – 0009-0009-1721-5327
 Nina Szczepańska – 0009-0003-7448-4466

Abstract

Artificial intelligence is increasingly transforming the field of orthodontics by enhancing diagnostic precision, optimising treatment planning, and improving patient outcomes. This review explores current applications, benefits, limitations, and future prospects of artificial intelligence in orthodontic practice. By examining recent technological advancements and clinical implementations, this article aims to provide a comprehensive understanding of how artificial intelligence is reshaping contemporary orthodontics.

Streszczenie

Sztuczna inteligencja coraz bardziej rewolucjonizuje dziedzinę ortodoncji, zwiększając precyzję diagnostyczną, optymalizując planowanie leczenia i poprawiając wyniki terapeutyczne. Niniejsza praca przeglądowa przedstawia aktualne zastosowania, korzyści, ograniczenia oraz przyszłe perspektywy wykorzystania sztucznej inteligencji w praktyce ortodontycznej. Artykuł ma na celu wyjaśnienie, na podstawie analizy najnowszych osiągnięć technologicznych i zastosowań klinicznych, w jaki sposób sztuczna inteligencja zmienia współczesną ortodoncję.

Keywords: orthodontics; artificial intelligence

Słowa kluczowe: ortodoncja; sztuczna inteligencja

DOI 10.53301/lw/208277

Received: 10.06.2025

Accepted: 15.07.2025

Published: 31.03.2026

Corresponding author:

Konstancja Nosowicz
 Military Specialist Outpatient Clinic,
 Independent Public Healthcare Centre in Poznań,
 Szylinga Branch, Poznań
 e-mail: konstancjanosowicz@gmail.com

Introduction

The integration of artificial intelligence (AI) into healthcare represents one of the most significant technological achievements of the 21st century. Across various medical specialties, AI enhances diagnostic accuracy, streamlines workflows, and supports more informed clinical decision-making [1]. In orthodontics, the adoption of AI has been gaining momentum due to the central role of imaging, pattern analysis, and personalised treatment

planning [2]. The digital transformation of orthodontic records, including the widespread use of cone beam computed tomography (CBCT), intraoral scans, and cephalometric radiographs, has created favourable conditions for the introduction and application of AI algorithms.

AI encompasses a broad range of technologies, including machine learning (ML), deep learning (DL), and convolutional neural networks (CNNs), which are capable of analysing large and complex datasets to identify patterns

that may be imperceptible to clinicians [2, 3]. These technologies are used to automate time-consuming tasks such as outlining anatomical structures in cephalometry [4], improving the accuracy of skeletal assessment [5], and generating insights that support treatment planning.

Neural network learning involves a multi-stage optimisation process in which the model learns by analysing vast datasets. In deep networks, each layer transforms data into increasingly abstract representations, ultimately producing a diagnostic output. A key stage of this process is the training phase, during which the model adjusts its parameters in response to errors.

In this context, the role of specialist clinicians is crucial. In addition to supplying high-quality training data (e.g. manually annotated cephalometric landmarks, clinical case descriptions), they act as validators and reviewers of algorithm performance. Expert clinical knowledge is essential at every stage of developing AI systems: from preparing datasets to evaluating the accuracy and practical utility of models. Specialist clinicians also play a key role in identifying model limitations, improving result interpretability, and ensuring that AI tools align with real clinical needs [6].

As orthodontics continues to evolve, AI offers promising opportunities to enhance the quality of patient care, shorten appointment times, and achieve better treatment outcomes [1]. However, effective integration into orthodontic practice also requires consideration of ethical, regulatory, and practical challenges [7].

Aim of study

The aim of this article is to review the available literature on the evolving role of artificial intelligence in orthodontics, with particular emphasis on its current applications, benefits, limitations, and future development prospects.

Evolution and fundamental concepts of artificial intelligence in orthodontics

Artificial intelligence in healthcare refers to the simulation of human intelligence by machines, particularly computer systems capable of learning from data, recognising patterns, and making decisions. In orthodontics, AI relies primarily on machine learning, which enables systems to learn from experience without explicit programming, and on deep learning, which uses artificial neural networks to model complex patterns in data. Convolutional neural networks, in particular, have proven highly effective in image analysis and are widely used in orthodontic diagnostics based on radiographic imaging [2].

These technologies have enabled a shift from manual, time-consuming procedures to digitally assisted, automated systems. From predicting malocclusions to designing customised orthodontic appliances, AI supports orthodontists in making more informed and precise decisions [2, 3].

Applications in diagnostic imaging

Orthodontics relies heavily on imaging techniques such as cephalometric radiographs, panoramic radiographs,

and CBCT scans. AI plays a crucial role in streamlining the analysis of these modalities.

Cephalometric analysis is the cornerstone of orthodontic diagnostics and treatment planning. Traditionally, the identification of anatomic landmarks on cephalometric radiographs has been a manual process, characterised by variability both between different clinicians and within the same clinician at different times. AI, particularly deep learning models such as convolutional neural networks, can automate this process with high precision and reproducibility [4]. These systems can detect and annotate landmarks faster than experts, increasing workflow efficiency and diagnostic consistency while maintaining accuracy. A controlled study showed that AI was 2.12% more accurate and 95% faster than specialists [8].

Beyond landmark detection, AI models can perform segmentation, i.e. identification and extraction of specific anatomical structures such as teeth, the maxilla, the mandible, or the airway. This enables automated measurements, anomaly detection, and morphological classification. For example, CNNs are effectively applied to the segmentation of CBCT scans and the detection of dental caries or root resorption, increasing the objectivity and efficiency of image interpretation [9].

Enhancing treatment planning and monitoring

The integration of AI into orthodontic workflows has revolutionised the way clinicians plan, execute, and oversee treatment. Thanks to the use of large datasets and advanced algorithms, AI-based tools can enhance the precision, efficiency, and individualisation of orthodontic care.

One of the most groundbreaking applications of AI in orthodontics is predictive modelling. Machine learning algorithms, trained on large datasets from historical cases, are capable of forecasting treatment outcomes based on variables such as skeletal structure, the specific type of malocclusion, and the patient's age. Predictive tools enable orthodontists to:

- simulate multiple possible treatment pathways, helping select the most effective strategy;
- estimate treatment duration and the probability of complications;
- predict skeletal and dental growth in younger patients.

Such solutions are particularly valuable in complex cases requiring orthognathic surgery or interdisciplinary collaboration (e.g. with a periodontist or prosthodontist), where AI-based simulations can support planning by forecasting post-treatment occlusion and facial aesthetics [10].

AI has significantly streamlined the orthodontic appliance design process through integration with CAD/CAM systems. By utilising intraoral scans or CBCT (Cone Beam Computed Tomography), AI-based platforms can automatically:

- analyse tooth morphology and alignment;
- design custom-fitted archwires, brackets, and aligners;
- simulate optimal force vectors and tooth movements.

In the case of aligner therapy, AI can determine the optimal sequence and magnitude of movements necessary to achieve therapeutic goals, while minimising patient discomfort and the risk of material fatigue. Automation of appliance design not only increases the precision of orthodontic appliances but also shortens chair time and reduces human error – an important advantage in high-volume clinical settings [11].

Continuous treatment monitoring is vital for success. AI-based tools now allow for the assessment of treatment progress in real-time or at regular intervals through:

- sequential photographic analysis – AI compares facial or intraoral images taken at different treatment stages;
- cephalometric superimposition – AI automatically overlays anatomical landmarks from radiographs to evaluate skeletal and dental changes;
- 3D model comparison – periodic intraoral scans can be analysed for accuracy of tooth positioning and arch movements.

These tools are especially useful in aligner therapy, where patient compliance is critical. AI algorithms can detect and flag insufficient aligner wear or unexpected tooth movements, enabling early intervention and reducing the risk of relapse [12].

Applications of AI in orthodontic diagnostics

Artificial intelligence has significantly improved cephalometric analysis by automating the identification of anatomical landmarks on radiographic images. This has reduced human error and enhanced diagnostic efficiency. Platforms such as WeDoCeph, WebCeph, and CephX use deep-learning algorithms, particularly CNNs, to detect cephalometric landmarks with high accuracy [11, 13].

Studies have shown that these systems generate results comparable to those of experienced clinicians – often within seconds of image upload. The integration of these tools into orthodontic practice allows for faster diagnosis and reproducible landmark identification, which is crucial for precise treatment planning [11, 13, 14].

AI is increasingly used to assess skeletal maturity, which is essential for determining the optimal timing of intervention in growing patients. AI models trained on radiographic images, such as lateral cephalograms or hand-wrist radiographs, can estimate cervical vertebral maturation (CVM) stages and Fishman's skeletal maturity indicators (SMI).

Studies have demonstrated that AI systems achieve high levels of agreement with expert assessments in CVM classification. These tools assist clinicians in selecting the appropriate window for treatment commencement, particularly in growth-modification therapies [15–17].

Artificial intelligence is also applied in the analysis of the upper airway and temporomandibular joint (TMJ) structures using CBCT and magnetic resonance imaging (MRI). These systems support the diagnosis of airway obstruction, such as obstructive sleep apnoea, by segment-

ing and measuring the volume of the nasopharyngeal and oropharyngeal spaces [18, 19].

In TMJ diagnostics, AI systems classify disc displacement, identify degenerative changes, and analyse the morphology of the mandibular condyle based on imaging data, providing a faster and more standardised assessment than manual methods [20].

Clinical outcomes and effectiveness

Artificial intelligence has significantly improved the accuracy of orthodontic diagnosis and treatment planning. Using machine learning algorithms and deep learning models, AI systems are able to analyse complex datasets, resulting in more accurate identification of cephalometric landmarks and improved assessment of malocclusions. A randomised controlled trial demonstrated that AI-assisted diagnostics achieved higher precision in treatment planning compared with traditional methods, with a statistically significant improvement ($p < 0.05$) [10]. The integration of AI into orthodontic practice has also led to shorter treatment durations and a reduced number of appointments. In the study, the mean treatment duration for patients in the AI-assisted group was 14.6 ± 3.2 months, compared to 18.9 ± 4.5 months in the group without AI support ($p < 0.001$). Additionally, the AI group required fewer appointments (10.2 ± 2.1) than the traditional group (12.8 ± 3.4) [21].

Patients undergoing AI-assisted orthodontic treatment reported higher levels of satisfaction. A randomised controlled trial showed that the AI group achieved a mean satisfaction score of 9.2 ± 0.6 , compared with 8.1 ± 0.8 in the traditional group ($p < 0.001$). Factors contributing to the increased satisfaction included shorter treatment duration, fewer appointments, and improved treatment outcomes [21].

Challenges and limitations

Despite the growing potential of AI in orthodontics, several significant challenges remain. The integration of AI into orthodontic practice necessitates the collection and analysis of extensive documentation and patient data, including radiographs, 3D intraoral scans, and clinical treatment records. This raises serious concerns regarding patient privacy and data security. It is essential to comply with strict regulatory frameworks, such as the General Data Protection Regulation (GDPR) in Europe and the Health Insurance Portability and Accountability Act (HIPAA) in the United States. Orthodontic practices must implement advanced cybersecurity measures and data anonymisation techniques to protect sensitive information [22].

The use of AI in orthodontic diagnostics also raises questions regarding the determination of liability for the results obtained. Despite the increasing autonomy of algorithms, there is still a lack of clear legal regulations concerning liability for potential diagnostic errors or incorrect clinical decisions made based on AI suggestions. In practice, the specialist remains responsible for the entire course of treatment, regardless of whether AI-assist-

ed tools were used. This legal framework reflects the view that AI serves a supportive rather than a decision-making role, and that the final interpretation of data and therapeutic decisions rests with the clinician. Nevertheless, as technology continues to advance, it will be necessary to refine the legal and ethical frameworks to account for the specific nature of algorithmic recommendations and their impact on the treatment process [23].

AI models are only as unbiased as the data on which they were trained. If training datasets lack diversity or exhibit bias toward specific demographic groups, the resulting AI systems may perpetuate existing prejudices, leading to disparities in treatment outcomes. For example, the underrepresentation of certain ethnic groups in training datasets can result in reduced AI efficacy for these populations, thereby exacerbating health disparities. Addressing algorithmic bias requires the inclusion of diverse and representative datasets in the development of AI systems [24].

Despite significant advances of AI in orthodontics, this technology cannot replace the clinical experience of orthodontists. While AI systems excel at pattern recognition and data analysis, they are unable to account for patient preferences, psychosocial factors, or ethical considerations. Therefore, human oversight remains indispensable to interpret AI recommendations, make informed clinical decisions, and provide personalised care [25].

Future and perspectives

The integration of AI with other cutting-edge technologies, such as 3D printing and Virtual Reality (VR), has the potential to further personalise orthodontic treatment. AI algorithms can analyse vast amounts of data from previous cases to predict the most effective treatment plans for new patients. 3D printing enables the rapid production of customised orthodontic appliances, reducing manufacturing time while improving fit and comfort [26]. Moreover, VR technology can be used in patient education, in increasing patient awareness, and in treatment planning. The visualisation of tooth movements and anticipated treatment outcomes allows patients to better understand the course of therapy, which enhances satisfaction and cooperation. For clinicians, VR offers immersive simulations for training and planning complex procedures, which may improve clinical outcomes [27].

The development of AI systems capable of continuous learning is essential for increasing their clinical usefulness. Traditional AI models are static, meaning they are trained on fixed datasets and may not adapt well to new information or evolving clinical practices. Systems capable of continuous learning can adapt to new data in real time, improving their performance and relevance in dynamic clinical environments [12].

Implementing such systems requires rigorous quality-control mechanisms to monitor algorithm behaviour and prevent the incorporation of erroneous data. Moreover, their integration demands careful planning to ensure patient safety and data integrity [25].

Conclusions

Artificial intelligence is redefining orthodontics by providing tools that enhance diagnostic accuracy, streamline treatment planning, and support personalised patient care. Through innovations such as automated cephalometric analysis, predictive modelling, and intelligent orthodontic appliance design, AI enables clinicians to make faster and more informed decisions, ultimately improving efficiency and treatment outcomes.

Integration with complementary technologies such as 3D printing and virtual reality may further revolutionise workflow organisation in orthodontics, enhancing treatment precision and supporting a more personalised approach to patient care.

However, to fully harness the potential of AI in orthodontic practice, the existing challenges must be addressed. Data-privacy concerns must be resolved through strict adherence to regulations such as GDPR and HIPAA, as well as the implementation of advanced cybersecurity protocols. Also, algorithmic bias remains a significant concern, underscoring the need for diverse and representative training datasets to ensure equitable care for all patient groups. Equally important is continuous human oversight, as AI systems are not yet capable of replacing the clinical experience, ethical reasoning, and individualised patient approach provided by skilled orthodontists.

As AI technologies continue to advance, fostering interdisciplinary collaboration among clinicians, data scientists, and regulatory experts will be essential. Such collaboration will be key to integrating AI in an ethical, safe, and effective manner, opening the door to a new era of precise, personalised orthodontic care.

References

1. Kazimierczak N, Kazimierczak W, Serafin Z, et al. AI in orthodontics: revolutionizing diagnostics and treatment planning – a comprehensive review. *J Clin Med*, 2024; 13(2): 344. doi: 10.3390/jcm13020344
2. Monill-González A, Rovira-Calatayud L, d'Oliveira NG, Ustrell-Torrent JM. Artificial intelligence in orthodontics: Where are we now? A scoping review. *Orthod Craniofac Res*, 2021; 24 Suppl 2: 6–15. doi: 10.1111/ocr.12517
3. Topol EJ. High-performance medicine: the convergence of human and artificial intelligence. *Nature Medicine*, 2019; 25(1): 44–56. doi: 10.1038/s41591-018-0300-7
4. Lee Y, Pyeon J-H, Han S-H, et al. A comparative study of deep learning and manual methods for identifying anatomical landmarks through cephalometry and cone-beam computed tomography: a systematic review and meta-analysis. *Appl Sci*, 2024; 14: 7342. doi: 10.3390/app14167342
5. Kim HJ, Kim KD, Kim DH. Deep convolutional neural network-based skeletal classification of cephalometric image compared with automated-tracing software. *Sci Rep*, 2022; 12: 11659. doi: 10.1038/s41598-022-15856-6
6. Budd S, Robinson EC, Kainz B. A survey on active learning and human-in-the-loop deep learning for medical image analysis. *Med Image Anal*, 2021; 71: 102062. doi: 10.1016/j.media.2021.102062

7. Leonardi R, Vaid N. Artificial intelligence in orthodontics: concerns, conjectures, and ethical dilemmas. *Int Dent J*, 2025; 75(1): 20–22. doi: 10.1016/j.identj.2024.11.002
8. Blum FMS, Möhlhenrich SC, Raith S, et al. Evaluation of an artificial intelligence-based algorithm for automated localization of craniofacial landmarks. *Clinical Oral Investigations*, 2023; 27(5): 2255–2265. doi: 10.1007/s00784-023-04978-4
9. Lee JH, Kim DH, Jeong SN, Choi SH. Detection and diagnosis of dental caries using a deep learning-based convolutional neural network algorithm. *J Dent*, 2018; 77: 106–111. doi: 10.1016/j.jdent.2018.07.015
10. Li P, Kong D, Tang T, et al. Orthodontic treatment planning based on artificial neural networks. *Sci Rep*, 2019; 9: 2037. doi: 10.1038/s41598-018-38439-w
11. Pandey R, Kamble R, Kanani H. Revolutionizing smiles: advancing orthodontics through digital innovation. *Cureus*, 2024; 16(7): e64086. doi: 10.7759/cureus.64086
12. Davis SE, Embí PJ, Matheny ME. Sustainable deployment of clinical prediction tools – a 360° approach to model maintenance. *J Am Med Inform Assoc*, 2024; 31(5): 1195–1198. doi: 10.1093/jamia/ocae036
13. Mercier JP, Rossi C, Sanchez IN, et al. Reliability and accuracy of artificial intelligence-based software for cephalometric diagnosis. A diagnostic study. *BMC Oral Health*, 2024; 24: 1309. doi: 10.1186/s12903-024-05097-6
14. Leonardi R, Giordano D, Maiorana F, Spampinato C. Automatic cephalometric analysis. *Angle Orthod*, 2008; 78(1): 145–151. doi: 10.2319/120506-491.1
15. Kazmierczak W, Jedliński M, Issa J, et al. Accuracy of artificial intelligence for cervical vertebral maturation assessment – a systematic review. *J Clin Med*, 2024; 13: 4047. doi: 10.3390/jcm13144047
16. Kim DW, Kim J, Kim T, et al. Prediction of hand-wrist maturation stages based on cervical vertebrae images using artificial intelligence. *Orthod Craniofac Res*, 2021; 24 Suppl 2: 68–75. doi: 10.1111/ocr.12514
17. Sadeghi TS, Ourang SA, Sohrabniya F, et al. Performance of artificial intelligence on cervical vertebral maturation assessment: a systematic review and meta-analysis. *BMC Oral Health*, 2025; 25(1): 187. doi: 10.1186/s12903-025-05482-9
18. Orhan K, Shamshiev M, Ezhov M, et al. AI-based automatic segmentation of craniomaxillofacial anatomy from CBCT scans for automatic detection of pharyngeal airway evaluations in OSA patients. *Sci Rep*, 2022; 12(1): 11863. doi: 10.1038/s41598-022-15920-1
19. Jeong Y, Nang Y, Zhao Z. Automated evaluation of upper airway obstruction based on deep learning. *Biomed Res Int*, 2023; 2023: 8231425. doi: 10.1155/2023/8231425
20. Manek M, Maital, Bezerra Silva DF, et al. Temporomandibular joint assessment in MRI images using artificial intelligence tools: where are we now? A systematic review. *Dentomaxillofac Radiol*, 2025; 54(1): 1–11. doi: 10.1093/dmfr/twae055
21. Gaonkar P, Mohammed I, Ribin M, et al. Assessing the impact of AI-enhanced diagnostic tools on the treatment planning of orthodontic cases: An RCT. *J Pharm Bioallied Sci*, 2024; 16(Suppl 2): S1798–S1800. doi: 10.4103/jpbs.jpbs_1147_23
22. Gala K. Ethical and legal considerations in AI-driven health cybersecurity. *International Journal of Scientific Research in Computer Science, Engineering and Information Technology*, 2024; 10: 682–690. doi: 10.32628/CSEIT241051059
23. Roganović J, Radenković M, Miličić B. Responsible use of artificial intelligence in dentistry: survey on dentists' and final-year undergraduates' perspectives. *Healthcare (Basel, Switzerland)*, 2023; 11(10): 1480. doi: 10.3390/healthcare11101480
24. Allareddy V, Oubaidin M, Rampa S, et al. Call for algorithmic fairness to mitigate amplification of racial biases in artificial intelligence models used in orthodontics and craniofacial health. *Orthod Craniofac Res*, 2023; 26 (Suppl 1): 124–130. doi: 10.1111/ocr.12721
25. Fawaz P, El Sayegh P, Vande Vannet B. Artificial intelligence in revolutionizing orthodontic practice. *World J Methodol*, 2025; 15(3): 100598. doi: 10.5662/wjm.v15.i3.100598
26. Khan MI, Laxmikanth SM, Gopal T, Neela PK. Artificial intelligence and 3D printing technology in orthodontics: future and scope. *AIMS Biophysics*, 2022; 9: 182–197. doi: 10.3934/biophy.2022016
27. Huang Y, Cheng X, Chan U, et al. Virtual reality approach for orthodontic education at School of Stomatology, Jinan University. *J Dent Educ*, 2022; 86(8):1025–1035. doi: 10.1002/jdd.12915



THE EFFECT OF GARLIC ON CARBOHYDRATE METABOLISM AND GLYCAEMIC CONTROL: A REVIEW OF THE LITERATURE

Wpływ czosnku na metabolizm węglowodanów i kontrolę glikemii: przegląd literatury



Dominik Ireneusz Kret, Wiktoria Klaudia Szlachta, Antoni Anczyk, Karolina Handzel, Grzegorz Zaborowski, Jacek Wysoczański, Wiktor Kruczek, Filip Basta, Miłosz Tworek

Faculty of Medical Sciences in Katowice, Medical University of Silesia in Katowice, Poland

Dominik Ireneusz Kret – 0009-0007-6218-027X
 Wiktoria Klaudia Szlachta – 0009-0009-3739-4211
 Antoni Anczyk – 0009-0008-3817-3675
 Karolina Handzel – 0009-0004-5765-0458
 Grzegorz Zaborowski – 0009-0004-6977-7616
 Jacek Wysoczański – 0009-0005-1983-486X
 Wiktor Kruczek – 0009-0008-5193-2074
 Filip Basta – 0009-0008-4685-7778
 Miłosz Tworek – 0009-0000-5202-9364

Abstract

Aim of the paper: To present a systematic review of the scientific literature on the hypoglycaemic effects of garlic (*Allium sativum* L.), with a focus on its mechanisms of action, clinical efficiency, and potential adverse effects. **Materials and methods:** Publications from 2015 to 2024 available in the PubMed, Scopus, and Web of Science databases were analysed. These included randomised clinical trials, meta-analyses, and systematic reviews. **Results:** The collected data suggest that garlic supplementation, particularly in its natural form, can significantly reduce fasting blood glucose levels, with more pronounced effects observed over longer intervention periods. Some studies also report positive effects on glycated haemoglobin, postprandial glucose, and fructosamine levels. The proposed mechanisms of action include increased insulin secretion, improved tissue sensitivity to insulin, and modulation of oxidative and inflammatory processes. **Conclusions:** The evidence indicates that garlic has considerable potential as a natural adjunct in the prevention and treatment of metabolic disorders. However, further large-scale clinical trials are required to conclusively confirm its efficacy and safety.

Streszczenie

Cel badania: Celem niniejszego badania jest przegląd systematyczny aktualnej literatury naukowej dotyczącej hipoglikemicznego działania czosnku (*Allium sativum* L.), ze szczególnym uwzględnieniem mechanizmów jego działania, skuteczności klinicznej i możliwych działań niepożądanych. **Materiały i metody:** Przeanalizowano publikacje z lat 2015–2024 dostępne w bazach danych PubMed, Scopus i Web of Science. Obejmowały one randomizowane badania kliniczne, przeglądy systematyczne i metaanalizy. **Wyniki:** Zebrane dane wskazują, że suplementacja czosnkiem, zwłaszcza w jego naturalnej postaci, znacznie obniża poziom glukozy we krwi na czczo, a efekt ten jest tym bardziej wyraźny, im dłuższy jest czas trwania interwencji. Niektóre badania wskazują również na pozytywny wpływ na poziom hemoglobiny glikowanej, glikemii poposiłkowej i fruktozaminy. Mechanizmy działania czosnku obejmują między innymi zwiększone wydzielanie insuliny, poprawę wrażliwości tkanek na insulinę oraz modulację procesów oksydacyjnych i zapalnych. **Wnioski:** Zebrane dowody wskazują na znaczny potencjał czosnku jako naturalnego środka wspomagającego profilaktykę i leczenie zaburzeń metabolicznych, jednak aby w pełni potwierdzić jego skuteczność i bezpieczeństwo, konieczne są dalsze szeroko zakrojone badania kliniczne.

Keywords: diabetes; hyperglycaemia; garlic; *Allium sativum*; fasting blood glucose

Słowa kluczowe: cukrzyca; hiperglikemia; czosnek; *Allium sativum*; glikemia na czczo

DOI 10.53301/lw/213374

Received: 07.10.2025

Accepted: 20.10.2025

Published: 31.03.2026

Corresponding author:

Dominik Ireneusz Kret
 Faculty of Medical Sciences in Katowice,
 Medical University of Silesia in Katowice
 15 Poniatowskiego Str., 40-055 Katowice
 e-mail: kdominik2000@o2.pl

Introduction

Diabetes is a condition that is becoming increasingly common in both older and younger populations, making it a challenge faced by physicians across multiple specialties, not only because of the disease itself, but also due to the complications it causes. According to the WHO, in the 1980s, there were nearly 108 million people worldwide living with this disease. In 2021, the number reached 537 million, and forecasts estimate that by 2030, the total number of diabetics will exceed 640 million. Diabetes is one of the world's leading causes of death, and its increasing prevalence shows how serious the problem already is, and what we will be facing in the future [1].

Therefore, effective measures to support diabetes treatment are continually being sought. One substance with documented hypoglycaemic properties is *Allium sativum* L., commonly known as garlic. This spice, which has been known for centuries, is valued in many countries and cultures not only for its flavour but also for its medicinal properties, for example in the traditional treatment of colds. The aim of this study was to analyse the available literature on the effects of garlic on glucose control, discuss the mechanisms by which its active substances regulate blood glucose levels, and highlight potential side effects.

Chemical composition of garlic

Garlic contains a wide range of bioactive components, many of which have significant health-promoting benefits, including hypoglycaemic, anti-inflammatory, and anticancer properties. Garlic is rich in phytochemicals, including sulphur-containing components formed by enzymatic changes when plant cells are damaged. The most important of these are:

- allicin,
- ajoene,
- alliin,
- S-allyl cysteine (SAC),
- diallyl trisulfide (DATS),
- diallyl disulfide (DADS).

These compounds affect glycaemic levels, improve tissue sensitivity to insulin, and protect pancreatic beta cells from oxidative stress [2, 3].

Garlic contains a variety of vitamins, especially B vitamins and vitamin C, as well as minerals such as selenium, phosphorus, and potassium. It also provides fructans, in-

cluding inulin, which acts as a prebiotic. Garlic also contains polyphenols and flavonoids, such as quercetin and kaempferol, which have antioxidant properties and can regulate endothelial function and ongoing inflammatory processes [4].

The diversity of the content of these compounds depends on the form of garlic preparation (raw, cooked, aged extract, dried powder), growing conditions, and storage duration, as a result of which these preparations have different biological activities [5].

Mechanisms of action

The effect of garlic on blood glucose levels has been the subject of numerous scientific studies, as it can affect these levels through various mechanisms (Tab. 1). These mechanisms are well documented in preclinical research, but their direct confirmation in human studies still requires further analysis.

Side effects

Despite its numerous health-promoting properties, including the hypoglycaemic potential discussed in this paper, as well as its beneficial effects on other components of metabolic syndrome [20–22], garlic may cause adverse effects. The most commonly reported include unpleasant odour from the mouth and skin, which is more pronounced when raw garlic is consumed compared with its heat-treated forms [23], and gastrointestinal symptoms such as heartburn, reflux, nausea, bloating, and diarrhoea [24, 25]. Some patients may also experience allergic reactions, including urticaria, angioedema, and shortness of breath [26]. In addition, garlic may enhance the effects of certain medications, including anticoagulants, thereby increasing the risk of bleeding [27], as well as antidiabetic drugs such as metformin or glibenclamide, allowing therapeutic effects to be achieved with lower doses of medication [28, 29]. Therefore, garlic should be used with caution in both combination therapy and monotherapy, taking into account potential interactions and adverse effects.

Summary

An analysis of the available literature indicates that garlic has significant hypoglycaemic potential, associated with multiple molecular processes that are the subject of scientific research (Tab. 2). Its effectiveness depends on the form and method of processing. Studies suggest that

Table 1. Effects of garlic on the body and the molecular mechanisms underlying these effects

Effect of garlic on the body	Mechanism of action
Increased secretion of endogenous insulin	• Inhibition of pancreatic beta cell apoptosis [6, 7]
Increased tissue sensitivity to insulin	• Activation of the PI3K/AKT/Nrf2-Keap1 insulin pathway • Increased expression of the GLUT4 glucose transporter [6, 8]
Protection of pancreatic beta cells	• Reduction of oxidative stress [9, 10]
Inhibition of insulin resistance development	• Modulation of TNF- α and IL-6 • Reduction of reactive oxygen species [5]
Reduction of intestinal glucose absorption	• Inhibition of α -glucosidase and α -amylase activity [11, 12]
Support for the development of beneficial intestinal bacteria (including <i>Bifidobacterium</i> , <i>Lactobacillus</i>)	• Impact on the gut-pancreas axis [13]

Table 2. Review of meta-analyses (2015–2024)

Meta-analysis	Studies included	Scope of analysis	Conclusions	Comments
2015 (Hou LQ et al.) [14]	7 RCTs (513 participants)	Effect of garlic on FBG, PPG, and HbA _{1c} levels	<ul style="list-style-type: none"> Garlic consumption statistically significantly reduces FBG levels 	Insufficient data on the effect on PPG (a statistically significant reduction was reported in only one study) and HbA _{1c} (two studies with inconsistent results) [15, 16]
2017 (Emami S et al.) [17]	10 RCTs	Effect of garlic on FBG, PPG, and HbA _{1c}	<ul style="list-style-type: none"> Garlic consumption statistically significantly reduces FBG levels, with a more pronounced effect in individuals with concomitant lipid disorders Garlic consumption may reduce PPG, but does not significantly affect HbA_{1c} levels 	Garlic in its natural form has been shown to be more effective than supplements
2017 (Wang J et al.) [18]	9 RCTs (768 participants)	Effect of garlic on FBG, HbA _{1c} , and fructosamine levels	<ul style="list-style-type: none"> Garlic consumption statistically significantly reduces FBG levels Garlic significantly reduces fructosamine and HbA_{1c} levels (data reported in two studies) 	Five studies used garlic monotherapy compared to placebo, while the remainder used combination therapy with garlic and other hypoglycaemic drugs or insulin compared to a control group.
2023 (Fu Z et al.) [19]	19 RCTs (999 participants)	Effect of garlic on metabolic syndrome	<ul style="list-style-type: none"> Garlic mildly lowers FBG, but without statistical significance 	The effect of garlic on FBG was verified in only five studies, including 214 participants. Garlic significantly lowers TG, TC, LDL-C, DBP, BMI, and WC levels
2024 (Zhao X et al.) [2]	22 RCTs	Effect of garlic on FBG, HbA _{1c} , and lipid profile	<ul style="list-style-type: none"> Garlic statistically significantly lowers FBG levels (results from 8 RCTs) and causes a decrease in HbA_{1c} (results from 3 RCTs) 	Garlic significantly lowers TC and LDL-C levels, while increasing HDL-C levels. It does not significantly affect TG levels
RCT – randomised controlled trial; FBG – fasting blood glucose; PPG – postprandial glucose; glycated hemoglobin (HbA _{1c}) – haemoglobin A _{1c} ; TG – triglycerides; TC – total cholesterol; LDL-C – low density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; DBP – diastolic blood pressure; BMI – body mass index; WC – waist circumference				

fresh preparations have higher bioavailability and efficacy compared to supplements. It is also important to note that garlic, despite its natural origin, may cause side effects.

Available meta-analyses indicate that garlic can modulate carbohydrate metabolism, especially by lowering fasting blood glucose levels, and its hypoglycaemic effect is more pronounced when used in combination with anti-diabetic drugs than as monotherapy.

The effect of garlic on glycated hemoglobin levels is less consistent, although more recent meta-analyses indicate a reduction following long-term use. Some studies also suggest a beneficial effect on postprandial glucose and fructosamine levels, highlighting the need for further research in this area.

In summary, current scientific data suggest a potentially beneficial effect of garlic on glycaemic control, but the lack of methodological consistency and variability in available study results highlights the need for standardised, high-quality, multicentre clinical trials to more accurately assess its efficacy and safety.

References

- International Diabetes Federation. Diabetes now affects one in 10 adults worldwide [Internet]. Brussels: International Diabetes Federation; 2021 Nov 10 [cited 2025 Apr 22]. Available from: <https://idf.org/news/diabetes-now-affects-one-in-10-adults-worldwide/>
- Zhao X, Cheng T, Xia H, et al. Effects of Garlic on Glucose Parameters and Lipid Profile: A Systematic Review and Meta-Analysis on Randomized Controlled Trials. *Nutrients*, 2024; 16(11): 1692. doi: 10.3390/nu16111692
- Amagase H. Clarifying the real bioactive constituents of garlic. *J Nutr*, 2006; 136(3 Suppl): 716S–725S. doi: 10.1093/jn/136.3.716S
- Martins N, Petropoulos S, Ferreira IC. Chemical composition and bioactive compounds of garlic (*Allium sativum* L.) as affected by pre- and post-harvest conditions: A review. *Food Chem*, 2016; 211: 41–50. doi: 10.1016/j.foodchem.2016.05.029
- El-Saadony MT, Saad AM, Korma SA, et al. Garlic bioactive substances and their therapeutic applications for improving human health: a comprehensive review. *Front Immunol*, 2024; 15: 1277074. doi: 10.3389/fimmu.2024.1277074

6. Bhattacharya S, Maji U, Khan GA, et al. Antidiabetic role of a novel protein from garlic via NO in expression of Glut-4/insulin in liver of alloxan-induced diabetic mice. *Biomed Pharmacother*, 2019; 111: 1302–1314. doi: 10.1016/j.biopha.2019.01.036
7. Ali SI, Elkhalfi AME, Nabi SU, et al. Aged garlic extract preserves beta-cell functioning via modulation of nuclear factor kappa-B (NF- κ B)/Toll-like receptor (TLR)-4 and sarco endoplasmic reticulum calcium ATPase (SERCA)/Ca²⁺ in diabetes mellitus. *Diabetol Metab Syndr*, 2024; 16(1):110. doi: 10.1186/s13098-024-01350-8
8. Padiya R, Chowdhury D, Borkar R, et al. Garlic attenuates cardiac oxidative stress via activation of PI3K/AKT/Nrf2-Keap1 pathway in fructose-fed diabetic rat. *PLoS One*, 2014; 9(5): e94228. doi: 10.1371/journal.pone.0094228
9. Saravanan G, Ponnurugan P. Ameliorative potential of S-allyl cysteine on oxidative stress in STZ induced diabetic rats. *Chem Biol Interact*. 2011 Jan 15;189(1-2): 100–106. doi: 10.1016/j.cbi.2010.10.001
10. Padiya R, Khatua TN, Bagul PK, et al. Garlic improves insulin sensitivity and associated metabolic syndromes in fructose fed rats. *Nutr Metab (Lond)*, 2011; 8: 53. doi: 10.1186/1743-7075-8-53
11. Al-Malki AL. Inhibition of α -glucosidase by thiosulfinate as a target for glucose modulation in diabetic rats. *Evid Based Complement Alternat Med*, 2016; 2016: 7687915. doi: 10.1155/2016/7687915
12. Nickavar B, Yousefian N. Inhibitory effects of six allium species on α -amylase enzyme activity. *Iran J Pharm Res*, 2010; 8(1): 53–57. doi: 10.22037/ijpr.2010.788
13. Ha J, Kim J, Kim S, et al. Garlic-induced enhancement of Bifidobacterium: enterotype-specific modulation of gut microbiota and probiotic populations. *Microorganisms*, 2024; 12(10): 1971. doi: 10.3390/microorganisms12101971
14. Hou LQ, Liu YH, Zhang YY. Garlic intake lowers fasting blood glucose: meta-analysis of randomized controlled trials. *Asia Pac J Clin Nutr*, 2015; 24(4): 575–582. doi: 10.6133/apjcn.2015.24.4.15
15. Kumar R, Chhatwal S, Arora S, et al. Antihyperglycemic, antihyperlipidemic, anti-inflammatory and adenosine deaminase- lowering effects of garlic in patients with type 2 diabetes mellitus with obesity. *Diabetes Metab Syndr Obes*, 2013; 6: 49–56. doi: 10.2147/DMSO.S38888
16. Ashraf R, Khan RA, Ashraf I. Garlic (*Allium sativum*) supplementation with standard antidiabetic agent provides better diabetic control in type 2 diabetes patients. *Pak J Pharm Sci*, 2011; 24(4): 565–570
17. Emami, S., Rouhani, M. H., & Azadbakht, L. The effect of garlic intake on glycemic control in humans: a systematic review and meta-analysis. *Prog Nutr*, 2017; 19(1-S): 10–18. doi: 10.23751/pn.v19i1-S.5274
18. Wang J, Zhang X, Lan H, Wang W. Effect of garlic supplement in the management of type 2 diabetes mellitus (T2DM): a meta-analysis of randomized controlled trials. *Food Nutr Res*, 2017; 61(1): 1377571. doi: 10.1080/16546628.2017.1377571
19. Fu Z, Lv J, Gao X, et al. Effects of garlic supplementation on components of metabolic syndrome: a systematic review, meta-analysis, and meta-regression of randomized controlled trials. *BMC Complement Med Ther*, 2023; 23(1): 260. doi: 10.1186/s12906-023-04038-0
20. Bara-Ledesma N, Jimenez-Esteban J, Fabregate M, et al. Effect of encapsulated purple garlic oil on microvascular function and the components of metabolic syndrome: a randomized placebo-controlled study – the ENDOTALLIUM Study. *Nutrients*, 2024; 16(11): 1755. doi: 10.3390/nu16111755
21. Gyawali D, Vohra R, Orme-Johnson D, et al. A systematic review and meta-analysis of ayurvedic herbal preparations for hypercholesterolemia. *Medicina (Kaunas)*, 2021; 57(6): 546. doi: 10.3390/medicina57060546
22. Ried K. Garlic lowers blood pressure in hypertensive individuals, regulates serum cholesterol, and stimulates immunity: an updated meta-analysis and review. *J Nutr*, 2016; 146(2): 389S–396S. doi: 10.3945/jn.114.202192
23. Tamaki T, Sonoki S. Volatile sulfur compounds in human expiration after eating raw or heat-treated garlic. *J Nutr Sci Vitaminol (Tokyo)*, 1999; 45(2): 213–222. doi: 10.3177/jnsv.45.213
24. Ried K, Frank OR, Stocks NP. Aged garlic extract reduces blood pressure in hypertensives: a dose-response trial. *Eur J Clin Nutr*, 2013; 67(1): 64–70. doi: 10.1038/ejcn.2012.178
25. Borrelli F, Capasso R, Izzo AA. Garlic (*Allium sativum* L.): adverse effects and drug interactions in humans. *Mol Nutr Food Res*, 2007; 51(11): 1386–1397. doi: 10.1002/mnfr.200700072
26. Ma S, Yin J. Anaphylaxis induced by ingestion of raw garlic. *Foodborne Pathog Dis*, 2012; 9(8): 773–775. doi: 10.1089/fpd.2012.1133
27. Macan H, Uykimpang R, Alconcel M, et al. Aged garlic extract may be safe for patients on warfarin therapy. *J Nutr*, 2006; 136(3 Suppl): 793S–795S. doi: 10.1093/jn/136.3.793S
28. Gupta RC, Chang D, Nammi S, et al. Interactions between antidiabetic drugs and herbs: an overview of mechanisms of action and clinical implications. *Diabetol Metab Syndr*, 2017; 9: 59. doi: 10.1186/s13098-017-0254-9
29. Poonam T, Prakash GP, Kumar LV. Influence of *Allium sativum* extract on the hypoglycemic activity of glibenclamide: an approach to possible herb-drug interaction. *Drug Metabol Drug Interact*, 2013; 28(4): 225–230. doi: 10.1515/dmdi-2013-0031



MEDICAL SERVICES IN THE POLISH CAMPAIGN OF 1939 – A SYNTHESIS. PART II: PREPARATIONS AND WAR PLANS

Służba zdrowia w kampanii polskiej 1939 roku – synteza. Część II. Przygotowania i plany wojenne



Aleksander Rutkiewicz^{1,2}

1. Faculty of Health Sciences, University of Bielsko-Biała, Poland
2. Department of Anaesthesiology and Intensive Care, Beskid Oncology Centre, John Paul II Municipal Hospital in Bielsko-Biała, Poland

Aleksander Rutkiewicz – 0009-0000-8398-5818

Abstract

Introduction and objective: This research paper was created as part of the project “Polish Campaign of 1939 – Synthesis” conducted by the War Studies University. The aim of this three-part article is to provide a comprehensive account of the role of the Polish health service during the 1939 campaign. The subsequent sections examine the initial capabilities of the health service, wartime planning and preparations, and the actual participation of medical services during the campaign. The primary research question focuses on the factors that led to the collapse of Poland’s medical services in September 1939. **Material and methods:** The author drew on historical materials collected at the Central Military Archive (Warsaw-Rembertów) and the Archives of the Polish Institute and the General Władysław Sikorski Museum (London). This synthesis also incorporates previous research by historians, published sources, and a wide range of personal recollections and testimonies. **Results:** The Polish health service suffered a defeat in 1939. The system for evacuating and treating the wounded collapsed within the first days of the war. Evacuation difficulties, rapidly depleting stocks of prophylactic serums, and delays in surgical care led to widespread complications from infected wounds, including numerous cases of tetanus. **Conclusions:** The main factors that led to the medical crisis in 1939 included the very limited initial capacity of the medical service (staff shortages, underdeveloped hospitals), low financial investment and insufficient material reserves, the highly centralised medical supply system, reliance on railways for medical evacuation, the low degree of motorisation in both the Polish Army and society, poor road conditions, and the nature of the enemy’s operations – sudden, deep attacks on the rear, disruption of communication networks, and targeting of civilian areas.

Streszczenie

Wprowadzenie i cel: Praca powstała w ramach projektu „Kampania polska 1939 roku – synteza”, prowadzonego przez Akademię Sztuki Wojennej. Celem trzyczęściowego artykułu jest kompleksowe przedstawienie działań polskiej służby zdrowia w trakcie kampanii polskiej 1939 roku. W kolejnych częściach artykułu omówiono wyjściowy potencjał służby zdrowia, plany i przygotowania wojenne oraz udział służby zdrowia w kampanii wojennej. Podstawowe pytanie badawcze dotyczyło przyczyn załamania się systemu pomocy rannym we wrześniu 1939 roku. **Materiał i metody:** Podstawowy zasób źródeł, który posłużył do opracowania artykułu, znajduje się w Centralnym Archiwum Wojskowym (Warszawa-Rembertów) oraz w Archiwum Instytutu Polskiego i Muzeum im. gen. Władysława Sikorskiego (Londyn). W syntezie uwzględniono również wcześniejsze ustalenia historyków, źródła drukowane oraz szeroki wybór wspomnień i relacji. **Wyniki:** Polska służba zdrowia poniosła w 1939 roku klęskę. System ewakuacji i pomocy rannym w ciągu kilku pierwszych dni wojny uległ załamaniu. Problemy z ewakuacją, szybko wyczerpujące się zapasy surowic profilaktycznych oraz spóźniona pomoc chirurgiczna były powodem masowo stwierdzanych powikłań septycznych ran, w tym licznych przypadków tężca. **Wnioski:** Do najważniejszych przyczyn, które doprowadziły do katastrofy sanitarnej w 1939 roku, należą: bardzo skromny potencjał wyjściowy służby zdrowia (deficyt kadr, niski poziom szpitalnictwa), małe nakłady finansowe na służbę zdrowia oraz brak odpowiednich rezerw materiałowych, centralizacja systemu zaopatrzenia sanitarnego, wiodąca rola kolei w planach ewakuacji sanitarnej, niski stopień motoryzacji Wojska Polskiego oraz polskiego społeczeństwa, zły stan dróg, a także sposób prowadzenia walki przez wroga (gwałtowne i głębokie uderzenia na tyły polskiego państwa, porażenie sieci komunikacyjnych, atakowanie celów cywilnych).

Keywords: medical services; medical evacuation; Polish campaign of 1939

Słowa kluczowe: służba zdrowia; ewakuacja medyczna; kampania polska 1939

DOI 10.53301/lw/208276

Received: 05.05.2025

Accepted: 15.07.2025

Published: 31.03.2026

Corresponding author:

Aleksander Rutkiewicz

Faculty of Health Sciences,

University of Bielsko-Biała, Bielsko-Biała

e-mail: olorut@o2.pl

Introduction

This article continues the first part of the study, which comprehensively examined the capacity of the Polish healthcare system on the eve of the outbreak of World War II, including methodological considerations. The second part focuses entirely on the preparations and plans for mobilising and utilising the healthcare system in wartime conditions.

Tasks of the healthcare system during war

Any discussion of the organisation and operational planning of the healthcare system in wartime must begin by revisiting a fundamental question: what role are health services expected to play during armed conflict? Answers to this question can be found in numerous publications and manuals from the interwar period. In one such document – a 1933 manual titled “Zaopatrywanie i ewakuacja w ramach pułku piechoty” [“Supply and Evacuation within an Infantry Regiment” – we read: *The medical service ceases to be a mere adjunct to combat and becomes one of the most vital factors for victory; its fundamental operational basis and primary task is to maintain the army’s numerical strength through:*

- protecting the army’s health,
- treatment – namely, restoring to military service all those who have lost their capacity for duty, whether as a result of wounds, gas injuries, or illness [1].

The concept of “protecting the army’s health” encompasses broadly understood preventive measures, including the prevention of infectious diseases (vaccinations, isolation of the sick, sanitary supervision of water sources and food, etc.), as well as soldiers’ health education, periodic examinations, and ongoing medical care. Although, for obvious reasons, war shifts the focus of medical services toward treatment, prevention does not lose its importance. Quite the opposite. During military operations the risk of epidemics increases, wartime conditions are not conducive to hygiene, and soldiers are more likely to engage in risky behaviours, including sexual ones. The army, exposed to the hardships of combat and long marches, sleep deprivation, and adverse weather conditions, becomes vulnerable to disease, injuries, and psychophysical exhaustion.

With regard to treatment priorities, combat injuries were only ostensibly paramount. Statistics from the fronts of the First World War clearly demonstrated that the principal source of losses was not wounds but disease. However, the Polish Campaign of 1939 was brief, and the fighting was violent and dynamic; consequently, the treatment of combat injuries assumed primary importance.

If a wounded soldier did not die immediately from his injuries, he faced the risk of developing complications. Two fundamental clinical issues arise here: haemorrhage and infection. For the wounded to survive, appropriate countermeasures in both areas had to be taken at an early stage of treatment and evacuation. The limitations of medicine at the time (including the field of medical evacuation) meant that if a massive haemorrhage developed as a result of a wound, the patient had little chance of survival. In practice, transport to a field hospital for bleed-

ing casualties constituted a form of selection: survival depended largely on whether the haemorrhage was not immediately fatal.

Greater possibilities for surgical intervention existed in cases of wound infection. By its very nature, a combat wound is contaminated. Fragments of uniform and equipment, as well as debris from the skin surface, enter the wound together with the projectile or shrapnel. If not properly protected by a dressing, the wound is at risk of secondary contamination. This is particularly dangerous when soil – rich in bacteria – enters the wound. Among the most perilous are streptococcal infections and those caused by anaerobic bacteria, leading to gas gangrene and tetanus. World War I marked a turning point in the management of combat wounds. A certain standard of care was developed at that time. At this point, it is worth returning once again to the previously cited manual: *Bacteriological studies conducted during the Great War [World War I – author’s note] revealed that within 24 to 36 hours, the entire area surrounding a wound is already deeply infected. Consequently, after this period, surgical intervention produced poorer recovery outcomes. From this follow some of the main guidelines for the medical service, as well as for commanders who in any way participate in directing medical units or influence the regulation of their work, namely:*

- every wounded soldier should be brought as quickly as possible to a qualified surgeon,
- every wounded soldier should be operated on as soon as possible – no later than 24–36 hours after being wounded.

If, as a result of a large influx of wounded or considerable transport distances, it proved impossible to operate on all casualties within the above-mentioned time frame, initial wound debridement was to be performed. This procedure consisted in the excision of tissues crushed by the projectile and those that had become infected. With a properly applied dressing, the wound could remain stable for 4–5 days, by which time the casualty had to reach a qualified surgeon [1].

The above argument can be summarised as follows: for the wounded, rapid surgical intervention is critical. To make this objective achievable, the following are required:

- surgical personnel equipped with appropriate instruments and materials;
- an efficient evacuation system based on effective means of transport;
- established operational principles for the system, including the triage of the wounded;
- non-surgical personnel responsible for initial wound care, medical triage, and care during transport;
- coordination and command, enabling system management and a flexible response to the evolving situation on the front lines.

As it turned out, the Polish healthcare system was unprepared for war in most of the aforementioned areas.

Doctrinal principles of the medical service in the field

The principles governing the operation of the medical service under field conditions were set out in the “Regulamin służby zdrowia w polu” (“Regulations for Medical Service in the Field”), introduced for official use in 1929 and re-issued in 1932 [2]. The document was complemented by

a series of instructions addressing, among other matters, the operation of military medical facilities, the functioning of medical services within military units, the management of military medical facilities during wartime, and the organisation of hospital trains. Relevant guidance was also included in the quartermaster regulations [3–7].

The foundation on which the system of medical assistance in the field was based was the principle of staged evacuation and treatment, with triage of the wounded and sick carried out at every stage to determine evacuation and treatment priorities. With each successive echelon, the scope of medical intervention increased. The scheme envisaged evacuation from lower to higher levels by the medical formations of the next echelon (Fig. 1).

The overall medical operations of large units (infantry divisions and cavalry brigades) were the responsibility of the chief medical officer (head of the medical service). At the infantry division level, the principal medical sub-unit was the medical company; in a cavalry brigade, it was the mounted medical platoon. Infantry divisions were also to include a small field hospital, although hospitals were generally operational-level (army-level) formations.

First aid on the battlefield was provided by the medical patrols of infantry companies (or artillery batteries) or by cavalry squadron medics. Transport of the wounded to the battalion or regimental aid station (BAS/RAS)

took place on stretchers carried with special straps or on transport carts. In cavalry units, evacuation from the front line was conducted on a medic's horse or using special cavalry stretchers – a sheet stretched between horses. During assaults or under heavy enemy fire, so-called ‘casualty nests’ were organised, making use of natural terrain cover or shell craters. Deployment of the BAS/RAS was the responsibility of the infantry battalion or cavalry regiment physician. At the BAS, patients underwent their first medical examination. The physician established an initial diagnosis, implemented necessary treatment (checking or applying dressings, emergency haemorrhage control, immobilisation of fractures, administration of preventive sera and circulatory stimulants), issued a preliminary evacuation card, and made an initial decision regarding evacuation priority.

From the BAS/RAS, the wounded were evacuated to the Main Aid Station (MAS) of the infantry division or cavalry brigade. Transport was the responsibility of the medical company of the infantry division or the mounted medical platoon of the cavalry brigade. The wounded were picked up from the so-called ‘wagon station’ – the furthest point reachable by medical wagons. Wagon stations were not organised in cavalry units. At this stage, transport was carried out by horse-drawn wagons (Fig. 2).

The wounded reached the MAS either directly or through Advanced Aid Stations (AAS), which were also deployed by the medical company. At the MAS, comprehensive medical triage was performed, and decisions were made regarding further evacuation. It is worth noting that, within this medical evacuation system, the regimental level of infantry was largely bypassed. The Chief Physician of the regiment organised an aid station only in the event of a gas attack; his primary responsibilities were coordinating the battalion medical service and organising evacuation routes at the regimental level. The direction of evacuation was essentially determined by the route of

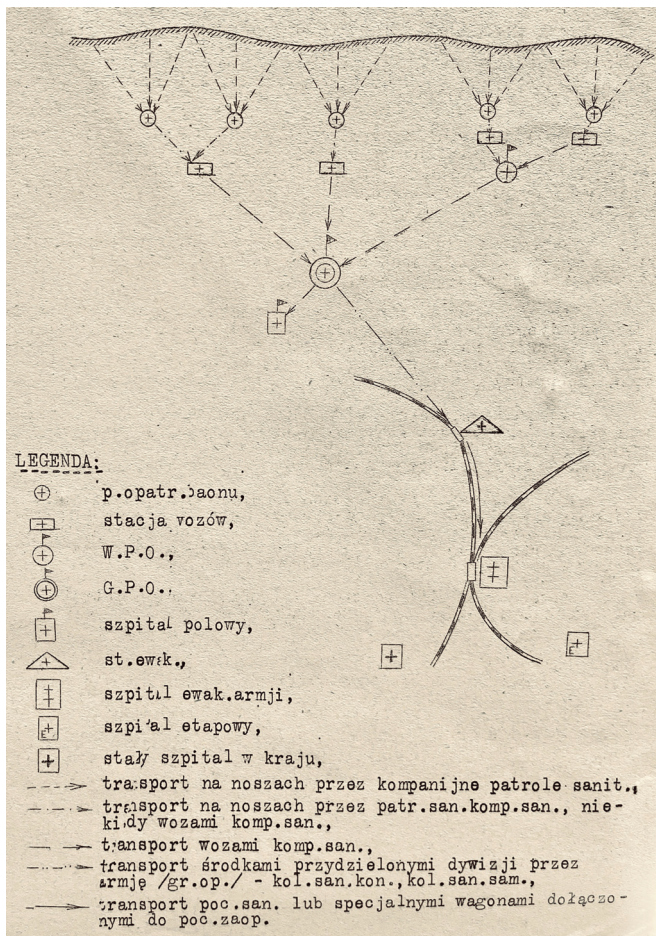


Figure 1. General scheme of medical evacuation. Source: “Zapopatrywanie i ewakuacja w ramach pułku piechoty” (“Supply and Evacuation within an Infantry Regiment”) [1]



Figure 2. The primary means of medical transport in the Polish Army at the tactical level in 1939 were horse-drawn vehicles, including simple supply wagons such as the wz. 19 (model 19) shown here. As part of the material mobilisation, peasant carts were also requisitioned; however, their technical condition very often left much to be desired. The photograph likely dates from the early 1930s. Source: National Digital Archives



Figure 3. The transport of the wounded at the operational level was to be carried out primarily by rail. Plans for mobilisation included assembling 36 permanent and semi-permanent hospital trains, which could be supplemented by improvised trains. The photograph shows one of the ambulance coaches at Poznań railway station. Source: National Digital Archives

the nearest railway line, where an evacuation station was established (Fig. 3 and Fig. 4).

The prevailing doctrine of the Polish Army assumed a manoeuvre-oriented character of combat. The organisational structure and operating principles of field ser-



Figure 4. Interior of the dressing and treatment room of one of the ambulance coaches. The train units were to be assembled by the Ministry of Communications, while medical and supply materials were to be provided by the reserve personnel of the regional hospitals, which were responsible for train mobilisation. However, the limited budget allocated for the purchase of the mobilisation materials significantly hindered this task. Source: National Digital Archives

vices, including the medical service, were theoretically aligned with these assumptions. This is well captured by the words of the introduction to the previously cited instruction: *The execution of supply and evacuation – especially in mobile warfare, with fluctuating and shifting fronts, in areas poor in quality roads, such as our eastern territories – will encounter numerous and varied difficulties. It is the commander's responsibility to anticipate these difficulties, overcome them, and undertake timely remedial measures* [1].

The Polish evacuation system did not adhere to a rigid model. At the operational level, it could be modified with relative flexibility by reinforcing the most critical directions with additional medical units. In practice, however, this flexibility remained largely theoretical. The lack of a fully developed command structure, severe deficiencies in communications, an insufficient number of motorised transport vehicles, and the reliance of evacuation on railway lines all hindered – and at times outright prevented – its implementation. It should also be noted that the organisational structure of the medical service was heavily weighted toward large, cumbersome hospital units transported by horse-drawn wagons, whose deployment required several hours. This significantly constrained the flexible deployment of hospitals in response to a changing tactical situation.

General plan for the operation and mobilisation of the medical service

Responsibility for preparing the medical service for war and for developing plans for its use rested with the General Staff of the Polish Army (Departments I and IV), in consultation with the Health Department of the Ministry of Military Affairs. The central authority overseeing the civilian medical service was the Health Service Department of the Ministry of Social Welfare, which also employed military representatives (Fig. 5).

Wartime preparations for the military health service encompassed three main areas:

- developing the wartime organisation of the medical service;
- planning for mobilisation;
- preparing wartime equipment tables and supply arrangements.

An inventory was also carried out of medical personnel subject and not subject to mobilisation (doctors, feldshers, dentists, nurses, and pharmacists), as well as hospitals, health centres, pharmacies, pharmaceutical depots, and other healthcare institutions. Maps were used to mark buildings suitable for conversion into field hospitals, as well as wells and railway sidings (Fig. 6).

In the final years of peace, cooperation between military authorities and the Ministry of Social Welfare proved inadequate. Among other shortcomings, a coherent programme for expanding civilian hospital infrastructure was neither developed nor implemented, and existing facilities were not prepared for wartime conditions. The causes were numerous, but the primary factor was insufficient funding. The situation was similar within the pharmaceutical industry. Only a few months before the outbreak of war was there an interventionist attempt to



Figure 5. Brig. Gen. Dr. Stanisław Rouppert – Head of the Health Department of the Ministry of Military Affairs until September 1939. Source: National Digital Archives

increase production, including that of sera. Among the few successes achieved were agreements regarding the preparation of medical supply depots by social insurance institutions [8]. Col. Dr. Babecki of the Health Service Department of the Ministry of Social Welfare assessed the state of the medical service bluntly: *The civilian healthcare system, much like the military, was unprepared for the event of war* [9].

The perception of the potential enemy left a distinct mark on strategic thinking about the anticipated conflict. For many years, the Soviet Union – rather than the Third Reich – had been regarded as the primary adversary. This strategic orientation had significant implications for the healthcare service as well. In the event of a war in the east, the geographical distribution of medical infrastructure would have been more favourable, with pharmaceutical production centres and hospital facilities located deep in the rear, beyond the enemy's immediate reach.

According to the 'W' ('War') Quartermaster Plan, the territory of Poland was divided into the 'Home Area,' under government jurisdiction, and the 'War Zone,' under the authority of the Commander-in-Chief. An 'Operational Area' was further designated within the War Zone for



Figure 6. As part of preparations for war, an inventory was carried out of personnel and sanitary infrastructure, as well as facilities and installations that could be used by the medical service. On this original map, prepared by the Field Resources Section of the Staff of Corps District No. 8, the locations of hospitals and other healthcare facilities, chemical-pharmaceutical plants, and pharmacies and chemists within Corps District No. 8 are marked. Source: Central Military Archives, ref. I.303.7.600

the activities of individual field armies [10]. With regard to medical administration, the Home Area fell under the jurisdiction of the Head of the Health Department of the Ministry of Military Affairs, Brig. Gen. Dr. Stanisław Rouppert, while the Head of the Health Service of the High Command was responsible for all medical matters within the War Zone. Col. Dr. Ksawery Maszadro, Commandant of the Medical Officer Cadet School in Warsaw, was appointed to this position.

The mobilisation of medical units was carried out primarily by regional hospitals, the Sanitary Training Centre, and the Main Sanitary Depot. Selected infantry regiments were tasked with mobilising medical companies for their respective divisions, while designated cavalry regiments were to mobilise mounted medical platoons for cavalry brigades. Furthermore, motor ambulance columns were to be mobilised by armoured battalions. Regional Polish Red Cross (PCK) districts were responsible for activating their own structures (Tab. 1, Fig. 7 and Fig. 8).

According to the plan, mobilisation was divided into alarm mobilisation (covert, silent) and general mobilisation. Priority in achieving combat readiness was given to medical units providing direct support to frontline formations, namely the medical companies of infantry divisions and the mounted medical platoons of cavalry brigades as part of the alarm mobilisation. At a relatively early stage of the alarm mobilisation, it was also planned to activate some field hospitals, war hospitals,

Table 1. Medical units included in the mobilisation plan. Compiled based on Rybka and Stepan [13]

Medical units	Mobilising unit	Ogólna liczba
Advanced surgical units	Sanitary Training Centre	5
Convalescent homes	Regional hospitals	10
Polish Red Cross rescue teams	Polish Red Cross districts	325
Corps district disinfection units	Regional hospitals	10
Polish Red Cross disinfection and bathing units	Polish Red Cross districts	37
Motor ambulance columns	Armoured battalions	17 (including 14 Red Cross units)
Horse-drawn ambulance columns	Supply train divisions	30
Sanitary companies	Regional hospitals, infantry regiments, Border Protection Corps battalion, Infantry Officer Cadet School, Infantry Reserve Training Centre	44
Railway disinfection stations	Regional hospitals	15
Medical service reserve centres	Regional hospitals	4
Mounted medical platoons	Cavalry regiments	12
Ambulance trains	Regional hospitals	36
Field bacteriological and chemical units	Regional hospitals, Sanitary Training Centre	62
Field disinfection and bathing units	Regional hospitals	59
Field sanitary depots	Regional hospitals	5
Polish Red Cross medical and food stations	Polish Red Cross districts	121
Independent sections of field hospitals	Regional hospitals	21
Main sanitary depots	Main Sanitary Depot No. 1, 10. Regional Hospital in Przemyśl	2
Evacuation hospitals	Regional hospitals	7
Field hospitals	Regional hospitals	52
War hospitals	Regional hospitals, Sanitary Training Centre	77
Mobile military hospitals	Regional hospitals	5
Surgical teams	Regional hospitals, Sanitary Training Centre	42

surgical teams, and hospital trains, which were classified as the so-called “yellow group.” Responsibility for mobilising these units rested with the regional hospitals in Warsaw, Grodno, Krakow, Lwów, Poznań, Toruń, and Przemyśl. In the second phase, following the announcement of general mobilisation, medical units at the op-



Figure 7. The Polish Red Cross (PCK) played a vital role in the country’s medical security plans for the event of war. According to mobilisation plans, the PCK was expected to mobilise, among other units, over 300 rescue teams. The photograph shows medical rescue demonstrations organised in the Krakow Market Square in June 1936. Source: National Digital Archives

erational level (army) were to be mobilised, followed by additional supporting echelons [11–13].

The general health service operational plan assumed that the wounded would be triaged and treated under field conditions and then evacuated according to the previously described scheme. Final triage was to be conducted at army evacuation hospitals. Patients unfit for further evacuation were to remain in the War Zone (in field hospitals of large units or at the army level) until their clini-



Figure 8. For military use, the Red Cross was also to provide 37 disinfection and bathing units. The photograph shows the supply wagons of one such unit moving through the streets of Krakow in June 1938. Source: National Digital Archives

cal condition stabilised sufficiently to permit transport. Definitive treatment was intended to be provided in military hospitals deep within the operational area and in the country's interior. Transport to the interior was to be carried out primarily by rail [11].

In the period preceding the war, an evacuation plan was prepared for patients from regional hospitals in Toruń, Poznań, Łódź, Krakow, and Warsaw to hospitals situated deeper within the country. During the covering operations intended to secure the general mobilisation and troop concentration, these facilities were to serve as temporary evacuation hospitals until the functional evacuation hospitals of the individual armies could be mobilised. Plans for utilising civilian hospitals remained unclear. However, the military reserved the right to requisition selected facilities, and the relevant documentation was submitted to the Ministry of Social Welfare [11, 12].

It was also envisaged that, during wartime operations, the supply of medical units with medical materials would be managed centrally from the two main sanitary depots. In addition, five field sanitary depots were expected to reach operational readiness by the fifteenth day of mobilisation [11, 12].

Analysis of the sources indicates that Col. Dr. Maszadro had not participated in preparations of the medical service for war prior to assuming his post. After the campaign, he testified: *I took no part in the preparatory work for the war, and no tasks pertaining to my designated position were entrusted to me. On 27 August, I was summoned to the General Staff, where I was instructed to familiarise myself with the O.d.B. (Ordre de bataille – author's note) prepared by the Staff. (...) Together with Col. Dr. Łaski and Maj. Zakrzewski, I reviewed the health service arrangements drafted by the General Staff for the event of war. However, I was given no precise data regarding the O.d.B. of units in the field, no tactical situation, and no forecasts. Nor was I provided with the personnel rosters for the armies or divisions* [14]. Lt. Col. Dr. Władysław Gergovich, Head of the Health Service for the Krakow Army, found himself in a similar predicament, reporting as follows: *As the Chief Medical Officer of OK V [Corps District No. 5 – author's note], I was unable during that period (two months, including two weeks of relocation leave) to examine in detail the overall mobilisation of the medical service of the Corps District. In my view, replacing the Chief Medical Officer of the Corps District two months before the war, and in particular excluding him from preparatory work in the staff of the future army, could not have had a positive effect on the functioning of the army's medical service, especially in the first days of the war* [11]. A similar account

was provided by Maj. Dr. Feliks Schneider, Deputy Head of the Health Service of the Pomeranian Army [15].

References

1. Zaopatrywanie i ewakuacja w ramach pułku piechoty. Zeszyt I. Część teoretyczna. Centrum Wyszkożenia Piechoty, Rembertów 1934
2. Regulamin służby zdrowia w polu. Ministerstwo Spraw Wojskowych, Warszawa 1929.
3. Regulamin służby zdrowia w wojskowych zakładach leczniczych. Ministerstwo Spraw Wojskowych, Warszawa 1932
4. Służba zdrowia w formacjach wojskowych podczas wojny. Ministerstwo Spraw Wojskowych, Warszawa 1935
5. Służba zdrowia w kompaniach i plutonach sanitarnych (konnych) oraz polowych przychodniach dentystycznych. Ministerstwo Spraw Wojskowych, Warszawa 1934
6. Służba zdrowia w pociągach sanitarnych. Ministerstwo Spraw Wojskowych, Warszawa 1934
7. Służba zdrowia w wojskowych zakładach leczniczych podczas wojny. Ministerstwo Spraw Wojskowych, Warszawa 1935
8. Archiwum Instytutu Polskiego i Muzeum im. gen. Sikorskiego (dalej: IPMS), Relacje z kampanii 1939 roku – służba sanitarna, sygn. B.I.118, H. Mazanek, Studium Służby Zdrowia z okresu wojny polsko-niemieckiej we wrześniu 1939
9. IPMS, Relacje z kampanii 1939 roku – służba sanitarna, sygn. B.I.118, Raport płk. lek. Dr. W.J. Babeckiego w sprawie przygotowania Polski do wojny i współpracy administracji wojskowej i cywilnej w zakresie służby zdrowia, dokument z 18.3.1940.
10. Rezmer W. Operacyjna służba sztabów Wojska Polskiego w 1939 roku. Wydawnictwo Tetragon, Warszawa 2010, s. 31–48
11. IPMS, Relacje z kampanii 1939 roku – służba sanitarna, sygn. B.I.118, Protokół przesłuchania płk. dr. Władysława Gergovicha, szefa służby zdrowia Armii „Kraków”, dokument z 06.07.1942
12. IPMS, Relacje z kampanii 1939 roku – służba sanitarna, sygn. B.I.118, Kwestionariusz służby zdrowia – okres przedwojenny płk. dr. Jana Kawińskiego, zastępcy Szefa Departamentu Zdrowia Ministerstwa Spraw Wojskowych, odpis z odpisu, dokument z 3.2.1940
13. Rybka R, Stepan K. Najlepsza broń. Plan mobilizacyjny „W” i jego ewolucja. Oficyna Wydawnicza „Adiutor”, Warszawa 2010
14. IPMS, Relacje z kampanii 1939 roku – służba sanitarna, sygn. B.I.118, protokół przesłuchania płk. dr. Ksawerego Maszadro, odpis, dokument z 3.2.1940
15. IPMS, Relacje z kampanii 1939 roku – służba sanitarna, sygn. B.I.118, protokół przesłuchania mjr. dr. Feliksa Schneidera, dokument z 10.8.1942



DOPING – A PATH TO CHRONIC KIDNEY DISEASE. CASE REPORT OF 45-YEAR-OLD POWERLIFTER

Doping – droga do przewlekłej niewydolności nerek.
Studium przypadku 45-letniego trójboisty



Karolina Borkowska^{1*}, Oliwia Mróz^{1*}, Katarzyna Romejko², Elżbieta Araminowicz-Kierklo^{3,4}, Stanisław Niemczyk²

1. Military Institute of Medicine – National Research Institute (a student at the Medical University of Warsaw), Poland
2. Military Institute of Medicine – National Research Institute, Department of Internal Medicine, Nephrology and Dialysis, Poland
3. First Military Clinical Hospital with Polyclinic, Independent Public Healthcare Centre in Lublin, Branch in Ełk, Poland
4. University of Warmia and Mazury in Olsztyn, Branch in Ełk, Poland

* Equal contribution (first authors)

Karolina Borkowska – [id 0009-0008-0886-8273](#)

Oliwia Mróz – [id 0009-0003-7095-5312](#)

Katarzyna Romejko – [id 0000-0003-1447-2917](#)

Elżbieta Araminowicz-Kierklo – [id 0000-0003-4985-7809](#)

Stanisław Niemczyk – [id 0000-0001-8151-860X](#)

Abstract

Chronic kidney disease may develop as a consequence of various environmental and lifestyle factors, including the use of anabolic-androgenic steroids, high-protein diets, and dietary supplementation. This case report presents a 45-year-old male powerlifter who was admitted to the hospital emergency department with exertional dyspnoea and reduced exercise tolerance. Laboratory tests revealed end-stage renal failure (creatinine 23.2 mg/dL, eGFR 2 mL/min/1.73 m²), anaemia, proteinuria, and markedly elevated creatine kinase levels. The patient reported long-term use of anabolic-androgenic steroids, a diet containing up to 3 g of protein per kilogram of body weight per day, and the intake of various unspecified dietary supplements. Kidney biopsy revealed extensive chronic and acute changes characteristic of nephropathy associated with anabolic-androgenic steroid use and long-standing hypertension. Conservative treatment proved ineffective, so haemodialysis therapy was initiated. During the hospitalisation, the patient developed pulmonary embolism and hospital-acquired pneumonia, the proper treatment was initiated and intensified. He was discharged in satisfactory condition with a recommendation to continue renal replacement therapy. The presented case highlights the risk of irreversible kidney damage associated with long-term use of performance-enhancing substances and an improper diet, underscoring the need for preventive and interventional efforts within the athletic population.

Streszczenie

Przewlekła choroba nerek może rozwijać się w wyniku działania różnych czynników środowiskowych i stylu życia, w tym stosowania sterydów anaboliczno-androgenowych, diety wysokobiałkowej oraz suplementów diety. Przedstawiamy przypadek 45-letniego mężczyzny, trójboisty siłowego, który zgłosił się do szpitalnego oddziału ratunkowego z powodu duszności oraz pogorszenia wydolności wysiłkowej. W badaniach laboratoryjnych stwierdzono skrajną niewydolność nerek (stężenie kreatyniny 23,2 mg/dl, eGFR 2 ml/min/1,73 m²), niedokrwistość, białkomocz oraz znacznie podwyższone stężenie kinazy kreatynowej. Wywiad ujawnił wieloletnie stosowanie sterydów anabolicznych, dietę zawierającą do 3 g białka/kg m.c./dobę oraz przyjmowanie różnych nieokreślonych suplementów. Biopsja nerki wykazała rozległe zmiany przewlekłe i ostre, typowe dla nefropatii związanej ze stosowaniem sterydów anaboliczno-androgenowych i przewlekłym nadciśnieniem tętniczym. Leczenie zachowawcze okazało się nieskuteczne, dlatego wdrożono leczenie hemodializami. W trakcie hospitalizacji wystąpiła zatorowość płucna i szpitalne zapalenie płuc. Pacjent został wypisany w stanie zadowolającym, z zaleceniem kontynuacji leczenia nerkozastępczego. Przedstawiony przypadek ilustruje ryzyko nieodwracalnego uszkodzenia nerek związane z długotrwałym stosowaniem substancji poprawiających wydolność fizyczną oraz niewłaściwą dietą, co powinno stanowić przedmiot działań profilaktycznych w populacji sportowców.

Keywords: chronic kidney disease; doping; high-protein diet; anabolic-androgenic steroids; drug-induced nephropathy

Słowa kluczowe: przewlekła choroba nerek; doping; dieta wysokobiałkowa; sterydy anaboliczno-androgenowe; nefropatia polekowa

DOI 10.53301/lw/210000

Received: 14.07.2025

Accepted: 28.08.2025

Published: 31.03.2026

Corresponding author:

Oliwia Mróz
Military Institute of Medicine –
National Research Institute
e-mail: s082597@student.wum.edu.pl

Introduction

Chronic kidney disease (CKD) has become a major public health concern, with more than 1.4 million patients worldwide currently undergoing renal replacement therapy [1, 2]. A key aspect in the diagnosis and prevention of CKD is the identification of factors predisposing to its development. Reducing these factors may slow disease progression or prevent its development [1]. Risk factors of increasing importance include a high-protein diet and the use of anabolic steroids and various dietary supplements. Anabolic-androgenic steroids (AAS) can damage the kidneys through multiple mechanisms, including direct toxic effects on the glomeruli and by inducing acute or chronic kidney injury [3]. AAS exert nephrotoxic effects by activating the renin-angiotensin-aldosterone (RAA) system, increasing endothelin production, generating reactive oxygen species, and modulating anti-inflammatory and pro-inflammatory cytokines involved in the pathogenesis of hypertension and renal damage [3, 4]. A diet containing very high amounts of protein may affect renal function, leading to glomerular hyperfiltration, tubular injury, and interstitial fibrosis, which over time may result in kidney failure [5].

Case report

A 45-year-old male presented to the hospital emergency department (ED) with exertional dyspnoea and worsening exercise tolerance for approximately two weeks. He brought results of outpatient laboratory tests showing a creatinine level of 20.4 mg/dL (N: 0.7–1.2 mg/dL), eGFR 2.5 mL/min/1.73 m², and haemoglobin concentration of 8.6 g/dL (N: 13.5–17 g/dL). He reported no other symptoms, including changes in urine output, haematuria, or oedema.

One week before presenting to the ED, the patient's primary care physician had diagnosed him with arterial hypertension, and initiated treatment with ramipril and bisoprolol. Otherwise, the patient was not treated for any chronic diseases.

The man trained intensively in powerlifting. During his workouts, he performed mainly exercises involving sustained musculoskeletal loading. To increase muscle mass and improve athletic performance, he used numerous dietary supplements and testosterone preparations. The patient admitted that over the last 15 years, he had periodically taken various forms of testosterone, other anabolic agents, including veterinary preparations, and antioestrogens (to counteract androgen-related side effects). His dietary protein intake reached up to 3 g/kg of body weight per day, at a body weight of 115 kg.

Upon admission, the patient's general condition was relatively good. He was communicative and oriented, and his respiratory and circulatory functions were stable (regular heart rate of 90 bpm, blood pressure 164/90 mmHg, oxygen saturation 98%). The man had an athletic build with pronounced muscular hypertrophy, with a body weight of 115 kg and height of 186 cm. On physical examination, skin pallor was notable. Mild oedema of the lower extremities was also observed.

Laboratory tests showed a creatinine level of 23.2 mg/dL (N: 0.7–1.2), eGFR 2 mL/min/1.73 m², urea concentration of 235 mg/dL (N: 18–55), and cystatin C level of 4.67 mg/L (N: 0.61–0.95). Inflammatory markers were elevated (CRP 4.9 mg/dL [N: <0.5 mg/dL]). Complete blood count results indicated microcytic anaemia with a haemoglobin level of 8.9 g/dL (N: 13.5–17) and MCV 78 fL (N: 80–99). Arterial blood gas analysis revealed mild metabolic acidosis, with pH 7.316 (N: 7.37–7.45) and HCO₃ 20.3 mmol/L (N: 21.0–26.0). Urinalysis revealed proteinuria of 100 mg/dL (N: <30 mg/dL) and hyposthenuria of 1.009 (N: 1.016–1.035), with no features of active urinary sediment on microscopic examination. Biochemical blood tests revealed a significantly elevated level of creatine kinase (9,432 U/L [N: 0–190]). Electrolytes and coagulation profile remained within reference ranges, and immunological tests for ANA and ANCA were negative.

Ultrasound examination of the urinary system revealed increased echogenicity of the renal parenchyma in both kidneys, with enhanced corticomedullary differentiation. A calcification was visualised in the region of the renal papilla of the upper pole of the right kidney. The pelvicalyceal systems were not dilated and contained no calculi. Significantly increased flow resistance was found in the segmental arteries of both kidneys, while the intrarenal veins showed signs of moderate congestion. Chest X-ray revealed a small amount of fluid in the right pleural cavity, along with signs of mild passive pulmonary congestion and an enlarged cardiac silhouette.

An attempt at conservative treatment of renal failure was initiated at the Department of Internal Medicine, Nephrology and Dialysis, Military Institute of Medicine – National Research Institute. Due to the lack of clinical improvement after 48 hours, haemodialysis therapy was initiated. Over the course of the patient's hospitalisation, five units of packed red blood cells were administered in response to progressive anaemia.

On the 9th day of hospital stay, a biopsy of the left kidney was performed. Histopathological examination of

the biopsy specimen revealed chronic nephropathy, acute tubular epithelial injury, severe arteriolar hyalinosis, and arteriosclerosis with marked reduction of the arterial lumen due to fibrous thickening of the tunica intima. Light microscopy revealed global sclerosis of most glomeruli and extensive interstitial fibrosis. Electron microscopy demonstrated damage to the glomerular capillary endothelium and double-contouring of some capillary walls.

One day after the kidney biopsy, the man reported chest pain and shortness of breath. Due to the patient's symptoms, CT pulmonary angiography was performed. Within the left lower lobe pulmonary artery, a longitudinal filling defect was identified, suggesting the presence of a thrombus. Artifacts and insufficient contrast enhancement of the peripheral pulmonary arteries prevented exclusion of peripheral pulmonary embolism. Bedside lung ultrasound revealed signs of massive pulmonary congestion. A decision was made to initiate anticoagulation therapy with enoxaparin.

After 10 days of hospitalisation, the patient developed pneumonia (procalcitonin 2.85 ng/mL [N: ≤ 0.046]). Empirical antibiotic therapy (ceftriaxone and ciprofloxacin) was initiated. Within three days of treatment, inflammatory markers decreased significantly (procalcitonin 0.56 ng/mL), and the patient's clinical condition improved. Due to increasing signs of fluid overload, daily haemodialysis was implemented.

Following a 23-day hospitalisation, the patient was discharged with instructions to strictly restrict physical activity, maintain a low-protein diet, and discontinue anabolic-androgenic steroid use. The patient continues renal replacement therapy via haemodialysis at a local dialysis centre.

Discussion

The use of AAS is increasingly prevalent among athletes, particularly bodybuilders and powerlifters [6]. However, recreational trainees now constitute the largest group of users [7]. By enhancing muscle strength and physical performance, AAS enable individuals to achieve results that exceed the body's natural physiological limits [8]. One of the most commonly used anabolic steroids is testosterone, which adversely affects kidney function through multiple mechanisms.

Testosterone activates the renin-angiotensin system (RAS), which increases blood pressure by enhancing tubular sodium reabsorption and promoting water retention within the vascular bed [4, 6, 7]. In the kidneys, progressive structural changes occur to limit the impact of hypertension on the organ (initially, the tunica media of the arterioles hypertrophies, leading to narrowing of the vascular lumen). Pathological remodelling of the arterioles impairs the control of arterial hypertension. Thickening of the vessel walls impairs oxygen transport, causing ischemic damage to the glomeruli, renal tubules, and interstitium [9].

Activation of the Rho kinase pathway by testosterone, which enhances the vasoconstrictive effect of angioten-

sin II, also has a negative impact on renal function [4]. Androgens, either directly or via the RAS, increase the production of endothelin, which strongly constricts blood vessels, increases sodium resorption, and exacerbates oxidative stress [4, 6]. Angiotensin II and reactive oxygen species (ROS) damage the tubular basement membrane, as a result of which it loses its selectivity, allowing substances that intensify inflammation and fibrosis to penetrate into the renal parenchyma [9].

Testosterone stimulates the synthesis of tumour necrosis factor alpha (TNF-alpha) and induces apoptosis of podocytes and proximal tubular cells, which contributes to tubulointerstitial fibrosis [4]. It inhibits the action of many antioxidants and multiplies the synthesis of ROS [10]. The persistently elevated production of ROS leads to kidney damage and is associated with a poorer prognosis in CKD [11].

High-protein diets are also commonly used among athletes, as increased daily protein intake may help build and maintain muscle mass [6]. According to the nutritional standards for the Polish population, the protein requirement for adults is 0.8–0.9 g/kg/day, whereas for athletes it increases to 1.2–1.7 g/kg/day [12]. As little as 6 weeks of a high-protein diet, in which protein accounted for 25% of the daily energy requirement, has been shown to cause glomerular hyperfiltration [5]. In the early stages, this manifests as an increase in eGFR and/or the onset of proteinuria. Over time, it may lead to the development of renal failure, particularly in individuals with risk factors for CKD [5].

One of the most commonly used supplements among athletes is creatine. As a dietary supplement, it is intended to support muscle mass gain and post-exercise muscle recovery [6]. When used appropriately, creatine supplementation has no clinically significant effect on renal function. However, it should be emphasised that current knowledge regarding the effects of creatine on renal function in individuals with chronic kidney disease remains insufficient. Creatine supplementation is not recommended for these patients [13].

Elevated creatine kinase activity (9234 U/L) in this patient suggests the coexistence of rhabdomyolysis, most likely associated with intense physical exertion [14, 15]. Rhabdomyolysis is relatively common among athletes and may lead to kidney injury due to the deposition of myoglobin and haeme derivatives in the kidneys. Accumulation of these molecules may cause renal tubular obstruction, vasoconstriction, enhanced inflammatory processes, and cellular injury mediated by reactive oxygen species [15, 16]. Kidney biopsy results did not confirm the typical features of this type of injury; however, in the present case, a multifactorial aetiology of renal failure should be considered. Recurrent episodes of rhabdomyolysis may have constituted an additional factor contributing to the progressive deterioration of renal function over the years.

The patient's laboratory results show a striking disproportion between the creatinine concentration [23.2 mg/dL (N: 0.7–1.2)] and cystatin C [4.67 mg/L (N: 0.61–0.95)], resulting from the patient's large muscle mass (115 kg

with a height of 186 cm). The eGFR calculated using the 2021 CKD-EPI formula based on both creatinine and cystatin C concentrations was 5 mL/min/1.73 m², whereas the eGFR calculated based on creatinine alone was 2 mL/min/1.73 m². When assessing renal function parameters, particular attention should be paid to patients in whom eGFR calculations based on creatinine may be underestimated or overestimated due to extreme muscle mass values – such as cachexia or excessively developed muscle mass [14]. In such cases, it is important to consider measuring cystatin C as an additional marker of renal function.

The adverse effects of AAS on renal function are better documented than those of a high-protein diet or creatine supplementation [6]. Due to the coexistence of multiple harmful practices employed by the patient, it is difficult to identify a single primary cause of the nephropathy. The histopathological findings confirm both the toxic effects of anabolic steroids on the glomeruli and tubulointerstitial structures and the damage resulting from long-term renal exposure to arterial hypertension.

Conclusions

A key element in diagnosing and preventing chronic kidney disease is the identification and modification of risk factors. These measures may slow disease progression or prevent its onset. This case report describes a patient whose disease development was associated with the use of anabolic steroids, supplements such as creatine, and a high-protein diet. Long-term use of anabolic steroids can lead to irreversible kidney damage, ultimately resulting in chronic renal failure. Patient education aimed at eliminating harmful behaviours can provide substantial benefits in both the prevention and management of chronic kidney disease.

References

1. Chen TK, Knicely DH, Grams ME. Chronic kidney disease diagnosis and management: a review. *JAMA*, 2019 Oct 1; 322(13): 1294–1304. doi: 10.1001/jama.2019.14745
2. Kazancıoğlu R. Risk factors for chronic kidney disease: an update. *Kidney International Supplements*, 2013; 3(4): 368–371. doi: 10.1038/kisup.2013.79
3. Davani-Davari D, Karimzadeh I, Khalili H. The potential effects of anabolic-androgenic steroids and growth hormone as commonly used sport supplements on the kidney: a systematic review. *BMC Nephrol*, 2019 May 31; 20(1): 198. doi: 10.1186/s12882-019-1384-0
4. Dousdampanis P, Trigka K, Fourtounas C, Bargman JM. Role of testosterone in the pathogenesis, progression, prognosis and comorbidity of men with chronic kidney disease. *Ther Apher Dial*, 2014 Jun; 18(3): 220–230. doi: 10.1111/1744-9987.12101
5. Ko GJ, Rhee CM, Kalantar-Zadeh K, Joshi S. The effects of high-protein diets on kidney health and longevity. *J Am Soc Nephrol*, 2020 Aug; 31(8): 1667–1679. doi: 10.1681/ASN.2020010028
6. Tidmas V, Brazier J, Hawkins J, et al. Nutritional and non-nutritional strategies in bodybuilding: impact on kidney function. *Int J Environ Res Public Health*, 2022 Apr 3; 19(7): 4288. doi: 10.3390/ijerph19074288
7. Parente Filho SLA, Gomes PEAC, Forte GA, et al. Kidney disease associated with androgenic-anabolic steroids and vitamin supplements abuse: Be aware! *Nefrologia (Engl Ed)*, 2020 Jan-Feb; 40(1): 26–31. English, Spanish. doi: 10.1016/j.nefro.2019.06.003.
8. Kanayama G, Hudson JI, Pope HG Jr. Long-term psychiatric and medical consequences of anabolic-androgenic steroid abuse: a looming public health concern? *Drug Alcohol Depend*, 2008 Nov 1; 98(1–2): 1–12. doi: 10.1016/j.drugalcdep.2008.05.004
9. Mennuni S, Rubattu S, Pierelli G, et al. Hypertension and kidneys: unraveling complex molecular mechanisms underlying hypertensive renal damage. *J Hum Hypertens*, 2014 Feb; 28(2): 74–79. doi: 10.1038/jhh.2013.55
10. Neugarten J, Golestaneh L. Gender and the prevalence and progression of renal disease. *Adv Chronic Kidney Dis*. 2013 Sep; 20(5): 390–395. doi: 10.1053/j.ackd.2013.05.004
11. Irazabal MV, Torres VE. Reactive oxygen species and redox signaling in chronic kidney disease. *Cells*, 2020 May 28; 9(6): 1342. doi: 10.3390/cells9061342
12. Jarosz M, Rychlik E, Stoś K, Charzewska J, ed. *Normy żywienia dla populacji Polski i ich zastosowanie*. Narodowy Instytut Zdrowia Publicznego – Państwowy Zakład Higieny, 2020. https://ncez.pzh.gov.pl/wp-content/uploads/2021/03/normy_zywienia_2020web.pdf
13. Davani-Davari D, Karimzadeh I, Ezzatzadegan-Jahromi S, Sagheb MM. Potential adverse effects of creatine supplement on the kidney in athletes and bodybuilders. *Iran J Kidney Dis*, 2018 Oct; 12(5): 253–260
14. Onopiuk A, Tokarzewicz A, Gorodkiewicz E. Cystatin C. A kidney function biomarker. *Adv Clin Chem*, 2015; 68: 57–69. doi: 10.1016/bs.acc.2014.11.007
15. Kim J, Lee J, Kim S, et al. Exercise-induced rhabdomyolysis mechanisms and prevention: A literature review. *J Sport Health Sci*, 2016; 5(3): 324–333. doi: 10.1016/j.jshs.2015.01.012
16. Panizo N, Rubio-Navarro A, Amaro-Villalobos JM, et al. Molecular mechanisms and novel therapeutic approaches to rhabdomyolysis-induced acute kidney injury. *Kidney Blood Press Res*, 2015; 40(5): 520–532. doi: 10.1159/000368528




SCHWANNOMA OF THE SMALL INTESTINAL MESENTERY IN A 38-YEAR-OLD WOMAN

Nerwiak osłonkowy (*schwannoma*) krezki jelita cienkiego u 38-letniej kobiety



Krzysztof Stanisław Winiarz

Oncology Outpatient Clinic, Independent Public Provincial Specialist Hospital in Chełm, Poland

Krzysztof Stanisław Winiarz –  0009-0009-6588-1738

Abstract

Schwannomas (also known as neurinomas or neurilemmomas) are generally benign, slow-growing tumours arising from the sheath of peripheral nerves. They occur very rarely within the mesentery of the small intestine. This study presents the case of a 38-year-old woman who was admitted to hospital following an epileptic seizure and was found to have elevated liver enzymes. An incidental tumour of the small-intestinal mesentery was identified and, after surgical excision, was histopathologically confirmed to be a benign schwannoma.

Streszczenie

Guzy o typie nerwiaka osłonkowego (*schwannoma*, *neurinoma*, *neurilemoma*) to powoli rosnące nowotwory wywodzące się z osłonek nerwów obwodowych. Bardzo rzadko rozwijają się w krezce jelita cienkiego. W pracy opisano przypadek 38-letniej kobiety przyjętej do szpitalnego oddziału ratunkowego z powodu napadu drgawek z możliwym urazem głowy oraz podwyższonymi parametrami wątrobowymi, u której wykryto przypadkowo guz krezki jelita cienkiego, który po wycięciu okazał się łagodnym nowotworem – *schwannoma*.

Keywords: neurilemoma; neurinoma; unknown intraperitoneal tumour; unknown mesenteric tumour; mesenteric schwannoma

Słowa kluczowe: nerwiak osłonkowy; *neurinoma*; nieznaną guz wewnątrzotrzewnowy; nieznaną guz krezki jelita; *schwannoma* krezki jelita

DOI 10.53301/lw/214142

Received: 17.01.2025

Accepted: 12.11.2025

Published: 31.03.2026

Corresponding author:

Krzysztof Stanisław Winiarz
Oncology Outpatient Clinic, Independent Public
Provincial Specialist Hospital in Chełm
e-mail: lek.k.winiarz@gmail.com

Introduction

Schwannoma (also known as neurinoma or neurilemoma) is one of the most common peripheral nerve sheath tumours. In addition to schwannomas, this group includes neurofibromas, perineurinomas, granular cell tumours, and malignant peripheral nerve sheath tumours [1, 2]. Schwannomas are benign, well-circumscribed tumours arising from a clonal population of Schwann cells. They typically grow slowly and often undergo cystic and degenerative changes [1]. Their morphological appearance can vary widely, and malignant transformation is extremely rare. These tumours may occur spontaneously or in association with syndromes such as neurofibromatosis type 2 (NF2), Carney complex, and schwannomatosis, as well as sporadically in areas previously exposed to radiotherapy (even up to 50 years after treatment). A key

element in the pathogenesis of schwannoma is the loss of merlin function resulting from genetic alterations of the *NF2* gene on chromosome 22 or secondary to merlin inactivation [2].

Schwannomas rarely arise within the central nervous system (CNS) (6.7% of all CNS tumours), visceral organs, or the gastrointestinal tract (primarily in the stomach – 0.2% of all gastric tumours) [1–3]. Autopsy studies indicate that the prevalence of sporadic central schwannomas is approximately 4.5% in the elderly population. In contrast, primary involvement of motor roots and the sympathetic nervous system is uncommon. Deeply located retroperitoneal and mediastinal schwannomas may reach considerable size before producing symptoms, typically due to local compression or bone erosion, as seen in giant sacral schwannomas [2].

The extremely rare occurrence of schwannoma in the mesentery of the small intestine can also pose significant diagnostic challenges [4].

Aim of study

The aim of this study was to describe the case of a 38-year-old woman with an incidentally detected schwannoma located in the small intestinal mesentery. The patient was admitted to hospital on an emergency basis due to a seizure with a suspected head injury and elevated liver function parameters.

Case report

A 38-year-old woman was admitted to the emergency department (ED) of a regional hospital following an epileptic seizure with a possible head injury. A few days prior, the patient had self-discontinued alprazolam, which she had been taking for many years.

A head computed tomography (CT) scan performed in the ED revealed no focal lesions, intracranial haemorrhage, or traumatic bone changes. However, features of mild cerebral cortical atrophy were noted.

Laboratory tests revealed moderately elevated liver aminotransferase levels with a normal bilirubin concentration. During history taking, the patient reported that she had not monitored her liver function tests while taking alprazolam. She denied any history of acute or chronic liver disease, and reported no use of oral contraceptives.

Abdominal ultrasonography (USG) showed features of generalised patchy hepatic steatosis, with no pathology of the gallbladder or bile ducts. A cystic lesion measuring 41×36 mm was identified in the right ovary, along with a mass measuring $52 \times 37 \times 39$ mm, likely located in the mesentery of the small intestine. A CT scan was recommended; the patient underwent internal medicine and neurological consultations and was subsequently admitted to the surgical department. Triple-phase CT of the abdomen and pelvis revealed a well-circumscribed, hypodense tumour in the small bowel mesentery at the level of the mid-abdomen/lower abdomen, measuring $52 \times 42 \times 55$ mm. The lesion was partially cystic, with mild contrast enhancement and calcifications in its inferior portion (Fig. 1). Differential diagnosis with echinococcosis was suggested, along with histopathological verification after complete tumour resection.

Additionally, a 42-mm cyst was described in the region of the right adnexa, with a suggestion for assessment by transvaginal ultrasound. Gynaecological consultation indicated that only periodic monitoring of the right ovarian cyst in an outpatient setting was necessary.

The patient was scheduled for expedited laparoscopic resection of the small-intestinal mesenteric mass. During the procedure, conversion to laparotomy was required due to difficulties in dissecting the lesion, which was ultimately removed in its entirety. The postoperative course was uneventful, and normalisation of liver enzyme levels was observed. The patient was discharged home in good general condition.

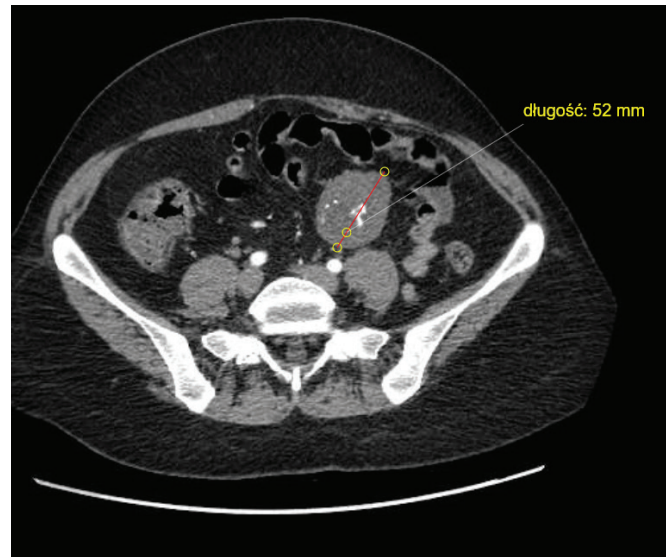


Figure 1. Tumour appearance on abdominal computed tomography

Histopathological examination confirmed the diagnosis of schwannoma. The resection margin was the tumour's pseudocapsule.

At the follow-up visit in the oncology outpatient clinic, the patient was in good general condition, and the surgical wounds were healing normally. No features suggestive of von Recklinghausen disease were present. She was scheduled for periodic oncological follow-up and remains under the care of both neurological and gynaecological outpatient clinics.

Discussion

Incidental findings are quite commonly identified in patients evaluated in emergency settings. Approximately 16% of reports from CT and other imaging studies performed in emergency departments in the United States include descriptions of incidental findings. The most frequently reported organ is the ovary ($n = 214$, 42% [5]), which was also observed in the case described here (one of two findings). Other studies indicate that the overall incidence of any incidental findings in CT scans performed on emergency department patients is approximately 31% [6].

Benign tumours such as schwannomas (also known as neurilemmomas or neurilemmomas) are rarely located in the retroperitoneal or intraperitoneal space and are mostly detected incidentally. They very rarely occur in the mesentery of the intestine, where they may exhibit long, slow, asymptomatic growth. At the diagnostic stage, these lesions often raise suspicion of a malignant process [4].

In 1998, Ramboer et al. described a case of a benign schwannoma incidentally detected in the mesentery of the small intestine. The authors highlighted that magnetic resonance imaging (MRI) provides additional value compared to CT, allowing more precise assessment of tumour location [7].

Similarly, Murakami et al. described a case involving a well-circumscribed mass with a cystic component lo-

cated near the duodenum. Diagnosis was based on contrast-enhanced CT, followed by gadolinium-enhanced MRI, and histopathological examination confirmed the lesion as a schwannoma [4].

Another case reported in the literature involved a 56-year-old woman with chronic abdominal pain, in whom USG and subsequent CT scan revealed a massive retroperitoneal tumour in the region of the right adrenal gland. Laboratory tests showed no hormonal activity. Although adrenal carcinoma was initially suspected, histological examination following adrenalectomy combined with tumour resection confirmed a benign schwannoma [8].

Maezawa et al. reported the case of a 32-year-old woman with a retroperitoneal tumour detected incidentally during ultrasound. CT and MRI revealed a 95-mm mass with a cystic component. Following resection, histological examination confirmed the diagnosis of schwannoma [9].

Although benzodiazepines (BZDs) are generally considered safe, their potential role in tumour induction remains unclear. Clonazepam, lorazepam, alprazolam, bromazepam, zolpidem, and zopiclone have been associated with an increased risk of malignant neoplasms. Exposure to BZDs increases the overall risk of cancer by up to 21%. Specifically, this may involve cancers of the brain (98%), oesophagus (59%), pancreas (41%), bladder (39%), prostate (36%), large intestine (25%), liver (18%), lungs (10%), and other malignancies (27%) [10].

Whether the occurrence of the schwannoma in the described case was related to long-term alprazolam use remains uncertain. Without imaging studies from earlier treatment periods or prior to treatment initiation, it is impossible to demonstrate such a correlation.

References

1. Yoon HY, Kim CB, Lee YH, Kim HG. Gastric schwannoma. *Yonsei Med J*, 2008 Dec 31; 49(6): 1052–1054. doi: 10.3349/ymj.2008.49.6.1052
2. Hilton DA, Hanemann CO. Schwannomas and their pathogenesis. *Brain Pathol*, 2014 Apr; 24(3): 205–220. doi: 10.1111/bpa.12125
3. Salari N, Ghasemi H, Fatahian R, et al. The global prevalence of primary central nervous system tumors: a systematic review and meta-analysis. *Eur J Med Res*, 2023 Jan 20; 28(1): 39. doi: 10.1186/s40001-023-01011-y
4. Murakami R, Tajima H, Kobayashi Y, et al. Mesenteric schwannoma. *Eur Radiol*, 1998; 8(2): 277–279. doi: 10.1007/s003300050379
5. Hanna TN, Shekhani H, Zygmunt ME, et al. Incidental findings in emergency imaging: frequency, recommendations, and compliance with consensus guidelines. *Emerg Radiol*, 2016 Apr; 23(2): 169–174. doi: 10.1007/s10140-016-1378-1
6. Evans CS, Arthur R, Kane M, et al. Incidental radiology findings on computed tomography studies in emergency department patients: a systematic review and meta-analysis. *Ann Emerg Med*, 2022 Sep; 80(3): 243–256. doi: 10.1016/j.annemergmed.2022.03.027
7. Ramboer K, Moons P, De Breuck Y, et al. Benign mesenteric schwannoma: MRI findings. *J Belge Radiol*, 1998 Feb; 81(1): 3–4.
8. Pittasch D, Klose S, Schmitt J, et al. Retroperitoneal schwannoma presenting as an adrenal tumor. *Exp Clin Endocrinol Diabetes*, 2000; 108(4): 318–321. doi: 10.1055/s-2000-7760
9. Maezawa T, Narita M, Sano T, et al. [A case of retroperitoneal schwannoma treated by laparoscopic resection]. *Hinyokika Kyo*, 2009 Mar; 55(3): 129–131
10. Iqbal, U, Nguyen P-A, Syed-Abdul S, et al. Is long-term use of benzodiazepine a risk for cancer? *Medicine (Baltimore)*, 2015 Feb; 94(6): e483. doi: 10.1097/MD.0000000000000483



CARDIOLOGIC CARE OF PATIENTS WITH CHRONIC KIDNEY DISEASE. SELECTED INSIGHTS FROM THE 29TH INTERNATIONAL CONGRESS OF THE POLISH CARDIAC SOCIETY



Pacjent z przewlekłą chorobą nerek u kardiologa.
Wybrane zagadnienia XXIX Międzynarodowego Kongresu
Polskiego Towarzystwa Kardiologicznego

Katarzyna Romejko

Military Institute of Medicine – National Research Institute, Department of Internal Medicine, Nephrology and Dialysis, Poland

Katarzyna Romejko –  0000-0003-1447-2917

Abstract

The 29th International Congress of the Polish Cardiac Society was held in Krakow from 25 to 27 September 2025. During numerous sessions, the challenges associated with diagnosing and treating cardiovascular diseases in patients with chronic kidney disease were discussed. This report summarises the recommendations presented during the Congress regarding the management of cardiovascular complications in this group of patients. Particular attention was devoted to guidelines and diagnostic challenges related to the use of anticoagulant therapy in patients with chronic kidney disease and atrial fibrillation. Recommendations concerning renin-angiotensin system blockade were also presented, along with approaches to managing hyperkalaemia, hypotension, and deterioration of renal function during this treatment. The Congress also summarised guidance on lipid-lowering therapy in chronic kidney disease and addressed issues related to the management of heart failure in these patients. The problem with the diagnosis of chronic kidney disease was also highlighted, as it is associated with intensified progression of renal dysfunction and the development of cardiovascular complications.

Streszczenie

W dniach 25–27 września 2025 roku w Krakowie odbył się XXIX Międzynarodowy Kongres Polskiego Towarzystwa Kardiologicznego. Wśród licznych sesji nie zabrakło zagadnień dotyczących trudności w leczeniu i diagnostyce chorób układu sercowo-naczyniowego u pacjentów z przewlekłą chorobą nerek. W sprawozdaniu podsumowano przedstawione podczas Kongresu zalecenia dotyczące terapii chorób układu sercowo-naczyniowego u pacjentów z przewlekłą chorobą nerek. Omówiono wytyczne oraz trudności diagnostyczne związane ze stosowaniem leczenia przeciwkrzepliwego u pacjentów z przewlekłą chorobą nerek i migotaniem przedsionków. Przedstawiono ponadto zalecenia dotyczące blokady układu renina-angiotensyna oraz postępowanie w przypadku hiperkaliemii, hipotensji lub pogorszenia funkcji nerek podczas stosowania leków blokujących układ renina-angiotensyna. Podsumowano zalecenia dotyczące terapii hipolipemizującej w przewlekłej chorobie nerek oraz problemy związane z leczeniem niewydolności serca w tej grupie pacjentów. Przedstawiono również zagadnienie niedostatecznego rozpoznania przewlekłej choroby nerek, co wiąże się z szybszą progresją dysfunkcji narządu oraz rozwojem i progresją powikłań sercowo-naczyniowych.

Keywords: chronic kidney disease; cardiovascular complications; Congress of the Polish Cardiac Society

Słowa kluczowe: przewlekła choroba nerek; powikłania sercowo-naczyniowe; Kongres Polskiego Towarzystwa Kardiologicznego

DOI 10.53301/lw/215677

Received: 08.12.2025

Accepted: 16.12.2025

Published: 31.03.2026

Corresponding author:

Katarzyna Romejko
Military Institute of Medicine –
National Research Institute,
Department of Internal Medicine,
Nephrology and Dialysis,
128 Szaserów Str., 04-141 Warsaw
e-mail: kromejko@wim.mil.pl

Introduction

Cardiovascular complications – such as atherosclerosis, coronary artery disease, peripheral artery disease, hypertension, and heart failure – begin to develop at early stages of chronic kidney disease (CKD). As renal function declines, these conditions progress and ultimately represent the leading cause of death in this patient population. Currently, CKD affects approximately 10% of the global population, and its prevalence continues to rise. It is estimated that by 2040, CKD will become the fifth cause of death worldwide. Therefore, understanding the specific nature of cardiovascular complications in this group of patients is crucial. In clinical practice, cardiologists encounter individuals with CKD at various stages of the disease and with different cardiovascular disease severity. What matters most is not only implementing appropriate treatment strategies that slow down the progression of cardiovascular complications – and thereby improve survival – but also individualizing treatment, which remains one of the greatest challenges.

During the Congress, key issues related to the management of cardiovascular diseases in patients with CKD were discussed.

Report

Professor Beata Naumnik, MD, PhD (1st Department of Nephrology and Transplantation with Dialysis Centre, University Clinical Hospital in Białystok) presented the challenges associated with anticoagulant therapy in patients with CKD and atrial fibrillation. In this group, as the estimated glomerular filtration rate (eGFR) decreases, the risk of both thromboembolic complications and bleeding increases. Anticoagulant treatment with vitamin K antagonists (VKAs) should give way to direct oral anticoagulants (DOACs) – such as apixaban, rivaroxaban, and dabigatran – because of the numerous limitations of VKA therapy, including lack of efficacy, potential interactions with many drugs used in CKD, the risk of calcification and calciphylaxis, an increased risk of bleeding as well as ischemic complications, and the need for INR monitoring. The only situations in which VKAs must be used are antiphospholipid syndrome, the presence of a mechanical prosthetic heart valve, and moderate-to-severe rheumatic mitral stenosis.

DOAC dosing based on eGFR is as follows:

- eGFR >90 mL/min/1.73 m²: apixaban 2 × 5 mg, rivaroxaban 1 × 20 mg, dabigatran 2 × 150 mg;
- eGFR 50–90 mL/min/1.73 m²: apixaban 2 × 5 mg, rivaroxaban 1 × 20 mg, dabigatran 2 × 150 mg (same as for eGFR >90 mL/min/1.73 m²);
- eGFR 30–49 mL/min/1.73 m²: apixaban 2 × 5 mg, rivaroxaban 1 × 15 mg, dabigatran 2 × 110 mg;
- eGFR 15–29 mL/min/1.73 m²: apixaban 2 × 2.5 mg, rivaroxaban 1 × 15 mg; dabigatran is contraindicated.

Decision-making is particularly challenging in patients undergoing renal replacement therapy, who face an elevated risk of bleeding due to concurrent heparin use, platelet dysfunction, and a heightened risk of falls. Studies presented by Professor Naumnik indicate that apixaban offers greater benefits than warfarin in haemo-

dialysed patients, with a similar risk of thromboembolic events but fewer major bleeding episodes. However, because robust evidence is still lacking, treatment decisions should be made on an individual basis, with patients actively involved in the process. There are currently no studies involving patients undergoing peritoneal dialysis.

The lecture also highlighted the possibility of using the Dialysis Risk Score in haemodialysed patients (previous transient ischaemic attack or ischaemic stroke – 3 points; diabetes – 1 point; age >75 years – 1 point; gastrointestinal bleeding within the past year – minus 1 point) as an alternative to the CHA₂DS₂-VASc score for assessing thromboembolic risk. If the Dialysis Risk Score is below 2 points, DOACs should not be used. Additionally, left atrial appendage closure may be considered. With a score of ≥2 points, apixaban at a dose of 2 × 2.5 mg, rivaroxaban at 1 × 10 mg, or left atrial appendage closure may be used.

During the next lecture, Professor Marcin Adamczak, MD, PhD (Department of Nephrology, Transplantology and Internal Diseases, Medical University of Silesia in Katowice) presented recommendations regarding the use of renin-angiotensin system (RAS) blockade in patients with CKD stages G4/G5. In patients with non-diabetic CKD, RAS inhibitors (angiotensin-converting enzyme inhibitors, ACEIs, or angiotensin receptor blockers, ARBs) should be used at the highest tolerated dose, with benazepril, ramipril, and lisinopril remaining the preferred agents. Continuation of RAS inhibitor therapy is also recommended in patients with an eGFR <30 mL/min/1.73 m², whereas in individuals with diabetes particular caution is advised when eGFR falls below this threshold. It has been demonstrated that RAS inhibitors reduce the risk of CKD progression and prolong the time to initiation of renal replacement therapy (ACEIs and ARBs), decrease all-cause and cardiovascular mortality (ACEIs), and lower the risk of developing cardiovascular complications (ARBs). Therefore, continuation of therapy with these agents is of great importance.

The most common reasons for discontinuing RAS inhibitors in this group of patients are hypotension, hyperkalaemia, and worsening renal function. In the event of hypotension, modification of antihypertensive therapy is recommended, beginning with a reduction in the doses of antihypertensive drugs other than renin-angiotensin system (RAS) inhibitors. In cases of mild or moderate hyperkalaemia (serum potassium concentration 5.0 [5.5]–6.4 mmol/L), potassium supplements taken by the patient should initially be discontinued, and the patient should be advised to stop using potassium-containing salt substitutes. It should also be determined whether the patient is taking medications that increase serum potassium levels – such as non-steroidal anti-inflammatory drugs, heparin, co-trimoxazole, trimethoprim, or ketoconazole – and these drugs should be discontinued if necessary. If the patient has chronic constipation, appropriate treatment should be initiated.

Management of hyperkalaemia should not consist of discontinuing RAS inhibitors. Instead, it should include the use of loop diuretics, thiazide or thiazide-like diuretics, or chlorthalidone in patients with an estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m², sodium-

glucose co-transporter 2 (SGLT2) inhibitors, sodium bicarbonate in patients with metabolic acidosis, and either calcium patiromer or sodium zirconium cyclosilicate.

Deterioration of renal function after initiation of RAS inhibitor therapy is also a common reason for discontinuing these drugs. Experts suggest measuring serum creatinine 7–14 days after starting treatment or after increasing the dose. If serum creatinine increases by less than 30%, continuation of therapy is recommended. However, if it increases by 30% or more, discontinuation of these drugs is suggested and diagnostic evaluation for other causes of renal function deterioration should be undertaken, including dehydration, hypotension, the use of non-steroidal anti-inflammatory drugs, and the presence of renal artery stenosis.

Professor Marcin Barylski, MD, PhD (Department of Internal Medicine and Cardiac Rehabilitation, Medical University of Lodz) presented recommendations regarding lipid-lowering therapy in patients with CKD. These individuals belong to a high or very high cardiovascular risk group. The goal of lipid-lowering therapy is to reduce low-density lipoprotein cholesterol (LDL-C) concentrations below 55 mg/dL or by at least 50% from baseline. The target that achieves the lower LDL-C concentration should be chosen.

According to the 2019 European Society of Cardiology (ESC) guidelines for the management of dyslipidaemias, patients with CKD stages G3–G4 should be treated with statins or a combination of statins and ezetimibe. Due to reduced efficacy and a less favourable impact of lipid-lowering therapy in dialysis patients, continuation of therapy should be considered for those starting renal replacement therapy, particularly in patients with previously diagnosed atherosclerotic cardiovascular disease (ASCVD). In dialysis patients without diagnosed ASCVD, initiation of statin therapy is not recommended. The lipid-lowering agents recommended in CKD are statins, ezetimibe, and proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors. Among statins, atorvastatin is the safest option and can also be used in patients with an eGFR below 30 mL/min/1.73 m². Rosuvastatin may impair kidney function and is therefore contraindicated in patients with an eGFR below 30 mL/min/1.73 m², whereas pitavastatin may be used only at a dose of 2 mg.

PCSK9 inhibitors (alirocumab, evolocumab, and inclisiran) should be used with caution in patients with severe renal impairment. In cases of elevated plasma triglyceride levels in CKD, statins and eicosapentaenoic acid are indicated. If there is no improvement, the addition of fenofibrate may be considered. However, fenofibrate is contraindicated in individuals with an eGFR below 30 mL/min/1.73 m².

Professor Agata Bielecka-Dąbrowa, MD, PhD (Department of Cardiology and Adult Congenital Heart Diseases, Polish Mother's Memorial Hospital Research Institute in Łódź and Department of Preventive Cardiology and Lipidology, Medical University of Lodz) discussed the management of heart failure, specifically focusing on patients with CKD. In this population, implementing the full range

of recommended heart failure therapies is not always advised.

Treatment with spironolactone, eplerenone, and sacubitril with valsartan is contraindicated if eGFR is below 30 mL/min/1.73 m². Finerenone, however, may be used with an eGFR value of 25 mL/min/1.73 m². Dapagliflozin is not recommended if eGFR is below 25 mL/min/1.73 m², whereas empagliflozin is contraindicated when eGFR is below 20 mL/min/1.73 m². High oral doses of torsemide (100 mg and 200 mg) are intended for patients with an eGFR below 20 mL/min/1.73 m² or with a plasma creatinine concentration above 6 mg/dL, and treatment should be initiated at a dose of 50 mg per day.

In acute heart failure, CKD may be a factor predisposing to the development of diuretic resistance and, consequently, may lead to the need for renal replacement therapy – even in those who have not yet reached end-stage renal disease.

Renal replacement therapy in patients with acute heart failure is recommended in case of pulmonary oedema, uraemic encephalopathy, uraemic pericarditis, haemorrhagic diathesis, as well as hyperkalaemia and metabolic acidosis resistant to conservative treatment. No beneficial effect were demonstrated for low-dose dopamine or nesiritide added to diuretic therapy in acute heart failure in patients with CKD.

Professor Beata Naumnik, MD, PhD (1st Department of Nephrology and Transplantation with Dialysis Centre, University Clinical Hospital in Białystok) discussed the issue of CKD underdiagnosis. In Poland CKD affects 4.5 million individuals, and approximately 80,000 patients die prematurely each year due to renal impairment.

Because the symptoms of CKD are nonspecific or may be entirely absent, the condition is often not diagnosed until the stage 4 or 5. Since cardiovascular complications occur even at early stages of CKD, the diagnosis of impaired renal function is essential – not only to slow down the progression of kidney function decrease, but also to reduce the risk of cardiovascular complications, which remain the leading cause of death in this population.

During the lecture, it was emphasised that CKD screening, including the assessment of eGFR and the urinary albumin-to-creatinine ratio (ACR), should be performed particularly in the following high-risk groups: patients with diabetes (1 in 3 have CKD), patients with hypertension (1 in 5 have CKD), and patients with cardiovascular disease (2 in 5 have CKD). Kidney function should also be assessed in patients with a history of acute kidney injury and in those with a positive family history of CKD.

Summary

During the 29th International Congress of the Polish Cardiac Society, several sessions were dedicated to present guidelines for the management of cardiovascular complications in CKD. The recommendations for anticoagulant therapy in patients with CKD and atrial fibrillation were discussed. RAS blockade were also presented, together with protocols for managing hyperkalaemia, hypoten-

sion, or declining renal function during the therapy. The indications for initiating and continuing lipid-lowering therapy in CKD were summarised, as well as the specific challenges associated with heart failure treatment in this

patient population. Finally, the issue of CKD underdiagnosis was highlighted, as it contributes to the progression of kidney function decrease and the further development of cardiovascular complications.