# 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

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**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 m$^2$), 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 intesified. 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 m$^2$), 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 zadowalającym, 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

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## 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 $m^2$, 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 $m^2$, 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 $HCO_3$ 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 angiotensin 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 $m^2$, whereas the eGFR calculated based on creatinine alone was 2 mL/min/1.73 $m^2$. 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. Kazancioğlu 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 consewquences 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