REPORT FROM THE 15TH CONGRESS OF THE POLISH SOCIETY OF NEPHROLOGY

Sprawozdanie z XV Zjazdu Polskiego Towarzystwa Nefrologicznego

Katarzyna Romejko, Elżbieta Głuch, Anna Grzywacz, Magdalena Markowska, Mateusz Nowak, Dorota Górska-Michałek, Magdalena Wiśniewska

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

Katarzyna Romejko – 0000-0003-1447-2917
Elżbieta Głuch – 0009-0003-5229-256X
Anna Grzywacz – 0000-0001-6348-4215
Magdalena Markowska – 0009-0007-8015-872X
Mateusz Nowak – 0009-0003-4190-7928
Dorota Górska-Michałek – 0009-0001-5647-3937
Magdalena Wiśniewska – 0000-0002-1686-1142

Abstract

This year's Congress of the Polish Society of Nephrology was held in Katowice on June 12–14. During the Congress, the current recommendations of the Polish Society of Nephrology on modern diagnostic and therapeutic modalities for kidney diseases were presented. New possibilities of pharmacological nephroprotection in chronic kidney disease were discussed. Several lectures focused on the innovative treatment of hyperkalaemia. Also, the guidelines on the use of intravenous contrast agents and therapeutic strategies for reducing cardiovascular risk in patients with chronic kidney disease were presented. An important part of the Congress consisted of lectures on hereditary kidney diseases and modern treatment approaches in IgA nephropathy. Finally, the organized workshops provided an opportunity to review interesting clinical cases and update knowledge on the benefits of a low-protein diet in patients with chronic kidney disease.

Streszczenie

Tegoroczny Zjazd Polskiego Towarzystwa Nefrologicznego odbył się w Katowicach w dniach 12–14 czerwca. Podczas wydarzenia przedstawiono aktualne stanowisko Towarzystwa dotyczące diagnostyki oraz nowoczesnego leczenia chorób nerek. Omówiono możliwości farmakologicznej nefroprotekcji w przewlekłej chorobie nerek. Kilka wykładów poświęcono nowoczesnym metodom leczenia hiperkaliemii. Zaprezentowano także zalecenia dotyczące stosowania dożylnych środków kontrastowych w grupie pacjentów z upośledzoną funkcją nerek. Ponadto omówiono możliwości terapeutyczne pozwalające zmniejszyć ryzyko chorób układu sercowo-naczyniowego w przewlekłej chorobie nerek. Istotną część Zjazdu stanowiły wykłady dotyczące chorób nerek uwarunkowanych genetycznie oraz nowoczesnych metod leczenia nefropatii IgA. Warsztaty umożliwiły zapoznanie się z ciekawymi przypadkami klinicznymi oraz zaktualizowanie wiedzy na temat korzyści płynących ze stosowania diety ubogobiałkowej u pacjentów z przewlekłą chorobą nerek.

Keywords: chronic kidney disease; Congress of the Polish Society of Nephrology

Słowa kluczowe: przewlekła choroba nerek; Zjazd Polskiego Towarzystwa Nefrologicznego

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

The 15th Congress of the Polish Society of Nephrology was held at the International Congress Center in Katowice in June 12–14, 2025. Honorary patronage was provided by His Magnificence, the Rector of the Medical University of Silesia. During the Congress, the current position of the Polish Society of Nephrology on the diagnosis and modern therapeutic modalities for patients with kidney disease was presented. Therapeutic options for managing complications of chronic kidney disease (CKD), including electrolyte and acid–base disturbances, as well as cardiovascular (CV) complications, which represent the leading cause of mortality in this patient population, were discussed. Methods for preventing cardiovascular disease (CVD) in CKD and the potential for pharmacological nephroprotection were presented. The safety of intravenous contrast agents in patients with impaired renal function was also addressed. The challenges associated with renal replacement therapy and the difficulties encountered in outpatient care for kidney transplant recipients were discussed. A session devoted to hereditary kidney diseases, as well as a session addressing the diagnosis and current treatment guidelines for atypical haemolytic uremic syndrome (aHUS) was an important part of the lectures. Another important part of the Congress were workshops during which noteworthy cases of patients with kidney diseases were presented, peritoneal dialysis (PD) was discussed, and dietary recommendations in CKD were outlined, which are currently regarded as an essential component of nephroprotection.

During the Congress, participants had also the opportunity to take part in the 2.5 km or 5 km DAVITA RUN, as well as in a dinner get-together. These events allowed for exchanging experiences and observations and fostered the development of new friendships among nephrology enthusiasts.

This Report presents the key issues addressed during the Congress.

Workshops

The 15th Congress of the Polish Society of Nephrology began with workshops that enabled active participant engagement. The workshop entitled *"Nephrological Declinations: How to Unravel Complex Cases in a Simple Way in the Department of Nephrology"* addressed seemingly well-known and commonly encountered clinical entities, which may nevertheless raise questions regarding appropriate treatment.

Professor Tomasz Hryszko, MD, PhD (Second Department of Nephrology, Hypertension, and Internal Medicine with Dialysis Unit, Medical University of Białystok) presented the topic of fluid therapy and diuretic treatment in specific clinical scenarios. In cases of hypernatraemia of unclear aetiology, defined as sodium levels > 145 mmol/L, urine osmolality ≤600 mOsm/kg H_2O indicates renal water loss, that is, a deficiency of or resistance to antidiuretic hormone (ADH). In cases of a high urine osmolarity, defined as >1000 mOsm/24 hrs, hypernatraemia results from osmotic diuresis. When urine osmolality is >600 mOsm/kg H_2O, volaemic status should be assessed. Hypovolemia may result from gastrointestinal (e.g., diarrhoea) or cutaneous (increased sweating) fluid loss, whereas hypervolemia may be associated with excessive salt intake, including iatrogenic sodium load arising from intravenous hypertonic saline administration. It should be remembered that 5% glucose is used for rehydration in hypernatraemic patients. If hyponatraemia is suspected, defined as a serum sodium <135 mmol/L, serum osmolality should be evaluated, with values below 275 mOsm/kg H_2O indicating hypotonic hyponatraemia. Such disorders are often triggered by exacerbation of heart failure (HF), the treatment of which requires the use of diuretics that increase water excretion, which may in turn cause a drop in serum sodium. Renal failure with decreased urine output is another condition that can cause hypotonic hyponatraemia. Urine osmolality should also be assessed. For values below 100 mOsm/kg H_2O, the drop in serum sodium may be due to polydipsia, whereas osmolality >100 mOsm/kg H_2O indicates that hyponatraemia is associated with inappropriate antidiuretic hormone secretion, for example in hypothyroidism, adrenal insufficiency, or the syndrome of inappropriate antidiuretic hormone secretion (SIADH). It may also develop as a consequence of liver cirrhosis or nephrotic syndrome. The dose of diuretics should be adjusted based on urinary sodium and urine output. Urine sodium of 70 mmol/L and urine output of 100 mL/h within 2 hours after therapy onset indicate effective diuretic response. Otherwise, the diuretic dose should be doubled. When switching from intravenous to oral diuretics, it should be remembered that the oral furosemide dose should be twice the intravenous (IV) dose (e.g., 20 mg of IV furosemide corresponds to 40 mg oral dose), whereas torasemide maintains a 1:1 ratio (10 mg IV dose corresponds to 10 mg oral dose). Isotonic or hypertonic hyponatraemia is most commonly caused by severe hyperglycaemia. Pseudohyponatraemia is also clinically important, as it arises from markedly elevated lipid levels, leading to falsely low serum sodium despite normal plasma osmolality.

Alicja Rydzewska-Rosołowska, MD, PhD (Second Department of Nephrology, Hypertension, and Internal Medicine with Dialysis Unit, Medical University of Białystok) presented guidelines for the treatment of urinary tract infections (UTIs) during the workshop, taking into account the associated diagnostic challenges. According to the latest recommendations, first-line antibiotic therapy for UTIs should consist of a single oral dose of fosfomycin, a five-day course of Furazidin, or a three-day course of pivmecillinam or trimethoprim/sulfamethoxazole. It is important to note that longer-duration antimicrobial treatment does not improve therapeutic efficacy. Recurrent UTI is diagnosed when symptoms reappear within two weeks. However, if symptoms occur later, with a negative urine culture obtained between the two episodes, or if the infection is caused by a different pathogen, a reinfection is diagnosed. There are no clear recommendations for the prevention of recurrent UTIs. Pharmacotherapy may be considered (although it is not strictly recommended) in women with recurrent

infections. In such cases, continuous trimethoprim-sulfamethoxazole prophylaxis may be used at a dose of 40 mg/200 mg once daily or three times weekly, or furazidin at 50 mg or 100 mg daily. Postcoital pharmacotherapy is also used to prevent recurrent UTIs in women and may include trimethoprim/sulfamethoxazole at 40 mg/200 mg or 80 mg/400 mg, or furazidin 50 mg or 100 mg administered once after intercourse. In addition to pharmacological prophylaxis, increased water intake, methenamine, cranberry products containing proanthocyanidins, and vaginal oestrogens in postmenopausal women have demonstrated beneficial effects. The diagnosis of UTI itself is often uncertain. Current guidelines emphasize that diagnosis should be based on clinical symptoms rather than solely on laboratory findings or urinalysis. One of the studies discussed demonstrated that unpleasant urine odour does not always correlate with an ongoing infection. Urine colour and clarity may be influenced by multiple factors, including medications (e.g., green urine after the use of propofol or orange urine following the administration of rifampicin); therefore, these characteristics should not be interpreted as definitive indicators of UTIs.

A session entitled *"Nephroprotection and Diet – From Guidelines to Practice"* was a particularly valuable part of the workshop. In addition to weight loss, increased physical activity, smoking cessation, and reduced sodium intake, restricting dietary protein intake is one of the non-pharmacological methods of nephroprotection. During the lecture, Professor Michał Nowicki, MD, PhD (Department of Nephrology, Hypertension, Transplantation, and Internal Medicine, Medical University of Łódź) discussed the important role of protein restriction in nephroprotection. The kidneys are involved in the catabolism and excretion of protein metabolites. A high-protein diet leads to the accumulation of toxic protein metabolites, thereby promoting kidney damage. The average protein requirement for an adult is 0.66 g/kg of body weight per day, while the safe intake is considered at 0.83 g/kg/day, according to the dietary recommendations of most nutritional associations. Unfortunately, the current average protein intake in developed countries reaches 1.35 g/kg/day, which significantly exceeds the recommended values. The 2024 KDIGO dietary guidelines for non-dialysis patients with stage G3–G5 CKD suggest a protein intake of 0.8 g/kg/day. A very low-protein diet (0.3–0.4 g/kg/day) may be considered in patients at risk of end-stage renal disease; this should be supplemented with amino acids or amino acid ketoanalogues, which are nitrogen-free amino acid precursors that are converted into amino acids in the body, thereby preventing essential amino acid deficiency. This approach allows protein requirements to be met at a level of 0.55–0.6 g/kg/day. A low-protein diet has been shown to exert multiple beneficial effects: it slows the decline in estimated glomerular filtration rate (eGFR) and reduces nephron overload, improves insulin resistance, lowers oxidative stress, corrects metabolic acidosis, decreases the production of uremic toxins, reduces proteinuria, limits the severity of secondary hyperparathyroidism, improves the lipid profile, enhances the nephroprotective effects of the renin–angiotensin–aldosterone system (RAAS), and delays dialysis therapy. Reducing protein intake also leads to improved digestion, decreased postprandial discomfort, and reduced constipation. A low-protein diet additionally results in lower salt consumption, which in turn contributes to improved blood pressure control. Reduced serum phosphorus levels associated with a low-protein diet allow for a decrease in the use of GI phosphate-binding agents and, in the case of supplementation with keto-analogue amino acids, reduced dosage of calcium-containing medications. These benefits of a low-protein diet contribute to an improved quality of life in CKD patients. Delaying the initiation of dialysis therapy provides time for the formation and full maturation of an arteriovenous fistula, as well as for preparation for kidney transplantation or preemptive transplantation. To enable patients to initiate and adhere to a low-protein diet, it is essential to educate them about the benefits of dietary modification and to provide ongoing dietary education and monitoring, which often require consultation with and supervision by a dietitian experienced in the management of patients with kidney disease.

Diagnostic imaging with intravenous contrast agents in CKD patients. Position statement of the Polish Society of Nephrology and the Polish Society of Radiology

The first session, featuring lectures by Prof. Zbigniew Serafin, MD, PhD (Department of Radiology and Diagnostic Imaging, Faculty of Medicine, Collegium Medicum of Nicolaus Copernicus University in Toruń), Prof. Przemysław Rutkowski, MD, PhD (Medical University of Gdańsk), Prof. Dorota Kamińska, MD, PhD, and Prof. Magdalena Krajewska, MD, PhD (4th Military Clinical Hospital with Polyclinic SPZOZ in Wrocław, Faculty of Medicine, Wrocław University of Science and Technology), focused on the safety of IV contrast agents in patients with CKD. This is a crucial issue, as patients with kidney disease often experience difficulties related to contrast-enhanced imaging due to elevated renal parameters. During the session, new guidelines for the use of IV contrast media, developed in collaboration with the Polish Society of Radiology, were presented. Both scientific societies recommend assessing renal function using the CKD-EPI formula prior to the administration of iodinated contrast media (ICM). If this formula cannot be used, the MDRD equation should be used instead. The timeframe for assessing renal function is 3 months in patients without a history of CKD or with stable CKD, and 7 days prior to ICM in patients with rapidly declining renal function. Patients at risk of acute kidney injury following ICM administration were defined as those with creatinine levels >1.5 mg/dL and/or eGFR <30 mL/min/1.73 m^2, with this risk considered to be low. The decision to administer hydration prior to ICM should be made on an individual basis following a thorough assessment of the patient's hydration status. Furthermore, routine pharmacological prophylaxis before ICM administration is not recommended. There is no need to discontinue RAASis, nonsteroidal anti-inflammatory drugs (NSAIDs), diuretics, or sodium–glucose cotransporter-2 inhibitors (SGLT2i) prior to contrast adminis-

tration. Metformin should be discontinued only in patients with eGFR <30 mL/min/1.73 m², in accordance with the summary of product characteristics. Monitoring of renal function 48–72 hrs after ICM administration is recommended in all hospitalized patients with eGFR ≤30 mL/min/1.73 m². Outpatients should have their eGFR monitored if their clinical condition deteriorates.

The approach of determining the time interval between successive ICM doses has also changed. A 48-hour interval between administrations should be maintained in patients with eGFR <30 mL/min/1.73 m², whereas individuals with normal renal function and patients with eGFR >30 mL/min/1.73 m² may receive ICMs at 4-hour intervals. It is important to note that gadolinium-based contrast agents (GBCAs) used in magnetic resonance imaging (MRI) attenuate X-ray radiation after excretion into the urinary tract and may lead to interpretative errors in computed tomography (CT) of the urinary tract. Therefore, CT should precede MRI for abdominal examinations. For thoracic and CNS scans, the CT/MRI sequence is not critical. Concerns were raised in the past regarding the use of GBCAs in MRI due to an increased risk of nephrogenic systemic fibrosis (NSF) reported following their administration. In terms of the risk of NSF, GBCAs are classified as high-risk (gadodiamide, gadopentetate, gadoversetamide), intermediate-risk (gadobenate dimeglumine, gadoxetate disodium), and low-risk agents (gadobutrol, gadoterate meglumine, gadoteridol). For low-risk agents, the risk of NSF is estimated to be <0.07%. High-risk GBCAs have now been withdrawn in Europe. Consequently, routine assessment of renal function is not recommended prior to the administration of low-risk gadolinium agents, also in individuals with renal impairment. Renal function should be assessed when intermediate-risk agents are planned, which are approved only for biliary and liver imaging in individuals with renal impairment. Intra-arterial administration of ICMs (e.g., during coronary angiography, intravascular procedures involving the abdominal aorta, or interventional radiology) differs from intravenous administration in that it requires greater contrast agent volumes due to repeated injections during the procedure, and ICMs reach the renal arteries at higher concentrations. Consequently, the risk of acute kidney injury in these patients is higher than that associated with intravenous administration. It is recommended to consider intravenous hydration, temporarily discontinue metformin, and monitor renal function for 48–72 hrs after contrast exposure in patients with eGFR <45 mL/min/1.73 m² undergoing coronary angiography or intravascular abdominal aortic surgery. Similar precautions are recommended for patients with eGFR <30 mL/min/1.73 m² undergoing interventional radiology procedures.

Individuals with a solitary kidney have their eGFR assessed in the same manner as the general population, and subsequent management depends on the eGFR value and the type of planned procedure, as outlined above. ICMs may be administered to patients undergoing peritoneal dialysis or haemodialysis regardless of residual urine output, and additional haemodialysis is not required unless the patient develops overhydration.

The current guidelines facilitate diagnostic and therapeutic procedures for all patients, particularly those with renal impairment. However, their practical implementation requires formal modifications to National Health Fund (NFZ) regulations, especially with regard to the financing of contrast-enhanced imaging, as previously the reporting of plasma creatinine levels and eGFR values to the NFZ was a prerequisite for reimbursement.

Therapeutic approach to aHUS depending on the clinical profile and TMA trigger

Professor Katarzyna Krzanowska, MD, PhD (Department of Nephrology, Dialysis, Transplantation, and Internal Medicine, University Hospital in Krakow) discussed the diagnostic and therapeutic management of atypical haemolytic uremic syndrome (aHUS). This rare condition should be suspected in the presence of haemolysis, thrombocytopenia, and acute kidney injury. Dysregulation of the alternative complement pathway, triggered by environmental stimuli in genetically predisposed individuals, leads to endothelial and platelet activation, ultimately resulting in thrombotic microangiopathy. Several regulatory proteins, including factor H, membrane cofactor protein (MCP), and factor I, play a crucial role in the dissociation of the alternative pathway C3 convertase and the proteolytic degradation of C3b. Mutations in the genes encoding these proteins, which are identified in patients with aHUS, lead to uncontrolled complement activation. A comprehensive differential diagnosis is essential, as the diagnosis of aHUS is established after excluding other causes. The stages of the differential diagnostic process and the disease entities associated with the clinical presentation of aHUS are shown in Figure 1.

Laboratory and genetic workup may be supplemented with kidney biopsy, although this is not strictly required. Treatment should be initiated as early as possible, as delayed therapy is a poor prognostic factor for the recovery of renal function. In Poland, targeted therapy is available through the B.95 drug programme and incorporates eculizumab or ravulizumab. Additionally, plasmapheresis and procedures required for symptomatic management should be performed, such as blood transfusion for severe anaemia or haemodialysis in cases of severe renal failure.

Treatment of hyperkalaemia

During the Congress, considerable attention was devoted to the management of hyperkalaemia in CKD patients. On the first day, Prof. Marcin Adamczak, MD, PhD (Silesian Medical University in Katowice), delivered a lecture entitled *"Current treatment of hyperkalaemia, including the use of calcium patiromer"*. On the second day, the entire session was dedicated to this topic and was entitled *"A new therapeutic approach to hyperkalaemia—from pathophysiology to personalized therapy"*. The session consisted of four lectures delivered by Prof. Tomasz Stompór, MD, PhD (Department of Nephrology, Hypertension and Internal Medicine, University of Warmia and Mazury in Olsztyn), Prof. Ilona Kurnatowska, MD, PhD (Clinical Department

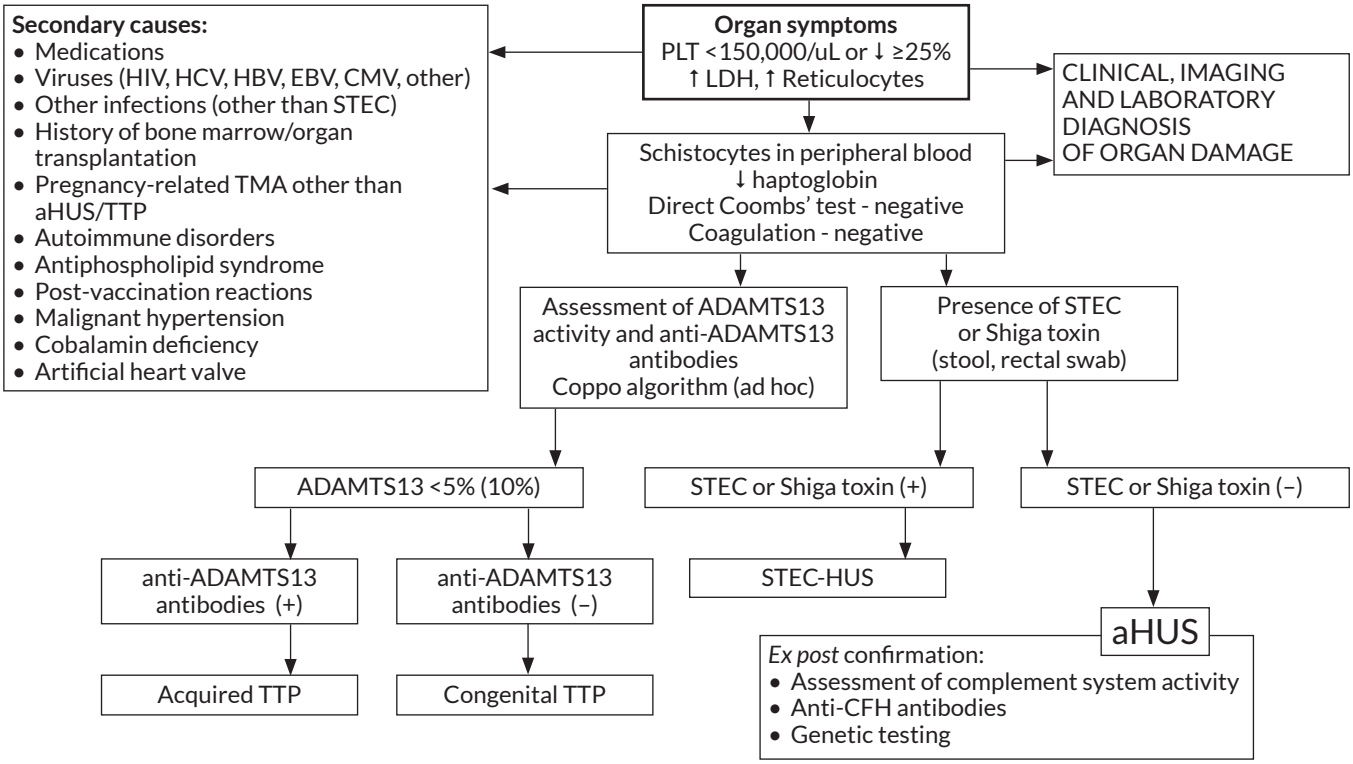

Figure 1. Suggested algorithm of TMA diagnosis

of Nephrology and Internal Medicine), N. Barlicki University Clinical Hospital in Łódź), Prof. Marcin Adamczak, MD, PhD (Medical University of Silesia in Katowice), and Prof. Beata Naumnik, MD, PhD (First Department of Nephrology, Transplantation and Internal Medicine with Dialysis Unit, Medical University of Białystok). On the final day of the Congress, Prof. Kazimierz Ciechanowski, MD, PhD (Department of Nephrology, Transplantation and Internal Medicine, Pomeranian Medical University), delivered a lecture entitled *"Hyperkalaemia in kidney diseases—aetiology, preventive methods, and current treatment options."* The therapeutic recommendations for hyperkalaemia discussed during the Congress are presented below.

Hyperkalaemia is defined as serum ionized potassium >5.0 mmol/L; however, in clinical practice, reference ranges recommended by individual laboratories should be considered. Hyperkalaemia is classified as mild (potassium >5.0–5.5 mmol/L), moderate (potassium 5.6–6.4 mmol/L), and severe (potassium ≥6.5 mmol/L). It may also be classified as acute or chronic. Pseudohyperkalaemia should be excluded, as it is common and occurs in up to 35% of patients with laboratory findings of elevated serum potassium. It most often results from improper blood sampling or haemolysis of red blood cells.

In cases of acute hyperkalaemia, treatment should be initiated immediately. Calcium preparations (calcium chloride or calcium gluconate; 1–2 ampoules administered intravenously) are used to stabilize the myocardial cell membrane in the management of acute hyperkalaemia. Additional measures include salbutamol (20 mg in 4 mL of 0.9% NaCl administered via nebulization, which reduces serum potassium by approximately 0.6 mmol/L), 8.4% sodium bicarbonate (40–60 mL administered intravenously, particularly in the presence of acidosis, reducing serum potassium by approximately 0.5 mmol/L), 10% glucose with insulin (1 unit of insulin per 4–5 g of glucose; for example, 250 mL of 10% glucose with 5–6 units of insulin administered intravenously, reducing serum potassium by approximately 0.5 mmol/L), intravenous infusion of 0.9% NaCl with furosemide (40–60 mg administered intravenously) and haemodialysis, which most effectively lowers serum potassium and is considered first-line therapy in patients receiving chronic haemodialysis or those with oliguria. In addition, polystyrene sulfonate (Resonium A; 30 g in 150 mL of water or a 10% glucose solution administered orally, and less frequently rectally) is commonly used in combination with agents that promote bowel movements (e.g., lactulose 30 mL orally), resulting in a reduction in serum potassium levels of approximately 0.5–1.0 mmol/L. The onset of action of polystyrene sulfonate is variable, ranging from several hours to several days. This agent acts in the large intestine and nonspecifically binds potassium ions in exchange for sodium ions. Common adverse effects of polystyrene sulfonate include gastrointestinal disturbances, hypernatraemia, hypokalaemia, and nonrespiratory alkalosis. Intestinal necrosis also represents a serious potential complication. or this reason, the drug is not recommended for chronic treatment.

Low-potassium diet, including the avoidance of potassium-containing salt substitutes, along with the use of diuretics that increase urinary potassium excretion (loop diuretics at eGFR <30 mL/min/1.73 m²) is recommended in the management of chronic hyperkalaemia in CKD patients. Sodium bicarbonate is advised (a decrease in pH by 0.1 increases serum potassium by ap-

proximately 0.4 mmol/L, and the target bicarbonate levels in CKD is 24–28 mmol/L) in patients with metabolic acidosis. Additionally, discontinuation of medications that promote hyperkalaemia, such as potassium supplements, NSAIDs, beta-blockers, and RAASis, or a reduction in their doses, should be considered. However, the approach to discontinuing RAASis in the context of hyperkalaemia has changed. Current recommendations emphasize maintaining RAASi therapy at the maximum tolerated dose whenever possible, given its long-term benefits in reducing cardiovascular risk.

The availability of two novel agents, calcium patiromer and sodium zirconium cyclosilicate, in Poland is a significant advancement in the management of chronic hyperkalaemia. Both are currently reimbursed for the treatment of hyperkalaemia in adult patients with G3b–G5 CKD who are receiving RAASis. As a result, these agents allow for continuing RAASi therapy, for example in the management of HF or HT. This is particularly important since CV complications represent the leading cause of mortality in CKD patients; therefore, the use of RAASis should not be limited in this patient population. Calcium patiromer is a nonabsorbable polymer that nonspecifically binds potassium ions in exchange for calcium ions in the distal large intestine, with an onset of action within 7 hrs after oral administration. Although this agent may cause gastrointestinal disturbances, hypokalaemia, and hypomagnesaemia, no serious adverse reactions have been reported. The recommended starting dose in adults is 8.4 g once daily. The dose may be adjusted as required within the range of 8.4–25.2 g daily to achieve target serum potassium levels. A minimum interval of 3 hrs should be maintained between patiromer and other oral medications. Sodium zirconium cyclosilicate, which selectively binds potassium ions in exchange for sodium ions, is another novel agent available for the chronic management of hyperkalaemia in adults. This agent has a rapid onset of action, occurring already 1 hour after ingestion, and binds potassium throughout the GI tract. It may also cause GI disturbances, hypokalaemia, and oedema; however, no serious adverse effects have been reported. Treatment consists of a correction phase (10 g three times daily), followed by maintenance therapy after reaching normokalaemia, which typically occurs within 24–48 hrs. Maintenance therapy is usually initiated at 5 g once daily, with the option to gradually increase the dose to 10 g once daily or reduce it to 5 g every other day, as needed; the lowest effective dose should be determined. Since it may transiently increase gastric pH, a minimum interval of 2 hrs should be maintained between the drug and orally administered medications with pH-dependent bioavailability. It is important to emphasize that, whenever possible, the inclusion of sodium-glucose cotransporter-2 (SGLT2) inhibitors should be considered in cases of hyperkalaemia. These agents are currently a cornerstone of nephroprotective therapy, as studies have demonstrated their beneficial effect in lowering serum potassium levels.

Hereditary kidney diseases – pathogenesis, diagnosis and treatment

During the session on hereditary kidney diseases, lectures were delivered by Magdalena Jankowska MD, PhD (Department of Nephrology, Transplantation and Internal Medicine, University Clinical Centre, Gdańsk); Prof. Michał Nowicki MD, PhD (Department of Nephrology, Hypertension, Transplantation and Internal Medicine, Medical University of Łódź); and Jakub Ruszkowski MD, PhD (Department of Nephrology, Transplantation and Internal Medicine, University Clinical Centre, Gdańsk), Prof. Krzysztof Pawlaczyk, MD, PhD (Department of Nephrology, Transplantation and Internal Medicine, Poznań University of Medical Sciences) and Prof. Ryszard Gellert, MD, PhD (Department of Nephrology and Internal Medicine, Medical Center for Postgraduate Education, Warsaw). Two clinical conditions were discussed during the session: autosomal dominant polycystic kidney disease (ADPKD) and Fabry disease.

ADPKD is the most common genetic cause of end-stage renal disease. It is caused by mutations in one of two genes encoding membrane proteins: *PKD1* (located on chromosome 16p13.13), which encodes polycystin-1 and accounts for 85–90% of cases, and *PKD2* (chromosome 4q21), which encodes polycystin-2 and accounts for 10–15% of cases. The absence of apparent *PKD1/PKD2* changes in several ADPKD-affected families may suggest a third locus. End-stage renal disease develops in approximately 70% of ADPKD patients at a mean age of 56 years, while average life expectancy ranges from 53 to 70 years, depending on the subtype, with shorter survival observed in *PKD1* compared to *PKD2* mutation. Patients with ADPKD experience a reduced quality of life, with depression, increased anxiety, low physical fitness, and increased pain sensitivity observed in approximately 60% of cases. Early diagnosis and timely intervention are critical for improving ADPKD prognosis. Lifestyle modifications are recommended as the first-line approach. Key measures include maintaining adequate hydration (at least 2–3 litres of fluids daily in patients with eGFR >30 mL/min/1.73 m² who are not treated with tolvaptan), adhering to a low-sodium diet, maintaining a healthy body weight, smoking cessation, engaging in regular physical activity, and limiting coffee consumption. Angiotensin-converting enzyme inhibitors (ACEi) are the first-line pharmacotherapy for HT. In cases of ACEi intolerance, angiotensin II receptor blockers (ARBs) may be used. Second-line agents include dihydropyridine calcium channel blockers and diuretics. Chronic administration of vasopressin V2 receptor antagonist tolvaptan, which slows cyst formation and CKD progression, is the only currently recommended treatment for ADPKD with proven efficacy. Tolvaptan is available in Poland through a dedicated drug programme. Serine-threonine kinase inhibitors (mTOR), metformin (in patients with diabetes), statins (for slowing disease progression), somatostatin analogues, SGLT2 inhibitors, ketogenic diet, complementary medicines, or dietary supplements are not recommended.

Fabry disease (FD), a lysosomal storage disorder caused by mutations in the gene encoding alpha-galactosidase A (GLA), resulting in a marked deficiency or inactivity of this enzyme, was another discussed genetic disorder. Despite being X-linked, symptoms can also manifest in women, with their severity depending on the pattern of

random X-chromosome inactivation. More than 1,170 variants of the GLA gene have been described. Enzyme deficiency results in the accumulation of glycolipids, including globotriaosylceramide (GL-3) and its deacylated form, globotriaosylsphingosine, in plasma and in multiple cell types throughout the body, particularly in the kidneys, myocardium, and nervous system, leading to organ damage and dysfunction. The accumulation of globotriaosylceramide is correlated with oxidative stress, complement activation, and inflammation, ultimately leading to progressive fibrosis and irreversible organ damage. In the kidneys, damage primarily involves vascular endothelial cells, mesangial cells, podocytes, and tubular epithelial cells. Renal dysfunction manifests as albuminuria, proteinuria, and impaired renal function, which can progress to end-stage renal disease. Typical extrarenal manifestations include acroparesthesia, hypohidrosis, reduced tolerance to extreme temperatures and physical exertion, sudden-onset asymmetric sensorineural hearing loss, gastrointestinal symptoms, and fatigue. Enzyme replacement therapy (ERT) with agalsidase alfa, the timely initiation of which improves cardiovascular and renal function, is the optimal treatment for FD. Its prompt use improves cardiovascular and renal function. Orally administered chaperone migalastat is another treatment option. By stabilizing mutant alpha-galactosidase A, it restores enzymatic activity and significantly slows the progression of renal failure. Clinical experience with migalastat is more limited than that with ERT, and its use is restricted to patients with eGFR >30 mL/min/1.73 m².

The aetiology of kidney failure remains unidentified in approximately 23% of patients receiving dialysis. Genetic testing in this population yields a definitive diagnosis in 10–40% of cases. To date, more than 300 genetic defects have been implicated in CKD. Advances in modern technologies allow for precise detection of mutations responsible for kidney disease. Given the expanding range of therapeutic options, early diagnosis is essential to enable timely initiation of appropriate treatment and to slow disease progression.

The 2025 pillars for pharmacological nephroprotection – position of the Polish Society of Nephrology

The issue of pharmacological nephroprotection in CKD patients was raised during many lectures. Nephroprotective agents have been categorized into several groups that constitute the pillars of treatment, with their use varying depending on the underlying aetiology of renal failure.

In cases of kidney disease developing secondary to diabetes mellitus (DM), five therapeutic components are considered essential:

- The first pillar involves metabolic control of DM, aiming to achieve a glycated haemoglobin level < 7%. Preferred agents include SGLT2is and glucagon-like peptide-1 (GLP-1) receptor agonists, such as semaglutide, liraglutide, and exenatide.
- Nephroprotection with SGLT2i or GLP1 receptor agonists, regardless of metabolic control, is the second pillar.
- The third pillar involves adequate blood pressure control with RAASis. Target blood pressure values are 130–139/70–79 mmHg, or 120–129/70–79 mmHg if well tolerated by the patient.
- RAASis as nephroprotective treatment at the highest tolerated dose (losartan and irbesartan are preferred) and finerenone are considered the fourth pillar.
- The fifth pillar involves correcting metabolic acidosis. For this purpose, sodium bicarbonate is administered, aiming to achieve a target HCO_{3-} levels of 24–28 mmol/L.

The aim of these interventions is to achieve glycaemic control, target blood pressure, and correction of metabolic acidosis within a three-month period.

In non-diabetic CKD, pharmacotherapy is largely analogous and focuses on antihypertensive agents. RAASis are preferred, with the aim of achieving a target blood pressure of ≤139/79 mmHg, or ≤129/79 mmHg if well tolerated (first pillar). These agents are administered at the highest tolerated doses, with benazepril, ramipril, and lisinopril being the preferred options (second pillar). SGLT2is, constituting the third pillar of therapy, are also used. Empagliflozin should not be initiated in patients with eGFR <20 mL/min/1.73 m², and dapagliflozin should not be used in those with eGFR <25 mL/min/1.73 m². Once initiated, treatment should be continued until the commencement of renal replacement therapy. Sodium bicarbonate should also be administered to achieve HCO_3^- level of 24–28 mmol/L (fourth pillar). In this patient population, the therapeutic goals additionally include achieving target blood pressure and correcting metabolic acidosis within a three-month period.

Reducing CV risk in CKD patients

The issue of CV complications in patients with CKD was discussed by Prof. Andrzej Więcek, MD, PhD (Department of Nephrology, Transplantation and Internal Medicine, Silesian Medical University in Katowice); Prof. Piotr Jankowski, MD, PhD (First Department of Cardiology and Interventional Electrocardiology and Arterial Hypertension, Jagiellonian University); Daniel Śliż, MD, PhD (Third Department of Internal Medicine and Cardiology, Medical University of Warsaw); and Prof. Jolanta Małyszko, MD, PhD (Department of Nephrology, Dialysis, and Internal Medicine, Medical University of Warsaw). CV complications, including HT, atherosclerosis, left ventricular hypertrophy, HF, and chronic coronary syndrome (CCS), develop in the early stages of renal failure and progress with declining eGFR, with their greatest severity seen in patients on renal replacement therapy. These complications represent the leading cause of mortality in CKD patients. According to the 2021 European Society of Cardiology Guidelines on Cardiovascular Disease Prevention in Clinical Practice, cardiovascular risk stratification in CKD patients is based on eGFR and the albumin-to-creatinine ratio (ACR). Consequently, the severity of albuminuria is regarded as the primary marker of CV risk in this population. According to these guidelines, CKD patients are classified as having

either high or very high CV risk. In addition to classical risk factors present in the general population, such as obesity, HT, dyslipidaemia, metabolic syndrome, lack of physical activity, smoking, chronic inflammation, and intestinal dysbiosis, CKD patients exhibit a range of non-classical risk factors that contribute to the progression of cardiovascular pathology. These factors include disturbances in calcium and phosphate metabolism, such as secondary hyperparathyroidism, elevated fibroblast growth factor 23 (FGF23) levels, and Klotho protein deficiency, as well as anaemia, fluid overload, metabolic acidosis, and nutritional disorders, including protein-energy wasting and malnutrition, which are associated with an intensified inflammatory response and accelerated atherosclerosis (malnutrition–inflammation–atherosclerosis, MIA). Additional contributors include sarcopenia, vitamin D and K deficiency, and increased levels of uremic toxins. Elevated fibroblast growth factor 23 (FGF23) levels in CKD patients have been shown to contribute to the development of HF by promoting myocardial fibrosis and left ventricular hypertrophy. Furthermore, FGF23 has been identified as an independent risk factor for mortality in patients with CKD stages G2–G4.

Metabolic disorders, CKD, and CVD are components of the cardiovascular-kidney-metabolic (CKM) syndrome. Although each represents a distinct clinical entity, the presence of one can exacerbate the course of the other two. It is now recognized that hyperglycaemia and insulin resistance, increased inflammation and oxidative stress, activation of the renin–angiotensin–aldosterone system, and neurohormonal dysregulation contribute to cardiac and renal dysfunction, and that impairment of one organ adversely affects the other. Obesity represents a key contributor to CKM syndrome. Increased adipose tissue leads to enhanced synthesis of inflammatory cytokines, such as tumour necrosis factor alpha and interleukin-6, as well as free fatty acids. These factors exacerbate insulin receptor dysfunction and result in elevated plasma glucose. Elevated levels of free fatty acids also lead to increased hepatic synthesis of triglycerides and very low-density lipoprotein cholesterol (VLDL-C), as well as an increase in low-density lipoprotein cholesterol (LDL-C), which is more atherogenic. Obesity is also linked to lower adiponectin levels. Additionally, activation of the sympathetic nervous system and RAAS in obesity leads to endothelial dysfunction, decreased nitric oxide synthesis, HT, and left ventricular hypertrophy, thereby promoting atherosclerosis and HF. Multiple pathophysiological processes observed in obesity and diabetes, including hyperfiltration, oxidative stress, increased inflammation, and profibrotic mechanisms, impair renal function and contribute to the development of CKD. HF exacerbates renal dysfunction, while renal impairment promotes CV complications. CKM syndrome is classified into four distinct stages. Stage 1 is characterized by excess body fat. Stage 2 includes impaired renal function and/or hypertriglyceridemia, type 2 DM, metabolic syndrome, and HT. Stage 3 is additionally characterized by subclinical atherosclerotic cardiovascular disease or subclinical HF. Stage 4 CKM is defined as clinical CV complications, including chronic coronary syndrome, HF, stroke, peripheral arterial disease, and atrial fibrillation. The risk of mortality increases with each stage of the syndrome, rising from 5.5% in stage 1 to 15.2% in stage 4.

Patients with CVD receive multifaceted pharmacotherapy for CVD. SGLT2 inhibitors are used for both HF (with class IA recommendations from the European Society of Cardiology) and CKD. Blood pressure control is essential in this population, with RAASis serving as the primary agents. Sacubitril/valsartan has been shown to slow the progression of CKD, reduce albuminuria, decrease the risk of CV complications, and improve prognosis in patients with renal failure. Lipid-lowering therapy in CKD is primarily based on statins and, when indicated, ezetimibe, both of which reduce CV risk.

It is also possible to reduce CV risk n patients undergoing renal replacement therapy, who exhibit the most advanced CV complications and the highest mortality rates. In this population, selection of the optimal modality of renal replacement therapy, such as standard haemodialysis, haemodiafiltration, peritoneal dialysis, home dialysis, or daily dialysis, as well as extension of dialysis duration, are of critical importance. Blood pressure control is of critical importance and should be achieved using an appropriately determined so-called "dry weight," defined as the weight at which there are no signs of overhydration and blood pressure is within the normal range, whereas further reduction in dietary sodium intake or body water content may result in hypotension. Preservation of residual diuresis reduces the risk of fluid overload and electrolyte disturbances. The choice of vascular access for dialysis is of critical importance, as the use of intravascular catheters is associated with a higher risk of infectious and thromboembolic complications compared with arteriovenous fistulas (AVFs). In patients with severe HF, however, it should be carefully considered whether an AVF is truly the optimal option, as upper-extremity fistulas in particular may increase cardiac output. If an AVF is considered appropriate in such patients, a low-flow fistula or graft is preferred. The lowest left ventricular ejection fraction (EF) at which vascular access can be established is 10%. Kidney transplantation, which significantly reduces CV mortality remains the most effective treatment option for patients with end-stage renal disease.

IgA nephropathy – morphology, clinical picture and limits of interpretation

Prof. Agnieszka Perkowska-Ptasińska, MD, PhD (Department of Pathomorphology, Medical University of Warsaw), presented histopathological images of IgA nephropathy (IgAN). The pathogenesis of this disease involves abnormal glycosylation of immunoglobulin A1, leading to increased production of under-galactosylated IgA1, formation of IgA and IgG autoantibodies against Gd-IgA1 forming complexes with Gd-IgA1, and subsequent deposition of these complexes in the mesangium. This gives rise to mesangial cell activation followed by complement activation. Stimulation of the immune system results in overproduction of cytokines and growth factors, recruitment of inflammatory cells,

matrix expansion, and mesangial cell proliferation, all of which ultimately impair renal function. Mesangial proliferation and focal segmental glomerulosclerosis (FSGS) are the most common light microscopic findings in IgA nephropathy. Segmental loop necrosis and segmental crescent formation have also been described. Immunofluorescence microscopy demonstrates IgA deposits, as well as complement component C3, IgG, and IgM. Electron microscopy reveals mesangial deposits, alterations in podocyte structure, segmental thinning of the glomerular basement membrane, and, in some cases, splitting of the glomerular basement membrane. These histopathological findings allow for predicting the patient's future prognosis. Changes such as increased capillary cellularity or podocyte hypertrophy within sclerotic areas are associated with a poorer prognosis and should be taken into account during patient assessment, alongside established clinical predictors of progression to end-stage renal disease, including proteinuria exceeding >1 g/day, HT, and reduced eGFR at diagnosis.

Differentiating IgA nephropathy from type IV collagen disease (Alport syndrome) poses particular challenge due to the presence of glomerular basement membrane thinning in both conditions and the fact that they may coexist in the same patient, especially when the GBM thickness is <200 nm. Additionally, IgA deposits may be present in other clinical entities, such as infection-related glomerulonephritis or monoclonal gammopathy of renal significance (MGRS), which further complicates the interpretation of histopathological specimens.

IgA nephropathy – from pathogenesis to treatment

During another lecture, Prof. Tomasz Hryszko, MD, PhD (Second Department of Nephrology, Hypertension and Internal Medicine with Dialysis Center, Medical University of Białystok) discussed the current guidelines for the management of IgA nephropathy. According to the 2021 KDIGO recommendations, nephroprotective treatment with RAASis is the cornerstone of therapy for patients with proteinuria > 0.5 g/day. In patients with persistent proteinuria >1 g/day despite at least three months of optimized nephroprotective therapy and with eGFR >30 mL/min/1.73 m², immunosuppressive treatment with glucocorticoids should be considered. Emerging therapeutic approaches for IgA nephropathy include nephroprotective therapy with atrasentan (endothelin receptor antagonist), targeted-release formulation (TRF) budesonide, and sibeprenlimab.

Atrasentan is a selective endothelin-1 receptor antagonist (a vasoconstrictor peptide produced primarily by vascular endothelial cells). It significantly decreases proteinuria and exerts nephroprotective effects on the glomeruli. In a randomized, double-blind, phase 3 clinical trial published in 2025 in *The New England Journal of Medicine*, Heerspink et al. [1] evaluated the efficacy and safety of atrasentan in patients with biopsy-confirmed IgA nephropathy. After 36 weeks of treatment in a cohort of 270 patients, the drop in UPCR was significantly greater in the atrasentan group (−38.1%) than in the placebo group (−3.1%). No significant differences in the incidence of adverse events were observed between the two groups. Atrasentan is currently approved for use in the United States. Budesonide, administered as an oral modified-release formulation, acts locally in the distal ileum by inhibiting mucosal B cells within Peyer's patches, which are a major site of production of aberrantly glycosylated IgA1 (Gd-IgA1). In this way, budesonide exerts a targeted effect at the early stage of the pathogenic cascade leading to IgA nephropathy. Increased first-pass metabolism limits the drug's systemic bioavailability (estimated at approximately 10%), reducing the risk of treatment-related adverse events (mainly infections). The most commonly reported mild to moderate adverse events include acne, peripheral and facial oedema, weight gain, and increased white blood cell count.

Sibeprenlimab (VIS649) is a humanized IgG2 monoclonal antibody that binds to and neutralizes the activity of proliferation-inducing ligand (APRIL). APRIL, a member of the TNF superfamily, indirectly regulates immunoglobulin production by modulating the maturation and differentiation of B cells, including Gd-IgA1 antibodies, which play a key role in the pathogenesis of IgA nephropathy. Preliminary results reported by Mathur et al. [2] in 2024 demonstrated a significant reduction in proteinuria (from 47.2 ± 8.2% to 62.0 ± 5.7%, depending on the dose) in patients with IgA nephropathy treated with sibeprenlimab over a 12-month period vs placebo (20.0 ± 12.6%).

In summary, the current guidelines for the treatment of IgA nephropathy, which are based on conventional nephroprotective therapy with RAASis and, when indicated, immunosuppressive treatment with systemic GCs, will be gradually supplemented with more contemporary agents, such as atrasentan, targeted-release formulation budesonide, and sibeprenlimab. These therapies create the opportunity for more effective treatment with a lower risk of adverse effects.

How to break the inertia barriers: an interdisciplinary approach to CKD therapy

The session on the interdisciplinary approach to the treatment of CKD was led by Prof. Agnieszka Pawlak, MD, PhD (Department of Cardiology, Central Clinical Hospital of the Ministry of Interior and Administration, Warsaw), Prof. Krzysztof Pawlaczyk, MD, PhD (Clinical Department of Nephrology, Transplantation and Internal Medicine, Poznań University of Medical Sciences), and Prof. Michał Holecki, MD, PhD (Department of Internal Medicine, Autoimmune and Metabolic Diseases, Medical University of Silesia in Katowice). During the session, several important issues related to the initiation and continuation of CKD treatment, as well as organizational challenges in patient care, were discussed. Possible causes of delays in the initiation of optimal therapy were identified, which may lead to off-label treatment, including the use of suboptimal doses, omission of certain medications, or failure to implement currently recommended therapies. This issue is multifactorial and can be categorized into three main domains: factors

Figure 2. Participants of the 15th Congress of the Polish Society of Nephrology from Department of Internal Diseases, Nephrology and Dialysis, Military Institute of Medicine-National Research Institute with the Head of the Clinic, Professor Stanisław Niemczyk. From left: Magdalena Markowska, Mateusz Nowak, Katarzyna Romejko, Professor Stanisław Niemczyk, Anna Grzywacz, Elżbieta Głuch, Magdalena Wiśniewska, Dorota Górska-Michałek

related to the organization of the healthcare system, constraints associated with physicians' workload and clinical practice, and insufficient patient perception and understanding of the treatment process. The first domain encompasses the lack of clear recommendations from scientific societies and expert panels, difficulties in establishing optimal follow-up schedules, limited access to modern medications, communication barriers among healthcare professionals, and insufficient substantive support, including the absence of an interdisciplinary approach, which hinders effective collaboration between specialists. The second category, related to limitations in clinical practice, encompasses challenges in implementing comprehensive therapy. These include time constraints during clinical visits that impede optimal therapeutic decision-making, concerns regarding the financial burden of modern treatments for patients, problems with patient adherence, communication difficulties between physicians and patients, limited access to information on novel therapeutic options, and the absence of clear and practical treatment guidelines. The third category relates to patients' inadequate perception and understanding of the therapeutic process. This includes denial of disease presence or progression, fear of adverse effects associated with new treatments, misunderstanding of the principles of modern therapies, reluctance to receive IV treatments or polypharmacy, and financial inability to bear the costs of therapy. These challenges may impede the implementation of optimal treatment not only in CKD but also in other clinical conditions. Knowledge of these barriers is essential for the effective implementation and maintenance of therapies aligned with current medical evidence.

During the Congress, Janusz Ostrowski, MD, PhD (Postgraduate Medical Education Center), delivered a special lecture marking the 50th anniversary of the establishment of the Department of Nephrology, Transplantation, and Internal Medicine at the Medical University of Silesia in Katowice, entitled *"History of Silesian Nephrology"*, while Prof. Peter Stenvinkel (Division of Renal Medicine, Department of Clinical Science, Intervention and Technology, Karolinska Institutet, Stockholm, Sweden), an Honorary Member of the Polish Society of Nephrology, delivered a lecture entitled "*Learning from nature to make everyone healthier – a concept of planetary health*".

Conclusions

The 15th Congress of the Polish Society of Nephrology focused in contemporary diagnostic and therapeutic strategies for patients with kidney disease. Considerable attention was devoted to current guidelines for the management of complications of CKD, including electrolyte and acid–base disturbances. Novel thera-

peutic options and preventive strategies for cardiovascular disease in CKD patients, recognized as the leading cause of mortality in this population, were discussed in detail. Nephroprotective modalities and the safety of intravenous contrast agents in patients with impaired renal function were also addressed. A substantial portion of the Congress was devoted to sessions on hereditary kidney diseases, as well as lectures focusing on the diagnosis and current therapeutic guidelines for aHUS. The workshops, which included engaging case studies of patients with kidney disease, a presentation on peritoneal dialysis, and a discussion of dietary recommendations for CKD, attracted a lot of interest. The audience had the opportunity to ask questions, participate in discussions, and share their observations and concerns after each session. The 15th Congress was also an excellent opportunity for nephrologists to meet, exchange experiences, and establish new professional and social contacts.

References

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