ŻUBEREK, Michal, POPIŃSKA, Zuzanna, ŚLUSARCZYK, Daniel, ŻMUDA, Bartłomiej, JAKUBOWSKA, Wiktoria, PISERA, Piotr, KIEŁKOWICZ, Aleksandra and PACTWA, Filip. A review on the classification and current treatment of Chronic Kidney of Education. Health and 2023;49(1):86-106. 2391-8306. Disease. Journal Sport. eISSN https://dx.doi.org/10.12775/JEHS.2023.49.01.006 https://apcz.umk.pl/JEHS/article/view/47543 https://zenodo.org/records/10439896

The authors declare that there is no conflict of interests regarding the publication of this paper. Received: 18.12.2023. Revised: 26.12.2023. Accepted: 27.12.2023. Published: 30.12.2023.

A review on the classification and current treatment of Chronic Kidney Disease

Michał Żuberek, Faculty of Medicine Nicolaus Copernicus University in Torun, Collegium Medicum in Bydgoszcz, Jagiellonska 13, 85-067 Bydgoszcz, Poland https://orcid.org/0009-0008-2358-6784 zuberekmichal99@gmail.com

Zuzanna Popińska, Faculty of Medicine, Comenius University Bratislava Špitálska 24, 813 72 Bratislava, Slovakia https://orcid.org/0000-0002-8224-6770 Zuzpopinska@gmail.com

Daniel Ślusarczyk, Military Medical Academy Memorial Teaching Hospital – Central Veterans' Hospital, ul. Żeromskiego 113, 90-549 Lodz, Poland https://orcid.org/0009-0000-3338-976X dslusarczyk98@gmail.com

Bartłomiej Żmuda, Norbert Barlicki Memorial Teaching Hospital No.1 of the Medical University of Lodz, Stefana Kopcinskiego 22, 90-153. Lodz, Poland https://orcid.org/0009-0005-6290-0455 zmudabartek98@gmail.com

The journal has had 40 points in Ministry of Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 03,11.2023 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Health Sciences (Field of medical and health sciences); Medical sciences (Field of medical and health sciences); Cultural and religious studies (Field of medical and health sciences); Science-comomic geography and spatial management (Field of social sciences); Pedagogy (Field of social sciences); Cultural and religious studies (Field of needical and health sciences); Pedagogy (Field of social sciences); Pe

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Wiktoria Jakubowska, Medical University of Lodz, al. Tadeusza Kościuszki 4, 90-419 Łódź, Poland https://orcid.org/0009-0008-9290-503X wiktoria.jakubowska@stud.umed.lodz.pl

Piotr Pisera, Faculty of Medicine, Medical University of Lodz, al. Tadeusza Kościuszki 4, 90-419 Łódź, Poland https://orcid.org/0009-0002-7086-7307 ptrpsr5@gmail.com

Aleksandra Kiełkowicz, Central Clinical Hospital of Medical University of Lodz, Pomorska 251, 92-213 Lodz, Poland https://orcid.org/0009-0003-7837-0925, aleksandra.kielkowicz@gmail.com

Filip Pactwa, Faculty of Medicine, Medical University of Lodz, Kościuszki 4,90-419Lodz,Polandhttps://orcid.org/0000-0002-9559-5072filip.pactwa@onet.plCorresponding author:Michał Żuberek Faculty of Medicine Nicolaus Copernicus University in Torun, CollegiumMedicum in Bydgoszcz, Jagiellonska 13, 85-067 Bydgoszcz, Poland+48 728 943 883zuberekmichal99@gmail.com

Abstract

Introduction:

Chronic Kidney Disease (CKD) is a chronic condition which is characterized by the gradual loss of kidney function over time leading to End-Stage Renal Disease (ESRD). Role of kidneys is very vital to maintain homeostasis of the system. CKD is classified into five stages based on the estimated Glomerular Filtration Rate (eGFR). CKD is a long-term condition

which cannot be completely cured. Treatment focuses on managing underlying conditions, slowing disease progression and addressing complications.

Aim of the study:

Purpose of this study is to encapsulate available knowledge about classification and therapeutic options for patients with Chronic Kidney Disease and End-Stage Kidney Disease. Both old and new treatment methods have been summarized in the following publication.

Material and methods:

Literature available in the PubMed database was reviewed using the following keywords: "Chronic kidney disease", "ACE inhibitors", "Angiotensin II Type 1 Receptor Blockers", "Diabetic Kidney Disease", "End-Stage Kidney Disease", "Dapagliflozin",

Conclusions:

Chronic Kidney Disease is a condition which is progressive and affects more than 10% of the general population worldwide totaling over 800 million people. There are many causes of chronic kidney disease; therefore, it is important to focus on slowing the progression of the disease, minimizing complications and modifying risk factors.

Keywords: Chronic Kidney Disease; Kidney Failure; Chronic Treatment;

Introduction

Chronic kidney disease (CKD) signifies an irreversible deterioration of kidney function that progresses over time and develops over many years. Initially it is indicated solely by abnormalities in biochemical parameters, such as elevated levels of creatinine, urea, and the presence of albuminuria. However, as the loss of kidney function advances, subjective and objective symptoms of renal failure emerge, collectively referred to as uremia [1,2].

Referring to the text by John S. Thurlow et al. during the period from 2003 to 2016 stabilization of the incidence of CKD was observed in many more developed countries during

the period from 2003 to 2016, but a significant increase in incidence was also noted in Asian regions, Furthermore, the previously mentioned work highlights the increasing popularity of end-stage kidney disease (ESKD) treatment, which may stem from improved quality of life for patients and increased access to kidney replacement therapy in countries experiencing economic development. [3. Thurlow]. According to Kitty J. Jager et al the increase in risk factors among the population contributing to the development of chronic kidney disease, such as hypertension, obesity, and diabetes, has led to a rise in the number of patients affected by CKD, reaching nearly 840 million people worldwide in 2017, the same conclusions can be found in paper presented by Casaba P. Kovesdy [4,5]. The study aims to compile existing knowledge on the classification and therapeutic choices for treating Chronic Kidney Disease and End-Stage Kidney Disease. It covers overview of diagnostic and treatment alternatives, encompassing the latest methodologies.

Definition and stages of advancement

According to the latest edition of the KDIGO guidelines from 2012, There are 5 stages of advanced CKD, with KDIGO distinguishing two categories for stage 3, namely G3a and G3b. The range of glomerular filtration rate required to assign a patient to the appropriate category is described as follows: G1; \geq 90 ml/min/1.73m²; G2 60-89 \geq 90 ml/min/1.73m², G3a; 45-59 ml/min/1.73m², G3b; 30-44 ml/min/1.73m², G4; 15-29 ml/min/1.73m², G5 <15 ml/min/1.73m² [6]. The last of the aforementioned is a category referred to in the nomenclature as end-stage renal failure.[7,8,9].

The diagnosis of CKD is based on laboratory and imaging tests. To establish a diagnosis, one of evident for the following should be more than 3 months: or more of the following indicators that kidney 1. One suggests damage: -albuminuria (daily excretion of albumin in urine \geq 30 mg/day; Albumin-to-creatinine ratio \geq 30 mg/g),

-Electrolyte disturbances typical for CKD patients (hyperkalemia, hyponatremia, metabolic acidosis)

-Structural abnormalities detected in imaging studies (ultrasound showing reduced kidney size, effacement of the kidney outline)

-Abnormalities in histopathological examination indicating glomerular nephritis or other causes of CKD

-Reduced glon	nerular filtratio	on rate <60 r	nl/min/1.73m ² (corr	esponding to ca	ategories G3a-G5
according	to	the	KDIGO	2012	guidelines).

Methods for estimating and measuring Glomerular filtration rate (GFR)

As mentioned earlier, one of the diagnostic criteria for CKD and ESKD is a GFR <60 ml/min/1.73m² persisting for at least 3 months. It is essential to note that GFR generally decreases by 1-2 ml/min/1.73m² per year. Therefore, the natural decline in GFR with age suggests that nearly every individual above the age of 70 may meet both the time and reduced GFR criteria. However, this statement should be approached with caution because the development of CKD depends on various individual factors. Consequently, each doctor should take an individualized approach to assess the patient's health [2]. Additionally, considering the normal range for creatinine concentration in adult men (0.8 to)1.4 mg/dl) and women (0.6 to 1.1 mg/dl), it can be inferred, according to the study conducted by Ruyum Jin et al., that the eGFR value is inversely proportional to both age and creatinine concentration. In other words, as both age and creatinine concentration increase, eGFR decreases. There various methods available are for calculating GFR [10]. There are several methods for calculating GFR, with the ones listed below being commonly clinical used in general practice: 1. MDRD (Modification of Diet in Renal Disease): This method utilizes the MDRD equation to calculate GFR based on the patient's age, gender, serum creatinine concentration, and race. 2. CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration): A more modern method, also based on creatinine concentration, age, gender, and race, which provides more accurate MDRD equation, results than the especially at higher GFR values. 3.Cockcroft-Gault: This method uses a formula based on weight, gender, age, and serum creatinine level.

4.Creatinine clearance: This method involves comparing the concentration of creatinine in the serum before and after the administration of a substance containing a known amount of creatinine. It calculates how much creatinine is removed from the blood per unit of time. 5.Cystatin C: Cystatin C is a substance that is filtered by the kidneys and is neither reabsorbed nor secreted by the renal tubules. Its concentration in the blood can be used to estimate GFR.

Taking into account the work of Lesley A. Inker, the most optimal method for estimating GFR is the CKD-EPI formula [11].

Risk Factors

CKD is a highly complex medical condition that can develop in the course of other medical conditions, which, in turn, represent potentially modifiable risk factors for the occurrence of this disease phenomenon. Among these risk factors, it is necessary to distinguish between those that can be modified and those mentioned earlier, the progression of of which be altered to reduce the risk CKD can occurrence. Among the non modifiable risk factors for the occurrence of CKD we include: older age, male gender, elderly[2szczeklik,11inker,12 usherood,]. However, among the potentially modifiable factors, it is necessary to mention: the extent of proteinuria, arterial hypertension[12,13], hyperglycemia [13,14], hyperlipidaemia[14,21]. In the case of cigarette smoking, the meta-analysis by Jia Xia et al. suggests evidence confirming that smoking is an independent risk factor for the incidence of chronic kidney disease (CKD) and emphasizes the need for further research to confirm whether smoking cessation can reduce the occurrence of CKD in the general adult population[15]. Furthermore, in this subsection, it is important to mention that there are many factors that can exacerbate CKD such as: Hyperkalemia [16], dehydration [17], Obstruction in the urine flow (e.g., in the course of kidney stones) [18], Hypotension, for example, caused by the use of antihypertensive medications [19], nephrotoxicity of drugs such as NSAIDS/ACEI/ARB [20], hyperlipidaemia [21], diabetes[14].

Therefore, it can be boldly stated that providing care for a patient with CKD poses a challenge for the physician, due to, among other things, the aforementioned examples of risk factors that can exacerbate the course of CKD. It is worth noting that many drugs such as ACEI and ARB used to achieve one of the main goals of CKD treatment, namely reducing proteinuria, may pose a significant threat to the health and life of the patient.

Etiology

There are many causes leading to CKD or ESKD, but generally, they can be categorized based on functional or anatomical abnormalities of the kidneys. These changes consequently result in a reduction in the number of active functional units of the kidney, i.e., nephrons, which ultimately leads to a gradual decrease in GFR. According to Satyanarayana R. Vaidya et al. The global causes of CKD are diverse, and the predominant primary conditions leading to CKD and eventual end-stage renal disease (ESRD) include: Diabetes mellitus type 2 (30% to 50%), Hypertension (27.2%), Primary glomerulonephritis (8.2%), Chronic Tubulointerstitial nephritis (3.6%), Diabetes mellitus type 1 (3.9%), Hereditary or cystic diseases (3.1%). Furthermore, in the previously mentioned work, the causes of CKD were described and can be categorized into three main groups: prerenal (caused by decreased renal perfusion pressure), intrarenal (involving vascular, pathology), glomerular, or tubules-interstitium or postrenal (obstructive). [22] In the case of prerenal causes, special attention should be paid to patients with chronic heart failure with reduced ejection fraction, as in this situation, the persistently reduced blood flow to the kidneys is caused by the decreased ejection fraction of the left ventricle. This increases the risk of exacerbating CKD and kidney damage in the form of acute tubular necrosis (ATN), ultimately leading to a gradual loss of kidney function over time [23]. Regarding renal causes, nephrosclerosis is the most common vascular factor contributing to kidney disease, resulting in damage to blood vessels, glomeruli, and the interstitial area. It is noteworthy that other factors leading to chronic kidney disease (CKD) in the context of vascular pathology encompass the constriction of the renal artery during CKD therapy (e.g., with ACE inhibitors) or in cases of inadequately treated hyperlipidemia, leading to the development of atherosclerosis in the renal arteries. Consequently, over an extended period, ischemic changes culminate in the fibrosis of the renal glomeruli [18,21,23]. Glomerular kidney disease often manifests as albuminuria exceeding >3.5g per day. In such cases, it is referred to as nephrotic syndrome. When subnephrotic albuminuria is present (<3.5g/day) along with dysmorphic red blood cells, red blood cell casts, edema, and arterial hypertension, it is then termed nephritic syndrome. The most common causes include posttraumatic glomerulonephritis, bacterial endocarditis, nephropathy in systemic lupus erythematosus, and various forms of autoimmune vasculitis (e.g., GPA or IgA-associated vasculitis).[22,25]

Polycystic kidney disease (PKD) stands out as the prevailing chronic tubulointerstitial ailment. Additional etiologies encompass nephrocalcinosis, primarily attributed to heightened levels of hypercalcemia and hypercalciuria, sarcoidosis, Sjögren's syndrome, and reflux nephropathy observed in pediatric and young adult populations[24,25].

Regarding the postrenal etiology of CKD, there is mention here of obstructive nephropathy, frequently stemming from benign prostatic hyperplasia, nephrolithiasis, or a tumor located in the abdominal region, which chronically compresses the ureters [26]. Importantly, the loss of nephron function can be caused by most diseases in which the

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pathophysiology of a given disease involves a renal component as a complication. The most prevalent among the population with chronic CKD is diabetic kidney disease which was mentioned above.

It is worth noting that with the loss of nephrons, there is an occurrence of their overload, which consequently leads to hyperfiltration. The result of hyperfiltration is hypertrophy, hardening, and fibrosis of the organ's interstitium, resulting in serious complications such as: of uremic toxins. such as uric acid. -Accumulation urea. and creatinine. -Increased retention of phosphate ions and disturbances in the absorption of the active form of vitamin D3.

-Reduced erythropoietin synthesis, consequently leading to anemia.

-Pathologies related to the excretion of water and electrolytes, which consequently lead to disturbances in water-electrolyte balance and non-respiratory acidosis. It is worth mentioning that The most common causes of end-stage kidney disease, requiring dialysis therapy, include: Chronic glomerulonephritis -Hypertensive nephrosclerosis (kidney damage due to high blood pressure) -Diabetic nephropathy (kidney diabetes) damage due to -Polycystic kidney disease -Obstructive uropathy

-Autoimmune kidney diseases (e.g., IgA nephropathy) [6,9,13,25]

Symptoms

Chronic Kidney Disease is a pathology that, in its incipient stages, may manifest in an asymptomatic or oligosymptomatic manner. Frequently, arterial hypertension emerges as the initial and solitary clinical manifestation. Additionally, one of the early indicators implying compromised renal function is albuminuria. It is imperative to underscore that a patient in the advanced stage of CKD, namely ESKD, represents an individual whose symptomatic presentation may encompass virtually any organ system [6,7]. In this context, it is germane to expound upon the discrete gradations of albuminuria and the albumin-to-creatinine ratios: corresponding Stage A1 is demarcated by a classification indicative of scant or low-level albuminuria, characterized by an excretion of <30 mg of albumin in the urine over a 24-hour period, concomitant with albumin-to-creatinine of <30 ratio an mg/g.

Stage A2 is delineated as presenting with intermediate-grade albuminuria, evinced by urinary albumin excretion within the range of 30 to 300 mg over a 24-hour period and an albumin-tocreatinine ratio spanning from 30 to 300 mg/g. Stage A3 is classified as severe albuminuria, denoted by an excretion surpassing 300 mg of albumin in the urine over a 24-hour period, coupled with an albumin-to-creatinine ratio exceeding 300 mg/g. [2,6,27].

Furthermore, the clinical presentation varies depending on the stage of advancement of CKD. This is a salient consideration in daily clinical practice, as the expedited diagnosis of the underlying cause of renal function decline may prolong the interval without the necessity of dialysis therapy and, potentially, forestall its initiation altogether. Succinctly outlined below are the clinical features characterizing distinct CKD categories: G1: Typically devoid of clinical symptoms, often associated with elevated blood pressure, normative GFR, and frequently accompanied by albuminuria (A1-A2)

G2: Generally asymptomatic, commonly marked by exacerbations of the underlying disease (e.g., diabetes), impaired urine concentration leading to dehydration, deficiency of the active form of vitamin D3 resulting in hypocalcemia and, subsequently, secondary elevation of Parathyroid Hormone (PTH), diminished synthesis and secretion of erythropoietin, potentially manifesting anemia. as G3: Practically half of patients present with arterial hypertension, exacerbated incapacity for urine concentration leading to polyuria, polydipsia, increased levels of uremic toxins in the blood, and perturbations in the ionogram. G4: Incorporating all aforementioned aspects, this stage encompasses complications such as and cardiac insufficiency left ventricular hypertrophy (acute chronic). or G5: The symptoms of uremia, along with manifestations originating from other organ systems, become more pronounced in comparison to earlier stages. These systems include the cardiovascular, respiratory, endocrine, central nervous, digestive, and hematopoietic systems. [1,2,6]

Treatment

The decision to refer a patient with CKD to the care of a nephrologist is primarily contingent upon the prevailing healthcare system in a given country. Often, these decisions do not align seamlessly with the recommendations outlined by KDIGO in 2012. Works published by Moyer VA and the U.S. Preventive Services Task Force, as well as Teresa K

Chen et al. [28,29], draw attention to indicators signifying the necessity for nephrological include: supervision. These -GFR <30 mL/min/1.73m2. 25% GFR. -A decrease greater than equal to in the or -Progression of the CKD with a sustained decrease in the GRF of more than 5 ml/min per year.

-A consistent finding of significant albuminuria. -Persistent unexplained hematuria. due to a -Secondary hyperparathyroidism, persistent metabolic acidosis, anemia erythropoietin deficiency. -Hypertension resistant to treatment with four or more antihypertensive agents. of -Persistent abnormalities serum potassium. nephrolithiasis. -Recurrent extensive or

-Hereditary kidney disease or unknown cause of CKD.

Furthermore, the aforementioned works and KDIGO guidelines emphasize that therapeutic intervention primarily involves treating the underlying disease, concomitant conditions, and complications, as well as preventing their occurrence. Moreover, the utmost importance lies in impeding the progression of CKD.

Actions that can slow the progression of the disease include: -Treatment focusing on nephroprotection using medications such as SGLT2 inhibitors, ACE inhibitors, and ARBs [30,34].

-Lifestyle modifications (e.g., regular physical exercise) [31].

-Addressing complications such as anemia, disturbances in calcium-phosphate metabolism [32,33].

-Managing and treating concurrent conditions such as arterial hypertension [13,34], lipid disorders, and diabetes [34,35].

Furthermore, patients should be vaccinated against influenza, hepatitis B, and infectionscausedbyStreptococcuspneumoniae[36].

Lipid-lowering therapy

According to the consensus guidelines of the Polish Lipid Association/College of Family Physicians in Poland/Polish Cardiac Society/Polish Society of Laboratory Diagnostics/Polish Diabetes Association/Polish Society of Arterial Hypertension, patients with advanced Chronic Kidney Disease (CKD) categorized as G3-G5 according to KDIGO fall into the high or very high-risk category [37]. Depending on the patient's risk group, lipid-lowering therapy should be administered to achieve specific LDL-C and non-HDL values. For the very high-risk group, these values are: -LDL-C <55 mg/dl with a \geq 50% reduction compared to the baseline

-non-HDL <85 mg/dl

For the high-risk the values respectively group, are LDL-C <70 mg/dl >50% reduction compared baseline with а to the non-HDL < 100mg/dl Importantly, in the guidelines published in 2014 by KDIGO, the approach to lipid-lowering therapy is differentiated between patients not requiring dialysis and those requiring dialysis [38].

For the non-dialysis group, intensive lipid-lowering treatment is recommended, employing: -Statins as the first-choice medications -Statins in combination with ezetimibe if the therapeutic goal is not achieved -Statins in combination with a PCSK9 inhibitor in case of therapy ineffectiveness with the maximum tolerated dose of statin combination with ezetimibe in For dialysis patients, there is no need to initiate lipid-lowering therapy in the absence of atherosclerotic changes. However, if the decision is made to start dialysis therapy, the continuation of previously appropriate is possible. treatment

Nephroprotective

treatment

Nephroprotective treatment aims to reduce the loss of renal function by lowering arterial pressure, limiting proteinuria, and addressing modifiable and nonmodifiable risk factors mentioned earlier in this article. The fundamental principle of nephroprotective treatment is the management of arterial hypertension, with the ultimate goal of slowing the progression of CKD and mitigating the decline in glomerular filtration. Angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARB) are the most frequently employed medications, proving to be more effective in delaying CKD progression than other antihypertensive agents [39]. They lower intraglomerular pressure, inhibit the adverse effects of the renin-angiotensin-aldosterone system, and reduce proteinuria by approximately 50%. Consequently, guidelines universally recommend their use in all CKD patients (irrespective of blood pressure values) unless contraindications are identified.

According	to guidelines	published in	n various	articles	[22,29], the	treatment	objective is to
reduce	proteinuria	to	<1	g/24h,	ideall	y <0.	3 g/24h.

Treatment of	arterial	hypertension
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In patients with CKD, the first-line medications are renin-angiotensin-aldosterone system (RAAS) inhibitors, ACEI) and ARB . In cases of contraindications, nondihydropyridine calcium channel antagonists (amlodipine, nifedipine) can be employed, which may lead to a reduction in albuminuria. Post-kidney transplant, the preferred antihypertensive therapy includes non-dihydropyridine calcium channel antagonists and ARB. According to the latest guidelines [34,40], it is advisable to pursue antihypertensive therapy using a combination of two drugs, preferably in the form of a combined single pill. The recommended combination comprises either ACEI or ARB and a thiazide or thiazide-like diuretic. It is noteworthy that if GFR is <30 ml/min/1.73 m², thiazide diuretics should not be used as they lack efficacy. In this situation, they should be replaced with loop diuretics (furosemide). In recent years, some SGLT2 inhibitors (dapagliflozin), particularly in patients with diabetic kidney disease, have gained popularity in both antihypertensive and nephroprotective therapy [14,41]. According to the 2021 KDIGO guidelines [40], the target blood pressure values for CKD patients, regardless of albuminuria levels, are SBP <120 mm Hg. For kidney transplant recipients, the target is <130/80 mm Hg, while for those undergoing dialysis, it is recommended to maintain blood pressure below <140/90 mm Hg.

Treatment	of	anemia	in	patients	with	CKD
				1		

According to guidelines [32,34], a crucial aspect of anemia treatment in patients with CKD is the correction of iron deficiency to maintain ferritin levels within the range of 100 to 800 µg/l and the transferrin iron saturation index (TSAT) at 20-50%For every anemic patient with ferritin levels ≤ 500 µg/l and TSAT $\leq 30\%$, including those receiving erythropoiesis-stimulating agents (e.g., epoetin α). It is important to note that the target hemoglobin concentration in individuals with Chronic Kidney Disease (CKD) is >9 g/dl, ideally maintained between 10-11.25 g/dl. Additionally, efforts should be made to avoid exceeding values above 13 mg/dl, as this is associated with an increased risk of cardiovascular complications. Oral iron preparations such as iron sulfate may be employed as the first-line

treatmentinnon-dialysispatients.Erythropoiesis-stimulating agents are reserved for cases where, following iron deficiency
correction and exclusion of other causes of anemia, hemoglobin levels remain below 10 g/dl.Regarding the transfusion of red blood cell concentrates, it is only recommended for severe
anemia accompanied by symptoms if there is a lack of response or resistance to treatment
withwiththeaforementionedmedications.

Treatment of mineral and bone disorder

According to the 2017 KDIGO guidelines [33], monitoring serum calcium and phosphate levels, parathyroid hormone concentrations, and alkaline phosphatase levels should commence as early as stage G3. The cornerstone of treatment involves correcting vitamin D deficiency through supplementation with its active derivatives, such as alfacalcidol or calcitriol. Caution must be exercised to avoid aggressive treatment due to the potential risk of developing adynamic bone disease. Daily calcium intake should be limited to 2g per day, and dietary phosphate intake should be restricted.Regarding parathyroid hormone (PTH), its concentration should be maintained within 2 to 9 times the upper limit of the reference range. If these values are exceeded, treatment involves the use of the aforementioned active forms of D. combination of vitamin cinacalcet. or a these substances. The most serious of mineral and bone disorders is renal osteodystrophy, stemming from a combination of vitamin D deficiency and hyperphosphatemia. Consequently, its treatment is based addressing these factors. on

Renal replacement therapy (RRT)

Currently, there are three modalities of renal replacement therapy: kidney transplantation, hemodialysis (HD), and peritoneal dialysis (PD).According to the report on the state of renal replacement therapy in Poland in 2021 by Alicja Dębka-Ślizień et al. [42], 144 per 1 million inhabitants initiated treatment using HD or PD in the country. Liyanage T. et al.'s analysis [43] revealed that nearly 2.6 million people received RRT in 2010, with almost 2.3 million potentially facing premature death due to a lack of access to RRT. This highlights that RRT remains an imperfect method due to the high costs of treatment and insufficiently effective preventive strategies for CKD and ESKD. Both HD and PD meet all the assumptions outlined in various guidelines for CKD treatment. Classical hemodialysis

typically lasts for 4-5 hours and is performed three times a week, every other day. The frequency and intensity of HD are adjusted by the attending physician in the dialysis center to achieve a urea reduction ratio (URR) > 65%. HD is an extracorporeal blood purification method that replaces the excretory function of the kidneys, maintains the electrolyte balance of body fluids, corrects bicarbonate deficits, and eliminates accumulated toxins, including uremic toxins. The process occurs through a semi-permeable membrane of the dialyzer, where substances from the patient's blood move into the dialysate and vice versa in a diffusion mechanism following the concentration gradient – a key physical phenomenon enabling HD. Ultrafiltration allows for the removal of excess water. The creation of a vascular access is necessary for HD. According to the 2012 KDIGO guidelines [6], the preferred method is an arteriovenous fistula using the patient's own vessels, created by connecting an artery to a vein. The preferred site for creating the fistula is the vessels of the non-dominant forearm. To consider the fistula suitable for use, the flow through it should range from 500-1000 ml/min, and the surgical site should be healed, with arterialization of the vein, indicating widening and thickening of the vein lumen. Less preferred types of vascular access include arteriovenous fistulas from synthetic vessels, temporary or permanent dialysis catheters. There are no rigidly defined biochemical parameter values determining the initiation of HD for a patient; the clinical picture decides, but according to the KDIGO 2021 guidelines [44], initiating RRT is indicated when eGFR is <15 ml/min/1.73m² and when subjective and objective symptoms of uremia are present. Peritoneal dialysis, on the other hand, is a method in which the peritoneum serves as the equivalent of the semi-permeable membrane in the hemodialysis apparatus. Uremic toxins pass into the introduced peritoneal dialysis fluid with a volume of 2 to 2.5 liters via a catheter. The exchange of toxins and electrolytes occurs through the process of diffusion along the concentration gradient, and water removal through osmotic ultrafiltration. This method is preferred in cases where vascular access cannot be established or in the presence of hemodynamic instability during hemodialysis. Similarly to hemodialysis, there are no strictly defined biochemical parameters indicating the decision to initiate dialysis, and the decision is based on the clinical presentation. The final method of RRT is kidney transplantation (KTx), which, according to scientific literature [6,22,44], should be planned when the GFR drops to <15 ml/min/1.73m². The kidney can be obtained from a deceased or living donor. Conditions that must be met for KTx to be performed include, others: among -Blood group compatibility in the ABO system between the donor and the recipient

-Blood group compatibility in the ABO system between the donor and the recipient Negative result of a cross-match test conducted immediately before transplantation, i.e., the absence of recipient's anti-HLA antibodies directed against the donor's MHC system antigens. -The kidney transplantation process is highly complex and will not be addressed in this paper. Unfortunately, KTx is associated with numerous complications and requires the use of immunosuppressive therapy.

Conclusions

In summary, CKD is a complex phenomenon that requires significant commitment from clinicians and often necessitates interdisciplinary collaboration to ensure the proper treatment of the patient. Many methods developed in recent years have significantly improved the quality of life for patients with CKD and ESKD, as well as prolonged their lives. There is hope that the coming years will bring new discoveries in the field of nephrology, enabling the potential optimization of treatment for those with CKD and ESKD. In 2023, a reissue of the KDIGO guidelines regarding the treatment and management of patients with chronic kidney disease is planned. It is anticipated that the new standards will bring about many improvements in current treatment methods.

Author's contribution

Conceptualization, Michał Żuberek and Daniel Ślusarczyk; methodology, Michał Żuberek and Wiktoria Jakubowska ; software, Piotr Pisera and Aleksandra Kiełkowicz; check, Filip Pactwa and Aleksandra Kiełkowicz; formal analysis, Zuzanna Popińska and Daniel Ślusarczyk; investigation, Bartłomiej Żmuda and Wiktoria Jakubowska; resources, Michał Żuberek and Piotr Pisiera; data curation, Bartłomiej Żmuda and Zuzanna Popińska; writing rough preparation, Michał Żuberek; writing - review and editing, Michał Żuberek; visualization, Daniel Ślusarczyk and Filip Pactwa; supervision, Piotr Pisera and Filip Pactwa; project administration, Michał Żuberek All authors have read and agreed with the published version of the manuscript.

Funding Statement

The study did not receive special funding.

Institutional Review Board Statement

Not applicable

Informed

Consent

Statment

Not applicable

Data Availability Statement

The data presented in this study is available upon request from the correspondent author.

Acknowledgments

Not applicable

Conflict of Interest Statement

The authors report no conflict of interest.

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