REVIEW ARTICLE

Eurasian Journal of Medical and Biological Sciences
AN INTERNATIONAL JOURNAL
Eurasian J Med Biol Sci **4(1): 1-8**

Relationship between Kidney Diseases and Nutrition

Aleyna Şahin[1*], Ayşe Güneş Bayır[2]

[1] *Nobel Pharmaceutical Company, İstanbul, Türkiye*
[2] *Department of Nutrition and Dietetics, Faculty of Health Sciences, Bezmialem Vakif University, İstanbul, Türkiye*

Received : 01/10/2023 Revised : 25/03/2024 Accepted : 22/05/2024

ABSTRACT: The kidney is one of the most complex organs in the body physiologically, structurally and metabolically. There are different diseases that we can count as kidney disease. However, chronic kidney disease is the most important. Chronic kidney disease (CKD) is defined as a progressive and irreversible loss of kidney function in which the estimated glomerular filtration rate (GFR) decreases. CKD has long been a worldwide public health concern and poses a heavy health care and economic burden as there is information that a reduced GFR increases the risk of cardiovascular problems, hospitalization, cognitive dysfunction and overall death. Individuals with chronic kidney disease (CKD), especially those on dialysis, are prone to protein-energy wastage and this condition can be improved with different methods of nutritional support. Dietitian-guided dietary counseling is key to preventing and managing protein-energy wasting (PEW) in CKD. The major concerns associated with low-protein diets in clinical practice are the risk of PEW and adherence to dietary restrictions. Many studies evaluating low-protein diets in CKD patients have demonstrated acceptable safety, with no serious complications and low rates of PEW and malnutrition. High sodium intake has detrimental effects on blood pressure, cardiovascular health, kidney function and CKD progression. Dietary sodium restriction is strongly recommended in patients with CKD to control fluid retention, lower blood pressure, and reduce cardiovascular risk. In this review, it has been shown that nutritional interventions specific to kidney patients increase their quality of life and the positive effects of the treatment process. This review focuses on the relationship between adequate nutritional interventions and kidney diseases, and how kidney patients in treatment should be fed.

Keywords: Kidney, kidney diseases, diet, chronic kidney disease, proteins.

INTRODUCTION

Kidneys are very complicated organs and are of vital importance for the continuation of routine bodily activities. The survival of a human body is largely dependent on certain functions and events performed by the kidneys. With the normal functioning of our kidneys, keeping body fluids in a certain balance and healthy functioning of other organ systems are ensured (Wallace, 1998). Our kidneys are responsible for regulating the amount of fluid in the body. This task is carried out in the form of continuous filtration of the blood from the glomeruli to the renal tubules, followed by the exchange of water and solutes between the tubular and peritubular capillaries, which ensures the excretion of excess water, solutes and waste products in the urine. These events are regulated by the mechanisms of the kidneys, hormones and renal nerves (Osborn et al., 2021).

Human kidneys are two reddish bean-shaped organs located under the rib cage on either side of the spine (Wallace, 1998). Kidneys are the main organs in the human excretory system, involved in the filtration of blood before urine is formed. The functional unit of the kidney is the nephron, and there are millions of nephrons in the kidneys that play an important role in the filtration and purification of blood. The nephron is divided into two parts, the glomerulus and the renal tubule. Renal sympathetic (efferent) nerves play an important role in regulating kidney function, including glomerular filtration, sodium reabsorption, and renin release. The kidney is also innervated by sensory (afferent) nerves that transmit information to the brain to modulate sympathetic outflow. A better understanding of how renal nerves control the kidney under physiological and pathophysiological conditions is needed (Osborn et al., 2021).

Kidney diseases are one of the most common health problems, and the prevalence of these diseases may increase in the coming years, especially due to the increasing number of older patients with comorbidities (Zakrocka and Załuska, 2022). First, some important kidney diseases will be briefly mentioned in this review, and the investigation of the relationship between adequate nutritional interventions and kidney diseases and how to feed patients with kidney diseases were aimed using current sources.

GLOMERULONEPHRITIS

Glomerulonephritis is a pathology that includes kidney diseases characterized by immune-mediated damage to the basement membrane, mesangium or capillary endothelium, resulting in hematuria, proteinuria and azotemia (Kazi et al., 2023). Acute glomerulonephritis (AGN) is a disease representing an acute nephrotic syndrome, which is usually characterized by the sudden

* aleynaasaahin@gmail.com

onset of edema, hematuria, proteinuria, and hypertension, and varying degrees of decreased renal function (Yoshizawa, 2000). The acute glomerulonephritis with full-blown nephritic syndrome or rapidly progressing glomerulonephritis is rare (Sethi et al., 2022). It is mostly limited to patients with vasculitis associated with anti-neutrophil cytoplasmic antibodies and anti-glomerular basement membrane disease. Patients also present with asymptomatic hematuria and proteinuria, with or without decreased renal function. Acute glomerulonephritis may be caused by primary from kidney or secondary from a disease causing kidney symptoms (Kazi et al., 2023). For example; an acute post-streptococcal glomerulonephritis is a secondary AGN as a streptococcal infection in contrast to Staphylococcus aureus infection can also lead to glomerulonephritis. Glomerulonephritis accounts for 25% to 30% of all end-stage kidney disease cases; that is, approximately one quarter of patients with nephritic syndrome. In most cases, progression is relatively rapid, and end-stage kidney disease may occur within weeks or months after the onset of acute nephritic syndrome. Nephrotic syndrome of glomerulonephritis progresses with a clinical picture consisting of severe proteinuria (>3.5 g/day) and edema. Known chronic glomerulonephritis (CGN), also called chronic nephritis, is a primary glomerulopathy condition caused by multiple pathogenic factors such as extreme fatigue, a bacterial or viral infection, abnormal stress reaction, water and electrolyte disturbance, drug toxicity (Zhao et al., 2019). The primary goal is to prevent CGN from leading to end-stage renal disease by slowing its progression by identifying and controlling risk factors at an early stage

Nephrotic Syndrome (NS)

Nephrotic syndrome (NS) emerges as a condition characterized by edema, hypoalbuminemia, hyperlipidemia, and proteinuria at the same time it is a cause of end-stage renal disease (Wang and Greenbaum, 2019). Although nephrotic syndrome is not a disease, it is the first sign of disease that can damage the glomeruli. It may occur as a secondary condition in autoimmune diseases such as lupus. NS is the name given to the totality of kidney-related findings in the child's body. NS includes proteinuria, excessive loss of protein in the urine with many secondary changes in coagulation status, lipid and fluid balance in the body (Dumas De La Roque et al., 2018). It has various etiologies and can affect individuals from all age groups, from congenital causes in childhood to later adulthood Diabetic nephropathy, focal segmental glomerulosclerosis, and membranous nephropathy including an underlying diseases are the most common causes in adults (Politano et al., 2020). In case of NS, a damage or a dysfunction of components of the glomerulus, including the basement membrane, endothelial surface, or epithelial cells, leads to loss of protein in the urine. Only certain proteins (predominantly albumin) can be lost through the urine due to the limitations of pore size in the basement membrane and the burdens of the associated barriers.

Acute Kidney Failure (ARF)

Acute kidney injury or acute kidney failure is a syndrome characterized by a sudden, sustained and potentially reversible decrease in glomerular filtration rate and tubular function, affecting kidney function globally. It consists of a series of events that begin with the presence of risk factors and progress to progression of acute kidney injury characterized by stress, injury, and renal failure, resulting in death or use of renal replacement therapy (Abarca Rozas et al., 2020). Acute renal failure, also known as acute kidney disease, is a syndrome that manifests itself with the rapid loss of the excretory function of the kidney. It is typically diagnosed by accumulation of urea and creatinine, the end products of nitrogen metabolism, or a decrease in urine output, or both. It is the clinical manifestation of different disorders affecting the kidney acutely. Acute renal failure is very common in hospitalized patients and is very common in critically ill patients (Bellomo et al., 2012).

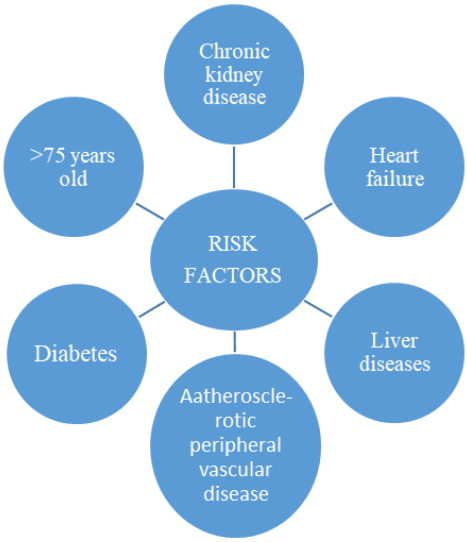

Figure 1. *Optimal risk factors for the development of acute kidney disease (ACD)* (Dumas De La Roque et al., 2018)

Chronic Kidney Failure

Chronic kidney failure or chronic kidney disease (CKD) is a condition of irreversible deterioration of kidney function that gradually progresses to end-stage kidney disease. Symptoms of chronic renal failure are usually seen when the GFR is between 10-25% of normal (Jones, 2012). Kidney failure is a disorder characterized by loss of function and resulting in an increased amount of metabolites accumulating in the blood. As a result, the fluid and electrolyte balance in the body is disrupted, leading to serious health problems. The gradual loss of kidney function over a period of several years is called chronic kidney disease or chronic kidney failure (Moudgil and Bagga, 1999). Management of chronic renal failure aims to delay the progression of kidney damage and to treat complications related to renal dysfunction. Recommended recommendations to delay progression include protein restriction, control of hypertension, use of angiotensin converting enzyme inhibitors, and control of hyperlipidemia. Proper protein and calorie intake is very important to prevent growth retardation (Jones, 2012). Nutritional supplements may often be required.

Kidney Stones

Kidney stone disease, also known as nephrolithiasis or urolithiasis, is one of the oldest diseases in the history of medicine (Wang et al., 2021). Urolithiasis is a lifelong disease that often recurs. If appropriate treatment is not applied, kidney stones can cause obstruction of the ureter, blood in the urine, frequent urinary tract infections, vomiting or painful urination. Some studies suggest that many environmental factors, including changes in lifestyle and diet, as well as global warming, contribute to an increase in kidney stone formation (Peerapen and Thongboonkerd, 2023). It is a global health problem that affects people in almost all developed and developing countries. Its prevalence is constantly increasing with a high recurrence rate after kidney stone removal. Obesity, diabetes, hypertension and metabolic syndrome are some risk factors for stone formation (Khan et al., 2016). Kidney stones are mineral deposits found free or attached to the renal papilla in the renal calyces and pelvis. They contain crystalline and organic components and are formed when urine becomes supersaturated with a mineral. Calcium oxalate is the main component of most stones, and most of them form on a calcium phosphate basis called Randall's plaques located on the renal papillary surface.

Type of kidney stones are calcium, struvite, uric acid, and cystine (Peerapen and Thongboonkerd, 2023). Kidney stones are generally classified according to their primary crystal composition Each of these stone types has similar and unique risk factors. Stone formation is quite common and increasing. The metabolic output and other contributions of the microbiome, including urease-producing bacteria, nanobacteria, and intestinal microbiota, are likely to contribute to kidney stone formation (Wang et al., 2021). The recurrence rate can be up to 50% within the first 5 years after the first stone formation attack (Khan et al., 2016). Although there are effective methods for treating the disease, preventive strategies for both recurrent stones are needed to reduce the physical and financial burden of this disease (Peerapen and Thongboonkerd, 2023). To prevent the occurrence of this disease and prevent kidney stone formation, it is important to identify its etiology and risk factors.

KIDNEY DISEASES AND NUTRITION

Diet is the basis for almost every aspect of care for patients with chronic kidney disease. Dietary interventions in the early stages of CKD can slow the progression of CKD and the onset of symptoms. In later stages, it may delay or eliminate the need for kidney replacement therapy. Medical nutrition therapy also plays important roles in preventing and treating protein-energy malnutrition, electrolyte imbalances, bone and mineral abnormalities (Kistler et al., 2021). CKD patients are at risk of many nutritional disorders such as malnutrition, PEW and electrolyte disturbances. Obesity also faces other challenges, such as secondary prevention of cardiovascular disease and maintaining a high-quality diet within reduced glomerular filtration shortenings. Malnutrition includes protein-energy malnutrition and micronutrient deficiencies. Protein-energy malnutrition caused by inadequate protein and energy intake results in muscle and fat loss; if severe enough, it can lead to increased frailty, susceptibility to disease, and even premature death (MacLaughlin et al., 2022). Starting from the early stages of chronic kidney disease, keeping the dietary energy, protein, sodium and phosphorus at normal levels has a very important place for the protection of the kidney. In the later stages of chronic kidney disease, protein-restricted diets can prevent or improve uremic symptoms or complications such as metabolic acidosis, mineral and bone disorders, insulin resistance, proteinuria, hypertension, and fluid retention (Cupisti et al., 2014). The main nutritional goals for people with chronic kidney disease are to slow the progression of kidney failure and minimize the uremic toxicity and metabolic disturbances of kidney failure, reduce proteinuria, maintain a healthy diet, and reduce the risk of secondary complications. In addition, nutritional requirements vary among patients at various stages of renal function and among patients with various comorbidities. The role of overnutrition is still unresolved. According to studies, obesity is a risk factor for the development of kidney disease (Gluba-Brzózka et al., 2017).

Many factors that increase the risk of malnutrition and PEW in CKD interact. These include a range of metabolic and endocrine abnormalities that develop due to impaired kidney function. For example, decreased erythropoietin, vitamin D, carnitine, testosterone, and thyroid hormone. Insulin resistance is quite common, leading to impaired suppression of muscle

protein breakdown; such as growth hormone resistance, which leads to a decrease in anabolic potential. There are also disturbances in adipocytokines such as leptin and ghrelin that may affect the hypothalamic regulation of appetite. This appetite disturbance in patients has also been associated with higher mortality. In particular, inflammation plays an important role in the development of PEW syndrome. Other factors believed to affect the risk of developing malnutrition in CKD include olfactory system and taste perversion. Polypharmacy was also found to be an important risk factor. Dialysis is also known to have a catabolic effect by increasing amino acid (AA) losses and has been associated with a decrease in appetite in patients on dialysis days (Slee and Reid, 2022).

Metabolic changes that occur with decreased kidney function often lead to changes in appetite and changes in food intake. As time progresses, this causes the loss of nutrient reserves of muscle and adipose tissue, which act as the body's stores. Uremia and associated infection, changing hormone levels, metabolic acidosis, and changes in bowel movements may lead to decreased dietary intake as chronic kidney disease progresses. Taste disorders, loss of appetite, reduced or restricted food intake, combined with edema can cause loss of fat and lean tissue. Therefore, assessing body weight alone may be insufficient to detect changes in nutrient stores (MacLaughlin et al., 2022). The approach to nutrition in patients with chronic kidney disease has been updated in recent years to move from very restrictive diets to a more sensitive diet, to ensure the correct intake of macro and micronutrients that improve the patient's nutritional status. Controlling dietary intake of adequate energy-providing protein, phosphorus, sodium and potassium remains a vigilant priority. A way is being followed to try to modulate the gut microbiota by creating the diet to include more plant foods. Increasing plant foods in the diet in this way can affect not only the development of the disease but also its prevention. It can help lower blood pressure, improve glycemic control in diabetic patients, and reduce obesity (Cigarrán Guldris et al., 2022).

Dietary modification is recommended in the treatment of chronic kidney disease (CKD). Individuals with CKD often have multiple comorbidities, such as high blood pressure, diabetes, obesity, and cardiovascular disease, for which dietary modification is also recommended. As CKD progresses, nutrition plays an important role in reducing the risk of cardiovascular disease and decline in kidney function. The goals of nutritional interventions in CKD include managing risk factors, maintaining optimal nutritional status at all stages of CKD, preventing the accumulation of toxic metabolic products, and avoiding CKD complications. Recommended dietary changes should be feasible, sustainable, and appropriate to patients' food preferences and clinical needs (Anderson et al., 2016). As chronic kidney disease progresses, the requirements and importance of use of different food groups change. These changes are accompanied by multiple nutritional and metabolic abnormalities observed during the continuous course of kidney disease. In order to provide the best care for individuals with chronic kidney disease, it is very important to understand the applicable nutritional principles and nutritional status, to identify patient-specific dietary needs, and to understand appropriate methods for preventing or treating potential or existing nutrient deficiencies and disorders. Micronutrient deficiencies may occur as a result of inadequate dieting, reduced absorption, dietary prescriptions that restrict foods with high micronutrient content, and dialysis procedures in patients with chronic kidney disease (MacLaughlin et al., 2022). Dietary interventions are very important for dialysis patients to maintain lean mass and provide adequate energy. For CKD between stage 3 and stage 5, 0.6-0.8 g/kg/day protein intake and 30-35 kcal/kg/day calorie intake are recommended. Despite the risk of negative protein balance, a low protein diet (LPD) is advocated in CKD given the risk of progression of CKD with a higher protein diet. Protein restriction can be achieved with an LPD in which the majority of energy is provided by fat and carbohydrates. LPD has a greater protective effect in younger patients than in older patients (Hanna et al., 2020).

There is some concern that multiple targets for protein intake for CKD patients may lead to confusion and create difficulties for implementation. Renal hyperfiltration caused by high dietary protein intake and the resulting protein waste products are thought to contribute to the decrease in kidney function (Kistler et al., 2021). There is evidence that protein-restricted diets may delay the need for dialysis. However, it is known that its effect on slowing down the rate of GFR decline is not that obvious. The severity of protein restriction depends on the level of residual renal function. In conclusion, a very low protein, very low phosphorus diet supplemented with essential amino acids and keto-acids has been the preferred choice for improving metabolic and nutritional parameters in the pre-dialysis stages. However, nutritional therapy is not just a matter of dietary protein intake, but also includes phosphorus and sodium restriction and adequate energy intake. This is a very important point, as maintaining a good nutritional status is a prerequisite and goal of nutritional therapy that provides patients with a good quality of life and physical performance. To this end, the energy supply must equal or even exceed the energy demand (Cupisti et al., 2014). Recommendations for protein intake in patients with end-stage renal disease indicate suspicions of worsening protein energy wastage and the need to prevent sarcopenia and cachexia. The recommended protein consumption in ESRD is 1.2 g/kg/day protein intake. This is because the amount of production is no longer an issue and can be removed with effective dialysis, but decreased muscle mass is a concern that can be considered acute (Hanna et al., 2020). For a patient with CKD who is not on dialysis, dietary changes are known to be an effective intervention to prevent the progression of CKD, although it is quite cost-effective. It should be emphasized that a carefully crafted diet plan is when CKD is first diagnosed, known as the period when interventions are most likely to be effective (Kramer, 2019).

Protein-energy malnutrition is a common picture in patients with chronic kidney disease. In addition, "protein-energy malnutrition", which is defined as abnormally low levels of body protein mass and energy reserves, is commonly encountered in this patient group, especially in patients undergoing dialysis. In addition to inappropriate food intake, it may be the result of increased catabolism due to protein energy malnutrition, oxidative stress, systemic inflammation, abnormal glucose and insulin

homeostasis, metabolic acidosis, failure to maintain anabolism/catabolism balance and vitamin D deficiency. Malnutrition and protein-energy malnutrition have been reported to be strongly associated with mortality in chronic kidney disease patients (Rysz et al., 2017).

High sodium intake has detrimental effects on blood pressure, cardiovascular health, kidney function and CKD progression. Restriction of sodium in the diet has an important place and is recommended in order to control fluid retention, reduce blood pressure and protect cardiovascular health in patients with CKD. Studies show that sodium restriction leads to reductions in blood pressure, extracellular fluid volume, and proteinuria in patients with moderate CKD. In addition, sodium restriction increased the effect of low protein diet by reducing intraglomerular pressure and had an effect on the regulation of the RAS system. It has been stated that sodium restriction can reduce proteinuria and slow down the progression of CKD. Almost all guidelines recommend reducing sodium intake to 2 to 2.3 g/day in adults. Given the risk of hyponatremia and adverse outcomes, dietary sodium intake < 1.5 g/day is not recommended for patients on dialysis as well as patients with advanced CKD (Kim and Jung, 2020).

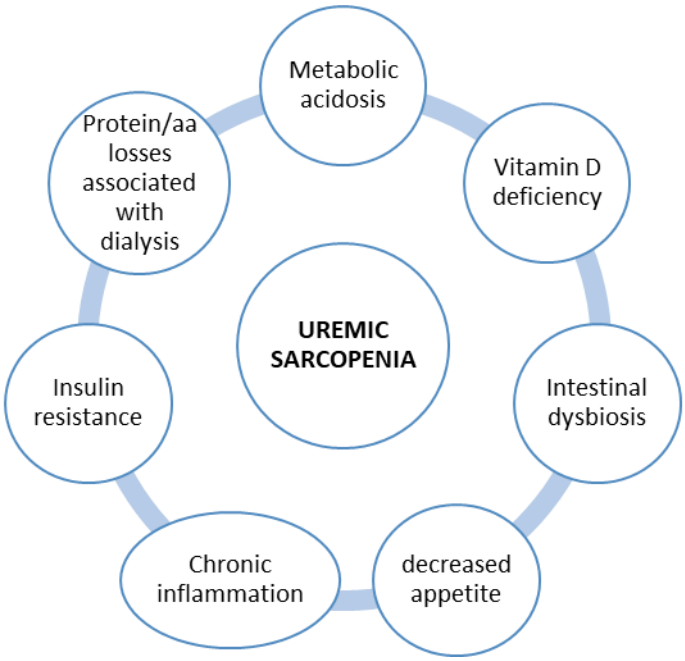

Figure 2. *Factors that may cause uremic sarcopenia* (Noce et al., 2021). *aa: amino acids*

Chronic kidney disease and decreased glomerular filtration rate are risk factors for the development of chronic metabolic acidosis. Preventing or correcting chronic metabolic acidosis has been found to slow the progression of chronic kidney disease. Dietary composition can strongly affect the acid-base balance. The main determinants of net endogenous acid production are the production of large amounts of hydrogen ions, mostly by animal-derived protein, which is balanced by the metabolism of base-producing foods such as fruits and vegetables. Alkaline treatment of chronic metabolic acidosis can be achieved with an alkali-rich diet or oral administration of alkaline salts. The primary goal of dietary therapy should be to increase the proportion of fruits and vegetables and to reduce the daily protein intake to 0.8-1.0 g per kg body weight. It is known that diet is the most important individual factor affecting acid-base status. While protein-rich foods such as meat, meat products, fish and cheese are the food groups with the highest acid load, fruits, vegetables, salads and juices have high alkalizing potential (Siener et al., 2018).

CONCLUSION

Kidneys are very complicated organs and are of vital importance for the continuation of routine bodily activities. The survival of a human body is largely dependent on certain functions and events performed by the kidneys. With the normal functioning of our kidneys, keeping body fluids in a certain balance and healthy functioning of other organ systems are ensured.

There are many details that need to be taken into consideration regarding nutrition in kidney diseases. It is an important part of the treatment that patients comply with these nutritional recommendations. Medical treatment for kidney diseases must be supported by nutrition.

The kidneys keep the water and salt balance constant by adjusting the excretion of water and minerals according to eating and drinking habits. If not enough sodium is excreted from the body and the sodium in the body increases, this causes water retention in the tissues. Too much potassium can lead to cardiac arrhythmias. In chronic kidney disease, attention should be paid to a balanced diet. Daily protein intake should be regulated. Foods with high potassium, sodium and phosphate content should

be avoided. Vegetable proteins are more advantageous than animal proteins. Vegetables also alkalize the body. Vegetables that do not have high potassium content should be preferred. Processed and ready-made foods should not be consumed due to their high sodium, phosphate and potassium content. Foods rich in potassium should be reduced. Especially bananas, legumes, nuts, dried fruits, vegetable and fruit juices, and tomatoes are very rich in potassium. Due to impaired kidney function, table salt (sodium chloride) should be reduced, as urinary sodium excretion decreases. Foods and drinks with low salt content should be preferred.

Acute glomerulonephritis (AGN) is a disease representing an acute nephrotic syndrome, which is usually characterized by the sudden onset of edema, hematuria, proteinuria, and hypertension. Chronic glomerulonephritis (CGN), also known as chronic nephritis, is a primary condition of glomerulopathy caused by multiple pathogenic factors such as extreme fatigue, a bacterial or viral infection, abnormal stress reaction, water and electrolyte disturbance, drug toxicity. Nephrotic syndrome (NS) is a condition characterized by hypoalbuminemia, hyperlipidemia and proteinuria. Nephrotic syndrome (NS) may occur as a secondary condition in autoimmune diseases such as lupus. Nephrotic syndrome (NS) includes proteinuria, excessive loss of protein in the urine with many secondary changes in coagulation status, lipid and fluid balance in the body. Acute kidney injury is a syndrome characterized by a sudden, sustained and potentially reversible decrease in glomerular filtration rate and tubular function, affecting kidney function globally. It consists of a series of events that begin with the presence of risk factors and progress to progression of acute kidney injury characterized by stress, injury, and kidney failure, resulting in death or use of renal replacement therapy. Chronic kidney failure (CKD) or chronic kidney disease (CKD) is a condition of irreversible deterioration of kidney function that gradually progresses to end-stage kidney disease. Major causes of chronic renal failure include obstructive uropathy, primary glomerular diseases, reflux neuropathy, and hypoplastic or dysplastic kidneys.

Diet is the basis for almost every aspect of care for patients with chronic kidney disease. Dietary interventions in the early stages of CKD can slow the progression of CKD and the onset of symptoms. In later stages, it may delay or eliminate the need for kidney replacement therapy. Medical nutrition therapy also plays important roles in preventing and treating protein-energy malnutrition, electrolyte imbalances, bone and mineral abnormalities in CKD patient.

ACKNOWLEDGEMENTS

None

CONFLICT OF INTEREST

The authors have not declared any conflict of interest

REFERENCES

Abarca Rozas, B., Mestas Rodríguez, M., Widerström Isea, J., Lobos Pareja, B., Vargas Urra, J. (2020). A current view on the early diagnosis and treatment of acute kidney failure. Un infoque actual para el diagnóstico precoz y tratamiento de la insuficiencia renal aguda. Medwave, 20 (5), e7928. https://doi.org/10.5867/medwave.2020.05.7928

Anderson, C.A., Nguyen, H.A., Rifkin, D.E. (2016). Nutrition Interventions in Chronic Kidney Disease. The Medical clinics of North America, 100(6), 1265–1283. https://doi.org/10.1016/j.mcna.2016.06.008

Bellomo, R., Kellum, J.A., Ronco, C. (2012). Acute kidney injury. Lancet (London, England), 380 (9843), 756–766. https://doi.org/10.1016/S0140-6736(11)61454-2

Cigarrán Guldris, S., Latorre Catalá, J.A., Sanjurjo Amado, A., Menéndez Granados, N., Piñeiro Varela, E. (2022). Fiber Intake in Chronic Kidney Disease: What Fiber Should We Recommend?. Nutrients, 14(20), 4419. https://doi.org/10.3390/nu14204419

Cupisti, A., D'Alessandro, C., Fumagalli, G., Vigo, V., Meola, M., Cianchi, C., Egidi, M.F. (2014). Nutrition and physical activity in CKD patients. Kidney & blood pressure research, 39(2-3),107–113. https://doi.org/10.1159/000355784

Dumas De La Roque, C., Prezelin-Reydit, M., Vermorel, A., Lepreux, S., Deminière, C., Combe, C., Rigothier, C. (2018). Idiopathic Nephrotic Syndrome: Characteristics and Identification of Prognostic Factors. Journal of clinical medicine, 7 (9), 265. https://doi.org/10.3390/jcm7090265

Gluba-Brzózka, A., Franczyk, B., Rysz, J. (2017). Vegetarian Diet in Chronic Kidney Disease-A Friend or Foe. Nutrients, 9(4), 374. https://doi.org/10.3390/nu9040374

Hanna, R.M., Ghobry, L., Wassef, O., Rhee, C.M., Kalantar-Zadeh, K. (2020). A Practical Approach to Nutrition, Protein-Energy Wasting, Sarcopenia, and Cachexia in Patients with Chronic Kidney Disease. Blood purification , 49 (1-2), 202-211. https://doi.org/10.1159/000504240

Jones M. (2012). Recognizing acute kidney injury. Clinical medicine (London, England), 12(3), 287–289. https://doi.org/10.7861/clinmedicine.12-3-287

Kazi, A.M., Hashmi, M.F. Glomerulonephritis. 2023 Jun 26. In: StatPearls, Treasure Island (FL): StatPearls Publishing; 2024. PMID: 32809479.

Khan, S.R., Pearle, M.S., Robertson, W.G., Gambaro, G., Canales, B.K., Doizi, S., Traxer, O., Tiselius, H.G. (2016). Kidney stones. nature reviews. Disease primers, 2 , 16008. https://doi.org/10.1038/nrdp.2016.8

Kim, S.M., Jung, J.Y. (2020). Nutritional management in patients with chronic kidney disease. The Korean journal of internal medicine, 35(6), 1279–1290. https://doi.org/10.3904/kjim.2020.408

Kistler, B.M., Moore, L.W., Benner, D., Biruete, A., Boaz, M., Brunori, G., Chen, J., Drechsler, C., Guebre-Egziabher, F., Hensley, M.K., Iseki, K., Kovesdy, C.P., Kuhlmann, M.K., Saxena, A., Wee, P.T., Brown-Tortorici, A., Garibotto, G., Price, S.R., Yee-Moon Wang, A., Kalantar -Zadeh, K. (2021). The International Society of Renal Nutrition and Metabolism Commentary on the National Kidney Foundation and Academy of Nutrition and Dietetics KDOQI Clinical Practice Guideline for Nutrition in Chronic Kidney Disease. Journal of renal nutrition : the official journal of the Council on Renal Nutrition of the National Kidney Foundation, 31(2), 116–120.e1. https://doi.org/10.1053/j.jrn.2020.05.002

Kramer, H. (2019). Diet and Chronic Kidney Disease. Advances in nutrition (Bethesda, Md.) , 10 (Suppl_4), 367–379. https://doi.org/10.1093/advances/nmz011

MacLaughlin, H.L., Friedman, A.N., Gemini, T.A. (2022). Nutrition in Kidney Disease: Core Curriculum 2022. American journal of kidney diseases : the official journal of the National Kidney Foundation, 79(3), 437–449. https://doi.org/10.1053/j.ajkd.2021.05.024

Moudgil, A., Bagga, A. (1999). Evaluation and treatment of chronic renal failure. Indian journal of pediatrics , 66(2), 241–253. https://doi.org/10.1007/BF02761215

Noce, A., Marrone, G., Ottaviani, E., Guerriero, C., Di Daniele, F., Pietroboni Zaitseva, A., Di Daniele, N. (2021). Uremic Sarcopenia and Its Possible Nutritional Approach. Nutrients, 13(1), 147. https://doi.org/10.3390/nu13010147

Osborn, J.W., Tyshynsky, R., Vulchanova, L. (2021). Function of Renal Nerves in Kidney Physiology and Pathophysiology. Annual review of physiology, 83 , 429–450. https://doi.org/10.1146/annurev-physiol-031620-091656

Peerapen, P., Thongboonkerd, V. (2023). Kidney Stone Prevention. Advances in Nutrition, 14(3), 555-569. doi: 10.1016/j.advnut.2023.03.002.

Politano, S.A., Colbert, G.B., Hamiduzzaman, N. (2020). Nephrotic Syndrome. Prim Care, 47(4), 597-613. doi: 10.1016/j.pop.2020.08.002.

Rysz, J., Franczyk, B., Ciałkowska-Rysz, A., Gluba-Brzózka, A. (2017). The Effect of Diet on the Survival of Patients with Chronic Kidney Disease. Nutrients, 9(5), 495. https://doi.org/10.3390/nu9050495

Sethi, S., De Vriese, A.S., Fervenza, F.C. (2022). Acute glomerulonephritis. Lancet, 399(10335),1646-1663. doi: 10.1016/S0140-6736(22)00461-5

Siener R. (2018). Dietary Treatment of Metabolic Acidosis in Chronic Kidney Disease. Nutrients, 10(4), 512. https://doi.org/10.3390/nu10040512

Slee, A., Reid, J. (2022). Disease-related malnutrition in chronic kidney disease. Current opinion in clinical nutrition and metabolic care, 25(3), 136–141. https://doi.org/10.1097/MCO.0000000000000830

Wallace, M.A. (1998). Anatomy and physiology of the kidney. AORN journal, 68(5), 800–824. https://doi.org/10.1016/s0001-2092(06)62377-6

Wang, C.S., Greenbaum, L.A. (2019). Nephrotic Syndrome. Pediatric clinics of North America, 66(1), 73–85. https://doi.org/10.1016/j.pcl.2018.08.06

Wang, Z., Zhang, Y., Zhang, J., Deng, Q., Liang, H. (2021). Recent advances on the mechanisms of kidney stone formation. International journal of molecular medicine, 48 (2), 149. https://doi.org/10.3892/ijmm.2021.4982

Yoshizawa N. (2000). Acute glomerulonephritis. Internal medicine (Tokyo, Japan), 39(9), 687–694. https://doi.org/10.2169/internalmedicine.39.687

Zakrocka, I., Załuska, W. (2022). Kynurenine pathway in kidney diseases. Pharmacological Reports, 74(1), 27-39.

Zhao, J., Chan, Y.C., He, B., Duan, T.T., Yu, Z.L. (2019). A patent herbal drug Yi-Shen-Hua-Shi granule ameliorates C-BSA-induced chronic glomerulonephritis and inhabits TGFβ signaling in rats. Journal of ethnopharmacology, 236, 258–262. https://doi.org/10.1016/j.jep.2019.02.044