SHORT COMMUNICATION

Exercise and lifestyle in chronic renal disease: A clinical guideline

Elena Moore

Moore E. Exercise and lifestyle in chronic renal disease: a clinical guideline. J Kidney Treat Diagn. 2022; 5(2):20-22.

ABSTRACT

The phrase "if exercise were a pill, it would be one of the most widely prescribed and cost-effective drugs ever invented" has been repeated many times, with slight variations, and for good reason: the evidence is compelling, and the message is clear: being active is the foundation for a longer, healthier, and happier life. Although other national and international kidney disease guidelines include some basic physical activity and lifestyle recommendations, this is the first document of its kind to lay out the evidence for people with kidney disease, including those on dialysis and those who have had a kidney transplant.

Key Words: *Chronic renal disease; Glomerular filtration rate; Hyperkalemia; Nephrotoxins*

INTRODUCTION

CKD affects 8% to 16% of the global population and is defined as a chronic impairment in kidney structure or function (eg, glomerular glomerular filtration rate [GFR] 60mL/min/1.73 m2 or albuminuria 30mg per 24 hours) for more than 3 months. Diabetes and hypertension are the most common causes of CKD in affluent countries. However, only about 5% of individuals with early CKD are aware of their condition. Staging and novel risk assessment techniques that integrate GFR and albuminuria can assist guide therapy, monitoring, and referral strategies for people with CKD [1]. Hyperkalemia, metabolic acidosis, hypophosphatemia, vitamin D deficiency, secondary hyperparathyroidism, and anemia are all consequences of CKD that must be monitored. Those at high risk of developing CKD (e.g., estimated GFR 30 mL/min/1.73 m2, albuminuria 300mg per 24 hours, or rapid reduction in estimated GFR) should see a nephrologist right away. CKD is more common in low- and middle-income countries than in high-income countries, as defined by a Glomerular Filtration Rate (GFR) of less than 60 mL/min/1.73 m2, albuminuria of at least 30mg per 24 hours, or markers of kidney damage (eg, hematuria or structural abnormalities such as polycystic or dysplastic kidneys) persisting for more than 3 months [2]. In Asia, Sub-Saharan Africa, and many poor nations, CKD is most typically linked to diabetes and/or hypertension, although additional causes such as glomerulonephritis, infection, and environmental exposures (such as air pollution, herbal medicines, and pesticides) are also common. Because advancing CKD is linked to negative clinical outcomes such as end-stage kidney disease (ESKD), cardiovascular disease, and increased mortality, early detection and treatment by primary care providers is critical [3]. The evaluation and management of CKD should be done using a risk-based approach, according to new professional guidelines. This review focuses on the diagnosis, evaluation, and management of CKD for primary care clinicians and includes discussion of novel calculators for determining risk of CKD progression that may be useful in clinical practice. There are further considerations for referring to a nephrologist and starting dialysis [4].

Chronic kidney disease is usually discovered as a result of routine screening with a serum chemical profile and urine tests, or as an unexpected finding. Symptoms such as gross hematuria, "foamy urine" (a sign of albuminuria), notarial, flank pain, and decreased urine flow are less prevalent. Patients with advanced CKD may have fatigue, nausea, vomiting, metallic taste, unintentional weight loss, pruritus, mental state changes, dyspnea, or peripheral edema. When examining a patient with CKD [5], doctors should look for other symptoms that could indicate a systemic etiology (e.g., hemoptysis, rash, lymphadenopathy, hearing loss, and neuropathy) or urinary obstruction (e.g, urinary hesitancy, urgency, or frequency or incomplete bladder emptying). Patients should also be evaluated for kidney disease risk factors, such as prior exposure to nephrotoxins (e.g., non-steroidal anti-inflammatory drugs [NSAIDs], phosphate-

Editorial Office, Journal of Kidney Treatment and Diagnosis, United Kingdom

Correspondence: Elena Moore, Editorial Office, Journal of Kidney Treatment and Diagnosis, United Kingdom, E-mail kidney@eclinicalsci.org

Received: 22-Feb-2022, Manuscript No. PULJKTD-22-4517; Editor assigned: 24-Feb-2022, PreQC No. PULJKTD-22-4517 (PQ); Reviewed: 09-Mar-2022, QC No. PULJKTD-22-4517(Q); Revised: 11-Mar-2022, Manuscript No. PULJKTD-22-4517 (R); Published: 25-Mar-2022, DOI: 10.37532/puljktd.22.5(2).20-22

based bowel preparations, herbal remedies containing aristolochic acid, antibiotic therapies such as gentamicin, and chemotherapies), history of nephrolithiasis or recurrent urinary tract infections, and presence of comorbidities [6].

DISCUSSION

The presence or absence of systemic illness, as well as the location of anatomic abnormalities, are used to classify the cause of CKD. Diabetes, autoimmune illnesses, chronic infection, cancer, and hereditary disorders are examples of systemic disease in which the kidney is not the only organ affected. Glomerular, tubule-interstitial, vascular, and cystic/congenital illnesses are the different types of anatomic sites. The cause of CKD may have a significant impact on prognosis and treatment options [7]. Polycystic kidney disease, for example, may develop to ESKD more quickly than other causes, necessitating examination for extra renal symptoms as well as consideration of specific medications like tolvaptan, a vasopressin V2 receptor antagonist that slows GFR reduction. Patients with CKD who have no known etiology should be referred to a nephrologist [8]. Nonwhite race, low education, low income, and food poverty are all sociodemo-graphic characteristics that lead to an elevated risk of CKD. African Americans and Pacific Islanders have a significantly higher risk of ESKD than whites. This is related to a rise in the prevalence of hypertension, diabetes, and obesity, among other factors. However, genetic variables are likely to play a role. Individuals with 2 APOL1 risk alleles (present in about 13% of African Americans) had a 2-fold risk of CKD progression and a 29-fold risk of certain CKD etiologies (eg, focal-segmental glomerular sclerosis and HIV-associated nephropathy) compared to those with 0 or 1 risk allele [9].

As a result, lowering cardiovascular risk is an important part of CKD care. Patients with CKD who are 50 years or older, regardless of their low-density lipoprotein cholesterol level, should be treated with a low- to moderate-dose statin. It's also a good idea to encourage people to quit smoking. According to professional opinion, persons with CKD should have systolic and diastolic blood pressures of less than 140mm/Hg and less than 90 mm Hg, respectively, according to the Eighth Joint National Committee (JNC 8) and Kidney Disease: Improving Global Outcomes (KDIGO) guidelines [10]. Adults with urine ACR of at least 30mg per 24 hours (or similar) should have systolic and diastolic blood pressures below 130mm/Hg and 80mm/Hg, respectively, according to the KDIGO guidelines. 5 More recently, the Systolic Blood Pressure Intervention Trial (SPRINT) found that more intensive blood pressure control (goal systolic blood pressure 120mm/Hg) was associated with a 25% lower risk of a major cardiovascular event (1.65% vs 2.19% per year) and a 27% lower risk of all-cause mortality compared to standard blood pressure control (goal systolic blood pressure 140mm/Hg) among people at risk. Importantly, those with and without baseline CKD experienced equal advantages from aggressive blood pressure control in terms of cardiovascular events [11]

It's also crucial to have the best diabetes management possible. Glycemic management, for starters, may help to slow the course of CKD, with most guidelines advising a hemoglobin A1c aim of less than 7.0%. Second, oral hypoglycemic medications may require dose changes. Drugs metabolized by the liver and/or partially excreted bythe kidneys (eg, metformin and some dipeptide peptidase 4 [DPP-4] and sodium-glucose cotransporter-2 [SGLT-2] inhibitors) should be avoided in general, whereas drugs metabolized by the liver and/or partially excreted by the kidneys (e.g., metformin and some DPP-4 and sodium-glucose cotransporter-2 [SG Third, in those with highly elevated albuminuria, the usage of certain pharmaceutical types such as SGLT-2 inhibitors should be investigated [12]. The Canagliflozin and Renal Events in Diabetes with Established Nephropathy Clinical Evaluation (CREDENCE) trial found that among 4401 patients with type 2 diabetes and CKD stage G2-G3/A3 (baseline eGFR 30 mL/min/1.73 m2 to 90 mL/min/1.73 m2 and urine ACR>300 to 5000 mg/24 hours) taking ACE-I or ARB therapy, those randomized to canagliflozin had a 30% lower risk (43.2 Prior studies have demonstrated that this class of medicines has cardiovascular benefits, which may extend to people with CKD who have lower levels of albuminuria [13].

Nephrotoxins should be avoided by all patients with CKD. While a comprehensive list is beyond the scope of this study, a handful stands out. NSAIDs should not be taken on a regular basis in people with CKD, especially if they are also on ACE-I or ARB medication. The US Food and Drug Administration does not regulate herbal remedies, and some of them (such as those containing aristolochic acid or anthraquinones) have been linked to a variety of kidney problems, including acute tubular necrosis, acute or chronic interstitial nephritis, nephrolithiasis, rhabdomyolysis, hypokalemia, and Fanconi syndrome. Acute phosphate nephropathy can be caused by phosphate-based bowel treatments (both oral and enema formulations) that are commonly accessible over the counter. Proton pump inhibitors are commonly prescribed and have been linked to acute interstitial nephritis and incident CKD in case reports and population-based research [14]. The incidence of CKD was 14.2 occurrences per 1000 persons in the population-based Atherosclerosis Risk in Communities cohort, compared to 10.7 events per 1000 people who did not take proton pump inhibitors. It is not necessary to stop using proton pump inhibitors all at once if you have CKD. Iron stores should be assessed as part of the initial workup for anemia; those who are iron deficient may benefit from oral or intravenous iron replacement. Patients whose hemoglobin levels remain below 10 g/dL despite addressing reversible causes may be referred to a nephrologist for further medical treatment, including erythropoietin-stimulating agents; however, these drugs have been linked to an increased risk of death, stroke, and venous thromboembolism, so the risks must be weighed against the potential benefits [15]. Electrolyte abnormalities are found in 3% to 11% of CKD patients. Dietary restrictions and supplement prescriptions are generally part of the first treatment plan. For patients with hyperkalemia, primary care providers should offer low-potassium diets, and for individuals with hypophosphatemia, low-phosphorus diets. Oral bicarbonate supplementation should be considered for individuals with a serum bicarbonate level that is consistently less than 22mmol/L, as studies have linked chronic metabolic acidosis to quicker CKD progression [16].

CONCLUSION

Chronic kidney disease affects 8% to 16% of the world's population and is the primary cause of death. Cardiovascular risk reduction,

albuminuria treatment, avoidance of possible nephrotoxins, and drug dose modifications are all part of optimal CKD management. Complications of CKD, such as hyperkalemia, metabolic acidosis, anemia, and other metabolic abnormalities, must also be monitored. Primary care professionals are critical in reducing the global burden of CKD by diagnosing, staging, and appropriately referring patients with CKD.

REFERENCES

1. Coresh J, Selvin E, Stevens LA, et al. Prevalence of chronic kidney disease in the United States. JAMA. 2007;298(17):2038-2047.

2. Hsu CY, Vittinghoff E, Lin F, et al. The incidence of end-stage renal disease is increasing faster than the prevalence of chronic renal insufficiency. Ann Intern Med. 2004;141(2):95-101.

3. Jha V, Garcia G, Iseki K, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013;382(9888):260-272.

4. Levin A, Stevens PE, Bilous RW, et al. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kid Intern Suppl. 2013;3(1):1-50.

5. Mills KT, Xu Y, Zhang W, et al. A systematic analysis of worldwide population-based data on the global burden of chronic kidney disease in 2010. Kidney Intern. 2015;88(5):950-957.

6. Genovese G, Friedman DJ, Ross MD, et al. Association of trypanolytic ApoL1 variants with kidney disease in African Americans. Science. 2010;329(5993):841-845.

7. Tzur S, Rosset S, Shemer R, et al. Missense mutations in the APOL1 gene are highly associated with end stage kidney disease risk previously attributed to the MYH9 gene. Human Gene. 2010;128(3):345-350.

8. Naik RP, Derebail VK, Grams ME, et al. Association of sickle cell trait with chronic kidney disease and albuminuria in African Americans. JAMA. 2014;312(20):2115-2125.

9. Grams ME, Rebholz CM, Chen Y, et al. Race, APOL1 risk, and eGFR decline in the general population. J Am Soc Nephrol. 2016;27(9):2842-2850.

10. Grams ME, Chow EK, Segev DL, et al. Lifetime incidence of CKD stages 3-5 in the United States. Am J Kid Dis. 2013;62(2):245-252.

11. Matsushita K, Coresh J, Sang Y, et al. Estimated glomerular filtration rate and albuminuria for prediction of cardiovascular outcomes: a collaborative meta-analysis of individual participant data. Lancet Diab Endocrinol. 2015;3(7):514-525.

12. Inker LA, Astor BC, Fox CH, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for the evaluation and management of CKD. Am J Kid Dis. 2014;63(5):713-735.

13. Taal MW, Chertow GM, Marsden PA, et al. Brenner and Rector's the Kidney E-Book. Elsev Health Sci. 2011.

14. Yang B, Xie Y, Guo M, et al. Nephrotoxicity and Chinese herbal medicine. Clin J Am Soc Nephrol. 2018;13(10):1605-1611.

15. Braden GL, O'Shea MH, Mulhern JG. Tubulointerstitial diseases. Am J Kid Dis. 2005;46(3):560-572.

16. Brown SC, O'Reilly PH. Iohexol clearance for the determination of glomerular filtration rate in clinical practice: evidence for a new gold standard. J Urol. 1991;146(3):675-659.