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Chronic Kidney Disease: A Review

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Abstract

Chronic kidney disease is a progressive disease with no cure and high morbidity and high mortality that occurs commonly in the general population, especially in people with diabetes mellitus and hypertension. Chronic kidney disease (CKD) affects between 8% and 16% of the population worldwide and is often under recognized by patients and clinical specialist. Defined by a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2, albuminuria of at least 30 mg per day, or markers of kidney damage (eg, hematuria or structural abnormalities such as polycystic or dysplastic kidneys) persisting for more than 3 months, CKD is more prevalent in low- and middle-income than in high-income countries. Globally, CKD is most commonly attributed to diabetes and/or hypertension, but other causes such as glomerulonephritis, infections, and environmental exposures (such as air pollution, herbal remedies, and pesticides) are common in Asia, sub-Saharan Africa, and many developing countries. Some glomerular and cystic kidney diseases might benefit from disease-specific therapies. Managing chronic kidney disease-associated cardiovascular risk, minimising the risk of infection, and preventing acute kidney injury are crucial interventions for these patients, given the high burden of complications, associated morbidity and mortality, and the role of non-conventional risk factors in chronic kidney disease.

Keywords: Chronic kidney disease • Glomerulonephritis • Complications.

Introduction

History of CKD

In 1960 with a ground breaking medical advance. The invention of the Teflon shunt made repeated access to a patient's blood possible. Kidney failure could then be treated with dialysis. KDOQI—Kidney Disease Outcomes Quality Initiative. The first guidelines were published in 1997, and today there are 12 guidelines, which are setting the standards for treatment of all aspects of kidney disease. Recognition of kidney disease as independent from other medical conditions is widely attributed to Richard Bright's 1827 book "Reports of Medical Cases," the first report confirms the emergence of diabetic nephropathy as the pre-eminent cause in india. Patients with ckd of unknown etiology are younger, poorer and more likely to present with advanced ckd there were some geographic variations. Chronic kidney disease (CKD) affects between 8% and 16% of the population worldwide and is often underrecognized by patients and clinicians. Defined by a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m2, albuminuria of at least 30 mg 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, CKD is more prevalent in low- and middle-income than in high-income countries. Globally, CKD is most commonly attributed to diabetes and/or hypertension, but other causes such as glomerulonephritis, infection, and environmental exposures (such as air pollution, herbal remedies, and pesticides) are common in Asia, sub-Saharan Africa, and many developing countries. Genetic risk factors may also contribute to CKD risk. For example, sickle cell trait and the presence of 2 APOL1 risk alleles, both common in people of African ancestry but not European ancestry, may double the risk of CKD. In the United States, the average rate of GFR decline is approximately 1 mL/min/1.73 m^2 per year in the general population and the lifetime risk of developing a GFR of less than 60 mL/min/1.73 m^2 is more than 50%. Early detection and treatment by primary care clinicians is important because progressive CKD is associated with adverse clinical outcomes, including end-stage kidney disease (ESKD), cardiovascular disease, and increased mortality. Recent professional guidelines suggest a risk-based approach to the evaluation and management of CKD [1].

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Literature Review

Signs and symptoms of chronic kidney disease develop over time if kidney damage progresses slowly. Loss of kidney function can cause a buildup of fluid or body waste or electrolyte problems. Depending on how severe it is, loss of kidney function can cause: Nausea, Vomiting, Loss of appetite, Fatigue and weakness, Sleep problems, Urinating more or less, Decreased mental sharpness, Muscle cramps, Swelling of feet and ankles, Dry, itchy skin, High blood pressure (hypertension) that's difficult to control, Shortness of breath, if fluid builds up in the lungs, Chest pain, if fluid builds up around the lining of the heart.

Signs and symptoms of kidney disease are often nonspecific. This means they can also be caused by other illnesses. Because your kidneys are able to make up for lost function, you might not develop signs and symptoms until irreversible damage has occurred.

Stages

- **Stage 1:** Normal eGFR ≥ 90 mL/min per 1.73 m^2 and persistent albuminuria
- **Stage 2:** eGFR between 60 to 89 mL/min per 1.73 m^2
- **Stage 3:** eGFR between 30 to 59 mL/min per 1.73 m^2
- **Stage 4:** eGFR between 15 to 29 mL/min per 1.73 m^2
- **Stage 5:** eGFR of < 15 mL/min per 1.73 m^2 or end-stage renal disease

Pathophysiology

1. Proximal tubules as a primary target of AKI

2. Intrinsic regenerative potential of proximal tubule cells

3. Injured proximal tubule as a driver of AKI to CKD progression

4. Injured proximal tubules in G2 /M cell cycle arrest promote renal inflammation and fibrosis

5. Aberrant reactivation of the developmental pathway promotes fibrosis.

6. Mitochondrial dysfuntion in injured proximal tubular cells

7. Fibroblast dysfuntion is a central pathogenic process in CKD progression after AKI.

Factors that can increase your risk of chronic kidney disease include: Diabetes, High blood pressure, Heart (cardiovascular) disease, Smoking, Obesity, Being Black, Native American or Asian American, Family history of kidney disease, Abnormal kidney structure, Older age, Frequent use of medications that can damage the kidneys.

Complications

Chronic kidney disease can affect almost every part of your body. Potential complications include: Fluid retention, which could lead to swelling in your arms and legs, high blood pressure, or fluid in your lungs (pulmonary edema). A sudden rise in potassium levels in your blood (hyperkalemia), which could impair your heart's function and can be life-threatening. Anemia, Heart disease, Weak bones and an increased risk of bone fractures, Decreased sex drive, erectile dysfunction or reduced fertility. Damage to your central nervous system, which can cause difficulty concentrating, personality changes or seizures. Decreased immune response, which makes you more vulnerable to infection. Pericarditis, an inflammation of the saclike membrane that envelops your heart (pericardium). Pregnancy complications that carry risks for the mother and the developing fetus. Irreversible damage to your kidneys (end-stage kidney disease), eventually requiring either dialysis or a kidney transplant for survival.

Prevention

To reduce your risk of developing kidney disease: Follow instructions on over-the-counter medications. When using nonprescription pain relievers, such as aspirin, ibuprofen (Advil, Motrin IB, others) and acetaminophen (Tylenol, others), follow the instructions on the package. Taking too many pain relievers for a long time could lead to kidney damage. Maintain a healthy weight. If you're at a healthy weight, maintain it by being physically active most days of the week. If you need to lose weight, talk with your doctor about strategies for healthy weight loss. Don't smoke. Cigarette smoking can damage your kidneys and make existing kidney damage worse. If you're a smoker, talk to your doctor about strategies for quitting. Support groups, counseling and medications can all help you to stop. Manage your medical conditions with your doctor's help. If you have diseases or conditions that increase your risk of kidney disease, work with your doctor to control them. Ask your doctor about tests to look for signs of kidney damage.

Pharmacoepidemiology

In 2017, 697.5 million cases of CKD (all stages) were recorded worldwide, for a global prevalence of 9.1%. From 1990 to 2017, the global all-age prevalence of CKD increased 29.3%, whereas the age-standardized prevalence remained stable. Globally, 1.2 million people died from CKD in 2017. The global all-age mortality rate from CKD increased 41.5% from 1990 to 2017. Diabetic nephropathy accounted for almost a third of disability-adjusted life years (DALYs) from CKD. Most of the burden of CKD was concentrated in the three lowest quintiles of the Socio-demographic index (SDI).

Race/ethnic-related demographics

In the US, the percentage of adults with CKD is as follows:

- Non-Hispanic whites: 12.7%
- Non-Hispanic Blacks: 16.3%
- Non-Hispanic Asians: 12.29%
- Hispanics: 13.6%

In non-Hispanic white and non-Hispanic Black persons, the prevalence of stage 3 and 4 CKD has remained stable since 2004. In Mexican Americans, the prevalence of CKD in Mexican Americans had been lower than in other racial/ethnic groups, but nearly doubled between 2003-2004 and 2015-2016, from 1.6% to 3.5%. The incidence rate of ESRD among Blacks in the United States is nearly 4 times that for whites. Choi et al found that rates of ESRD among Black patients exceeded those among white patients at all levels of baseline estimated glomerular filtration rate (GFR). Risk of ESRD among Black patients was highest at an estimated GFR of 45-59 mL/min/1.73 m^2, as was the risk of mortality.

Schold, et al. found that among Black kidney transplant recipients, rates of graft loss and acute rejection were higher than in white recipients, especially among younger patients. Hicks et al looked at the connection between Black patients with the sickle cell trait and their increased risk for kidney disease; the study found that sickle cell trait was not associated with diabetic or nondiabetic ESRD in a large sample of Black patients. Important differences also exist in the frequency of specific causes of CKD among different races. In the Chronic Kidney Disease in Children (CKiD) Study, for example, glomerular disease was much more common among nonwhite persons. Overall, FSGS in particular is more common among Hispanic Americans and Black persons, as is the risk of nephropathy with diabetes or with hypertension; in contrast, IgA nephropathy is rare in Black individuals and more common among those with Asian ancestry [2]. Chronic kidney disease is a progressive disease with no cure and high morbidity and mortality that occurs commonly in the general population, especially in people with diabetes and hypertension. Preservation of kidney function can improve outcomes and can be achieved through non-pharmacological strategies (eg, dietary and lifestyle adjustments) and chronic kidney disease-targeted and kidney disease-specific pharmacological interventions [3]. A plant-dominant, low-protein, and low-salt diet might help to mitigate glomerular hyperfiltration and preserve renal function for longer, possibly while also leading to favourable alterations in acid-base homoeostasis and in the gut microbiome. Pharmacotherapies that alter intrarenal haemodynamics (e.g., renin–angiotensin–aldosterone pathway modulators and SGLT2 [SLC5A2] inhibitors) can preserve kidney function by reducing intraglomerular pressure independently of blood pressure and glucose control, whereas other novel agents (eg, non-steroidal mineralocorticoid receptor antagonists) might protect the kidney through anti-inflammatory or antifibrotic mechanisms [4-6].

Some glomerular and cystic kidney diseases might benefit from disease-specific therapies. Managing chronic kidney disease-associated cardiovascular risk, minimising the risk of infection, and preventing acute kidney injury are crucial interventions for these patients, given the high burden of complications, associated morbidity and mortality, and the role of non-conventional risk factors in chronic kidney disease [7-9]. When renal replacement therapy becomes inevitable, an incremental transition to dialysis can be considered and has been proposed to possibly preserve residual kidney function longer. There are similarities and distinctions between kidney-preserving care and supportive care [10].

Additional studies of dietary and pharmacological interventions and development of innovative strategies are necessary to ensure optimal kidney-preserving care and to achieve greater longevity and better health-related quality of life for these patients [11-12].

Conclusion

Chronic kidney disease is a progressive disease with no cure and high morbidity and mortality that occurs commonly in the general population, especially in people with diabetes and hypertension. Preservation of kidney function can improve outcomes and can be achieved through non-

pharmacological strategies (e.g., dietary and lifestyle adjustments) and chronic kidney disease-targeted and kidney disease-specific pharmacological interventions.

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