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Chronic Kidney Disease: Unveiling the Silent Epidemic and Its Pathophysiology

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Annotation: Chronic Kidney Disease (CKD) is a significant and growing global health issue, marked by the gradual and irreversible decline in kidney function. This progressive condition leads to the accumulation of waste products in the body, resulting in severe complications. Early detection and timely intervention are critical in managing CKD, yet the disease often progresses silently. This review explores the pathophysiology of CKD, including key mechanisms such as glomerular hyperfiltration, tubulointerstitial fibrosis, and chronic inflammation. It also examines the primary risk factors for CKD, including diabetes, hypertension, age, and genetic predisposition. Diagnostic methods, including the use of estimated glomerular filtration rate (eGFR) and proteinuria, are discussed, highlighting their importance in early diagnosis and disease monitoring. The review further evaluates current management strategies, including lifestyle modifications, pharmacological treatments, and renal replacement therapies for advanced stages of CKD. Additionally, recent advancements in CKD research are highlighted, particularly in the fields of precision medicine, stem cell therapy, and gene editing, which show promising potential for slowing disease progression and improving patient outcomes. Through a comprehensive overview, this review presents the current state of CKD, while offering insight into the future of research and treatment, emphasizing the need for continued innovation to combat the growing burden of CKD worldwide.

Keywords: Chronic Kidney Disease, Pathophysiology, Precision Medicine, Risk Factors, Diagnosis, Therapeutic Strategies, Kidney Failure, Renal Replacement Therapy.

1. Introduction

Chronic Kidney Disease (CKD) is a long-term condition characterized by the progressive and irreversible damage to the kidneys over time, impairing their ability to filter waste products and excess fluid from the bloodstream. In healthy individuals, the kidneys filter metabolic waste, regulate electrolyte levels, and maintain fluid balance, contributing to the overall homeostasis of the body. However, in CKD, this filtration process becomes impaired as the kidneys progressively lose function. As a result, waste products, such as urea and creatinine, accumulate in the bloodstream, and imbalances in electrolytes and fluid regulation arise. If left untreated, CKD can progress to end-stage renal disease (ESRD), which requires renal replacement therapy (RRT) in the form of dialysis or kidney transplantation [1-2]. The global burden of CKD has been steadily increasing, primarily driven by the rising prevalence of risk factors such as diabetes, hypertension, and an aging population. Diabetes mellitus is one of the leading causes of CKD, as high blood glucose levels damage the blood vessels in the kidneys, impairing their filtering ability. Hypertension, another major contributor, causes increased pressure on the blood vessels of the kidneys, leading to endothelial damage and promoting the development of kidney dysfunction. Additionally, aging populations are at higher risk for CKD due to the natural decline in renal function with advancing age. As the global population continues to age, the incidence of CKD is expected to rise even further, creating a substantial public health challenge [3].

CKD poses a significant healthcare burden, particularly in low- and middle-income countries, where access to healthcare resources and diagnostic tools may be limited. In these regions, the lack of early detection and treatment often leads to a higher incidence of CKD progression to ESRD. As CKD progresses, it often remains asymptomatic in its early stages, with individuals experiencing few noticeable symptoms until the kidney function is significantly impaired. This silent progression underscores the importance of regular screening for at-risk populations, including individuals with diabetes, hypertension, and a family history of kidney disease, to identify CKD at its early stages when interventions are most effective [4]. The early stages of CKD, often referred to as stage 1 and stage 2, may be manageable through lifestyle modifications and pharmacological treatments aimed at controlling underlying risk factors. These include blood pressure control, glycemic management for diabetic patients, and dietary modifications to reduce kidney strain. However, as the disease progresses, typically to stage 3 and beyond, more intensive management is required. At these stages, individuals may experience worsening symptoms such as fatigue, edema, and changes in urine output. If CKD progresses to stage 5, also known as ESRD, the kidneys lose nearly all of their function, and renal replacement therapy becomes necessary. Renal replacement therapy options include dialysis and kidney transplantation. Dialysis, which can be done through hemodialysis or peritoneal dialysis, serves as a life-sustaining treatment for patients with ESRD, but it is not a cure and does not restore kidney function. Kidney transplantation, when available, offers a potential cure for eligible patients, but the demand for donor kidneys far exceeds the available supply [5].

1.1 Epidemiology and Prevalence

Chronic Kidney Disease (CKD) affects approximately 10-15% of the global population, with the highest incidence observed in older individuals and those with underlying health conditions such as diabetes and hypertension. These risk factors significantly contribute to the development and progression of CKD, as diabetes leads to kidney damage through high blood sugar levels, while hypertension causes stress on the kidneys' blood vessels, impairing their function. The increasing prevalence of these conditions, particularly in aging populations, is contributing to a rise in CKD cases, exacerbating the global health burden [6]. The global impact of CKD is made more severe by the rapid rise in diabetes, hypertension, and an aging population. As the prevalence of these risk factors increases, CKD has become one of the leading causes of mortality worldwide. The disease is often asymptomatic in its early stages, which can lead to delayed diagnosis and intervention, allowing kidney function to deteriorate. When left untreated, CKD progresses to end-stage renal disease (ESRD), requiring costly renal replacement therapies such as dialysis or kidney transplantation [7]. As CKD advances, it leads to numerous debilitating complications, including fluid retention, electrolyte imbalances, anemia, and cardiovascular problems, further deteriorating the patient's quality of life. Advanced CKD and ESRD impose a significant financial strain on healthcare systems, as they require continuous and expensive medical care. This includes not only dialysis or transplantation but also the management of related conditions such as cardiovascular disease. The increasing prevalence of CKD places immense pressure on medical resources, straining healthcare infrastructures, particularly in low- and middle-income countries with limited access to quality care [8].

2. Pathophysiology of Chronic Kidney Disease

Chronic Kidney Disease (CKD) progresses through a series of pathophysiological stages, beginning with minimal symptoms and gradually worsening as kidney function declines. The underlying mechanisms driving CKD are multifactorial, involving processes such as glomerular hyperfiltration, tubulointerstitial fibrosis, chronic inflammation, endothelial dysfunction, and oxidative stress. These factors collectively contribute to kidney damage and disease progression. In the early stages of CKD, glomerular hyperfiltration occurs as a compensatory mechanism. Remaining nephrons attempt to compensate for damaged nephrons by increasing their filtration rate, which helps to maintain kidney function [9]. However, this increased filtration pressure places significant stress on the glomerular capillaries, leading to further damage. Over time, the glomeruli become scarred (glomerulosclerosis), reducing the kidney's ability to filter waste and contributing to the progressive decline in kidney function. Another critical mechanism in CKD progression is tubulointerstitial fibrosis [10]. This process involves the accumulation of extracellular matrix proteins, particularly collagen, in the renal interstitial and tubular areas. As the renal tubules become damaged, they lose their ability to reabsorb electrolytes and water, exacerbating fluid retention and electrolyte imbalances. The fibrosis extends to the interstitial tissue, leading to further nephron loss and a decline in overall kidney function. Tubulointerstitial fibrosis is irreversible and strongly correlates with the progression to end-stage renal disease (ESRD) [11].

Chronic inflammation also plays a central role in CKD. When the kidneys are injured, immune cells such as macrophages, T cells, and neutrophils are recruited to the site of damage. These cells release inflammatory cytokines and growth factors, promoting further kidney injury and fibrosis. Key pro-inflammatory mediators, such as tumor necrosis factor-alpha (TNF-α), interleukins (e.g., IL-6), and transforming growth factor-beta (TGF-β), are involved in the activation of fibroblasts, which contribute to fibrosis [12]. The inflammatory response further exacerbates kidney damage, creating a vicious cycle that accelerates disease progression. Endothelial dysfunction is another important factor in CKD. Damage to the endothelial cells lining the blood vessels of the kidneys leads to an imbalance between vasodilatory and vasoconstrictive factors, impairing renal blood flow. This increase in vascular resistance further contributes to glomerular injury. Additionally, endothelial dysfunction is associated with systemic hypertension, a common comorbidity in CKD, which accelerates kidney damage [13]. The dysfunction also increases the risk of thrombotic events, which further impair kidney function. Oxidative stress, the imbalance between reactive oxygen species (ROS) and antioxidant defenses, also plays a significant role in CKD. ROS promote inflammation, fibrosis, and endothelial dysfunction, all of which contribute to the decline in kidney function. Additionally, dyslipidemia, commonly seen in CKD patients, accelerates atherosclerosis, stiffening renal blood vessels and impairing kidney perfusion, further exacerbating kidney damage [14].

3. Risk Factors for Chronic Kidney Disease

Several key factors predispose individuals to Chronic Kidney Disease (CKD), with diabetes mellitus and hypertension being the most prominent risk factors. In addition to these, other contributing factors include obesity, a family history of kidney disease, and the natural decline in renal function with aging. Understanding these risk factors is essential for the prevention and early management of CKD, as they play significant roles in the initiation and progression of the disease [15].

3.1 Diabetes Mellitus

Diabetes is the leading cause of CKD worldwide, accounting for a substantial proportion of cases. The pathogenesis of CKD in diabetic patients primarily arises from persistent hyperglycemia, which causes direct damage to the kidney’s glomeruli. Chronic high blood sugar levels lead to the accumulation of advanced glycation end products (AGEs), which bind to kidney cells and promote inflammation, oxidative stress, and fibrosis. Over time, these changes result in glomerulosclerosis, a condition characterized by scarring and hardening of the glomeruli, impairing the kidney's ability to filter blood effectively [16]. Diabetic nephropathy, a complication of diabetes, is one of the most common and serious forms of CKD, ultimately progressing to end-stage renal disease (ESRD) if left untreated. Diabetic nephropathy is marked by increased proteinuria, a key early sign of kidney damage, and decreased glomerular filtration rate (GFR). Tight glycemic control is essential to slow the progression of kidney damage in diabetic patients. Additionally, other interventions such as the use of angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) can help manage blood pressure and reduce proteinuria, further protecting kidney function [17].

3.2 Hypertension

Hypertension is the second most common cause of CKD, contributing significantly to the initiation and progression of kidney disease. High blood pressure leads to damage of the glomerular capillaries due to the excessive pressure exerted on the delicate kidney structures. This increased pressure accelerates the development of glomerulosclerosis, leading to reduced kidney function. Moreover, hypertension induces endothelial dysfunction, which reduces renal blood flow and exacerbates kidney injury [18]. As the kidneys attempt to compensate for the reduced function, they experience glomerular hyperfiltration, which further damages the nephrons. Over time, the kidneys undergo irreversible changes, with the accumulation of fibrosis and the development of tubulointerstitial damage. Managing hypertension is crucial in preventing CKD progression, as sustained high blood pressure accelerates kidney damage. Lifestyle changes, such as weight loss, a low-sodium diet, and regular exercise, along with the use of antihypertensive medications, are essential in controlling blood pressure and protecting kidney function. ACE inhibitors and ARBs are particularly beneficial in reducing both blood pressure and proteinuria, further slowing CKD progression [19].

3.3 Age and Genetics

Age is a significant risk factor for CKD, as kidney function naturally declines with advancing age. The glomerular filtration rate (GFR), which is a key indicator of kidney function, decreases progressively with age, even in individuals without underlying kidney disease. This decline in kidney function makes older adults more susceptible to the development of CKD, especially when compounded by other risk factors such as hypertension or diabetes. As people age, the kidneys also become less efficient in filtering waste products and maintaining fluid balance, making them more vulnerable to damage from environmental factors, medications, or other diseases [20]. Additionally, the risk of developing CKD increases in individuals with a family history of kidney disease. Inherited conditions, such as polycystic kidney disease (PKD) and Alport syndrome, predispose individuals to kidney failure at a younger age. PKD is particularly notable for its genetic basis, as it is an autosomal dominant condition that leads to the formation of cysts within the kidneys, eventually causing kidney enlargement and functional decline. Other genetic factors that influence kidney function, such as mutations in genes associated with glomerular filtration, also contribute to CKD susceptibility [21].

4. Diagnosis of Chronic Kidney Disease

Timely diagnosis of Chronic Kidney Disease (CKD) is critical to preventing irreversible kidney damage and managing the disease effectively. The primary diagnostic approach involves assessing kidney function through various biomarkers, such as serum creatinine, estimated glomerular filtration rate (eGFR), and proteinuria, which can provide vital information about the extent of kidney damage. One of the most common laboratory tests used in the diagnosis of CKD is the measurement of serum creatinine levels [22]. Creatinine is a byproduct of muscle metabolism, and its concentration in the blood reflects the kidney's ability to filter and excrete waste. Elevated serum creatinine levels are an indicator of reduced kidney function. The eGFR, calculated using serum creatinine along with factors such as age, sex, and ethnicity, is a critical measure to assess kidney function. An eGFR below 60 mL/min/1.73 m² for three months or longer is a key criterion for diagnosing CKD. Proteinuria, which refers to the presence of excess protein in the urine, is another important marker of kidney damage [23]. The urine albumin-to-creatinine ratio (UACR) is used to measure the amount of albumin in the urine, with higher levels indicating kidney injury. In addition to laboratory tests, imaging studies such as ultrasonography play a crucial role in diagnosing CKD. Ultrasound imaging can help assess kidney size, structure, and the presence of abnormalities like cysts, tumors, or obstructive uropathy [24]. These findings can provide valuable information regarding the underlying cause of CKD and help clinicians determine the best course of action for treatment. In some cases, when the cause of CKD is unclear or when a more detailed analysis is needed, a kidney biopsy may be performed. A biopsy involves taking a small sample of kidney tissue to examine the underlying pathological changes, which can help identify specific causes of CKD, such as glomerulonephritis or inherited kidney disorders. This procedure provides a more comprehensive understanding of the condition and aids in tailoring treatment strategies [25].

5. Clinical Management of Chronic Kidney Disease

The clinical management of Chronic Kidney Disease (CKD) focuses on controlling risk factors, slowing disease progression, and mitigating complications. The primary goal is to reduce the burden of CKD-related complications, preserve kidney function for as long as possible, and improve the patient's quality of life. Effective management strategies typically include a combination of lifestyle modifications, pharmacological treatments, and, in advanced stages, renal replacement therapy. Early intervention is crucial to prevent the progression of CKD to end-stage renal disease (ESRD) [26].

5.1 Lifestyle Modifications

Lifestyle modifications play an essential role in the management of CKD, particularly in the early stages, where interventions can help slow progression and reduce complications [27].

Dietary Adjustments: Dietary changes are fundamental in managing CKD. A low-protein diet has been shown to reduce the burden on the kidneys by minimizing the accumulation of waste products like urea and creatinine. Reducing protein intake can help prevent the acceleration of kidney damage. A low-sodium diet is also recommended to control blood pressure and reduce fluid retention, both of which are crucial for slowing the progression of CKD. Additionally, patients with CKD should avoid excessive potassium and phosphorus intake, as impaired kidney function may lead to difficulties in excreting these electrolytes, resulting in dangerous imbalances [28].

Weight Management: Obesity is a significant risk factor for CKD progression. Weight management through a balanced diet and regular physical activity can significantly improve cardiovascular health and slow CKD progression. Maintaining a healthy weight reduces the strain on the kidneys and helps manage associated comorbidities, such as hypertension and diabetes, that exacerbate kidney damage [29].

Regular Physical Activity: Physical activity not only contributes to weight management but also improves cardiovascular health, which is vital in CKD management. Exercise can help lower blood pressure, regulate blood sugar levels, and reduce the risk of developing cardiovascular disease, which is common in CKD patients. However, the intensity and type of exercise should be individualized based on the patient's overall health and stage of CKD [30].

5.2 Pharmacotherapy

Pharmacological interventions are essential to manage the underlying conditions contributing to CKD and to slow its progression. These treatments are tailored to individual needs based on the stage of CKD, comorbidities, and specific risk factors [31].

Antihypertensive Agents: Hypertension is a major contributor to the development and progression of CKD. Managing blood pressure is a cornerstone of CKD treatment, particularly in patients with proteinuria (excess protein in urine). Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are the most commonly prescribed medications for controlling blood pressure in CKD patients. These drugs not only lower blood pressure but also help reduce proteinuria, a key marker of kidney damage. ACE inhibitors and ARBs have been shown to slow the progression of kidney disease by reducing the pressure inside the glomeruli, thus protecting the kidneys from further damage. These medications are particularly effective in patients with diabetic nephropathy, where proteinuria is a hallmark of kidney injury [32].

Glycemic Control: For diabetic patients, strict blood glucose management is essential in slowing the progression of diabetic nephropathy, one of the leading causes of CKD. High blood glucose levels contribute to kidney damage by increasing the accumulation of advanced glycation end products (AGEs), which damage kidney tissues and promote inflammation. Tight control of blood sugar levels can prevent or delay the onset of CKD in diabetic patients and minimize the risk of complications such as diabetic retinopathy and neuropathy. Medications such as SGLT2 inhibitors, which help lower blood glucose levels, have also been shown to have renal protective effects and are increasingly incorporated into CKD management for diabetic patients [33].

Statins:
Dyslipidemia, particularly elevated levels of low-density lipoprotein (LDL) cholesterol, is common in CKD and is associated with an increased risk of cardiovascular disease. Statins are lipid-lowering medications commonly prescribed to manage dyslipidemia in CKD patients. Statins reduce LDL cholesterol levels and have been shown to reduce the risk of cardiovascular events, such as heart attacks and strokes, which are common in CKD patients. In addition to their lipid-lowering effects, statins may also have pleiotropic effects that contribute to renal protection, though their direct effect on CKD progression remains an area of active research [34].

5.3 Renal Replacement Therapy

In advanced stages of CKD (stage 4 and stage 5), when kidney function declines to the point of renal failure, renal replacement therapy (RRT) becomes necessary. RRT is required to replace the lost function of the kidneys and maintain fluid and electrolyte balance [35].

Dialysis:
Dialysis is a life-sustaining treatment that helps remove waste products, excess fluids, and electrolytes from the blood when the kidneys can no longer perform these functions. There are two primary types of dialysis: hemodialysis and peritoneal dialysis [1-3].

- **Hemodialysis:** This process involves the use of a machine to filter blood outside the body. The blood is drawn from the patient's body, filtered through a dialyzer (artificial kidney), and returned to the body. Hemodialysis is typically performed three times a week and requires access to the bloodstream through a fistula or catheter [4].
- **Peritoneal Dialysis:** In this type of dialysis, the peritoneal membrane inside the abdomen is used as a natural filter. A dialysis solution is introduced into the abdomen, where it absorbs waste products from the blood. After several hours, the solution is drained and replaced with fresh fluid. Peritoneal dialysis can be performed at home and may offer more flexibility than hemodialysis [5].

Kidney Transplantation: Kidney transplantation is the preferred treatment for eligible patients with ESRD, as it offers the potential for a cure rather than just symptom management. A kidney transplant involves surgically placing a healthy donor kidney into the patient's body, restoring normal kidney function. While transplantation provides the best long-term outcomes, the demand for donor kidneys far exceeds the available supply, and patients must undergo immunosuppressive therapy to prevent organ rejection. Despite these challenges, kidney transplantation remains the gold standard for treating ESRD [11], significantly improving the quality of life and survival rates for patients. Chronic Kidney Disease (CKD) has long been a challenging condition to treat, with no definitive cure for advanced stages. However, recent breakthroughs in scientific research have spurred the development of innovative therapies aimed at halting or even reversing kidney damage. These therapies are at the forefront of CKD treatment, and include stem cell therapy, gene editing techniques, and precision medicine. While these therapies are still in the early stages of development, they offer promise in improving kidney function and providing new hope for patients with CKD [12].

6.1 Stem Cell Therapy

Stem cell therapy has emerged as one of the most promising areas of research for CKD treatment. The concept behind stem cell therapy is to use stem cells—undifferentiated cells with the potential to develop into various types of tissue—to repair or regenerate damaged kidney tissue. In CKD, nephron damage occurs progressively, leading to irreversible kidney dysfunction. Stem cells could potentially replace or repair damaged kidney structures, including the glomeruli and tubules, thus improving kidney function [13]. Research into stem cell therapy for CKD has focused on both autologous (from the patient's own body) and allogenic (from a donor) stem cell sources, including mesenchymal stem cells (MSCs), induced pluripotent stem cells (iPSCs), and renal progenitor cells. Early studies have shown that stem cells can promote tissue regeneration, reduce inflammation, and even prevent further scarring in the kidneys. In animal models, stem cells have been shown to enhance kidney repair by regenerating nephrons, improving glomerular filtration rate (GFR), and restoring tubular function [14].

While the potential for stem cell therapy in CKD is substantial, there are significant challenges to its clinical application. These include ensuring the long-term safety and efficacy of stem cell treatments, determining the best cell types for kidney repair, and addressing issues related to immune rejection and the control of cell differentiation. Despite these challenges, clinical trials are underway to evaluate the effectiveness of stem cell therapy in humans, and the results so far are promising. If these therapies prove successful, they could revolutionize CKD treatment by offering a potential means to regenerate kidney tissue and restore kidney function in patients with advanced stages of CKD [15].

6.2 Gene Therapy and Precision Medicine

Gene therapy and precision medicine represent another exciting frontier in the treatment of CKD. Advances in gene editing, particularly with technologies like CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats), are opening up new possibilities for treating inherited kidney diseases and tailoring treatments to individual patients based on their genetic profiles [36].

Gene Therapy: Gene therapy involves the direct modification of genes within a patient's cells to correct genetic defects or enhance cellular function. In the context of CKD, gene therapy may offer a way to treat inherited forms of kidney disease, such as polycystic kidney disease (PKD), which is caused by mutations in specific genes. By using CRISPR technology, researchers can potentially correct these genetic mutations at the DNA level, preventing the formation of cysts in PKD or other structural abnormalities associated with genetic kidney disorders. CRISPR offers a powerful tool for editing specific genes with high precision, which could lead to new treatments that prevent or even reverse kidney damage caused by genetic defects. The application of gene therapy to CKD is still in its early stages, and challenges such as ensuring efficient delivery of gene-editing tools to the kidneys, avoiding off-target effects, and managing immune responses need to be addressed. However, the potential for gene therapy to provide a long-term solution for genetic kidney diseases is significant, particularly in preventing the onset of CKD in high-risk populations [37].

Precision Medicine: Precision medicine is an approach that takes into account individual genetic differences, lifestyle factors, and environmental exposures to tailor treatments specifically for each patient. In CKD, precision medicine has the potential to optimize treatment plans based on a patient's genetic makeup, identifying those most at risk for disease progression and customizing interventions to improve outcomes. For example, genetic testing can identify specific mutations that affect drug metabolism or kidney function, allowing clinicians to select the most effective medications and avoid adverse reactions. One of the most promising applications of precision medicine in CKD is the ability to predict disease progression based on genetic risk factors. Patients with certain genetic variants may be at higher risk for developing more severe forms of CKD, and early interventions could be tailored to prevent progression. For instance, precision medicine could guide the use of targeted therapies like SGLT2 inhibitors or other novel medications that are particularly effective in certain genetic subgroups of CKD patients. Precision medicine also holds the potential to improve the management of comorbidities commonly associated with CKD, such as cardiovascular disease and diabetes, by identifying personalized treatment strategies that address both the kidney disease and its associated complications. As the field of genomics continues to evolve, the integration of genetic data into routine clinical practice will allow for more individualized and effective management of CKD [38-40].

7. Conclusion

Chronic Kidney Disease (CKD) continues to be one of the most significant health challenges globally, affecting millions of individuals and placing substantial strain on healthcare systems. With its increasing prevalence, particularly in populations with risk factors such as diabetes, hypertension, and aging, CKD poses a critical threat to public health. As the disease progresses, it leads to a loss of kidney function, ultimately requiring costly treatments like dialysis or kidney transplantation, which further intensifies the burden on healthcare infrastructure. Early detection of CKD is essential for improving patient outcomes. Identifying the disease in its early stages allows for timely interventions, including lifestyle modifications, pharmacological treatments, and better management of risk factors, such as hypertension and diabetes, which are key contributors to kidney damage. By controlling these underlying conditions, it is possible to slow the progression of CKD and delay the need for renal replacement therapies. In addition to managing risk factors, novel therapeutic strategies are showing great promise in improving CKD outcomes. Advances in stem cell therapy and gene editing are at the forefront of CKD research, offering potential treatments that could restore kidney function or even reverse damage. Stem cell therapy has demonstrated the ability to regenerate damaged kidney tissue and improve renal function in experimental models. Similarly, gene editing technologies such as CRISPR may provide targeted treatments for genetic forms of kidney disease, offering hope for patients with inherited conditions like polycystic kidney disease. As research in these areas progresses, the development of new treatments holds the potential to transform CKD management. The combination of early detection, better management of risk factors, and innovative therapies offers hope for slowing disease progression, improving the quality of life, and reducing the global burden of CKD.

8. References

1. Zehravi M, Maqbool M, Ara I. Polycystic ovary syndrome and infertility: an update. International journal of adolescent medicine and health. 2021 Jul 22;34(2):1-9.
2. Lihite RJ, Lahkar M, Das S, Hazarika D, Kotni M, Maqbool M, Phukan S. A study on adverse drug reactions in a tertiary care hospital of Northeast India. Alexandria journal of medicine. 2017 Jul 11;53(2):151-6.
3. Zehravi M, Maqbool M, Ara I. Correlation between obesity, gestational diabetes mellitus, and pregnancy outcomes: an overview. International Journal of Adolescent Medicine and Health. 2021 Jun 18;33(6):339-45.
4. Maqbool M, Bekele F, Fekadu G. Treatment strategies against triple-negative breast cancer: an updated review. Breast Cancer: Targets and Therapy. 2022 Jan 11:15-24
5. Rasool S, Maqbool M. An overview about Hedychium spicatum: a review. Journal of Drug Delivery and Therapeutics. 2019 Feb 15;9(1-s):476-80.
6. Zehravi M, Maqbool M, Ara I. Depression and anxiety in women with polycystic ovarian syndrome: a literature survey. International Journal of Adolescent Medicine and Health. 2021 Aug 23;33(6):367-73.
7. Maqbool M, Gani I, Dar MA. Anti-diabetic effects of some medicinal plants in experimental animals: a review. Asian Journal of Pharmaceutical Research and Development. 2019 Feb 15;7(1):66-9.
8. Zehravi M, Maqbool M, Ara I. Polycystic ovary syndrome and reproductive health of women: a curious association. International journal of adolescent medicine and health. 2021 Apr 21;33(6):333-7.
9. Mohd M, Maqbool M, Dar MA, Mushtaq I. Polycystic ovary syndrome, a modern epidemic: an overview. Journal of Drug Delivery and Therapeutics. 2019 May 15;9(3):641-4.
10. Maqbool M, Fekadu G, Jiang X, Bekele F, Tolossa T, Turi E, Fetensa G, Fanta K. An up to date on clinical prospects and management of osteoarthritis. Annals of Medicine and Surgery. 2021 Dec 1;72:103077.
11. Majeed A, Bashir R, Farooq S, Maqbool M. Preparation, characterization and applications of nanoemulsions: An insight. Journal of Drug Delivery and Therapeutics. 2019 Mar 15;9(2):520-7.
12. Zehravi M, Maqbool M, Ara I. Healthy lifestyle and dietary approaches to treating polycystic ovary syndrome: a review. Open Health. 2022 May 2;3(1):60-5.
13. Maqbool R, Maqbool M, Zehravi M, Ara I. Menstrual distress in females of reproductive age: a literature review. International journal of adolescent medicine and health. 2021 Jul 22;34(2):11-7.
14. Ara I, Maqbool M, Fekadu G, Hajam TA, Dar MA. Pharmaceutical significance of Nigella sativa L., a wonder herb. Journal of Applied Pharmaceutical Sciences and Research. 2020;3(4):04-13.

15. Maqbool M, Nasir N, Mustafa S. Polycystic in ovarian syndrome and its various treatment strategies. INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES. 2018 Sep 1;5(9):8470-8.
16. Maqbool M, Zehravi M, Maqbool R, Ara I. Study of adverse drug reactions in pulmonary medicine department of a Tertiary care hospital, Srinagar, Jammu & Kashmir, India. CELLMED. 2021;11(2):8-1.
17. Ara I, Maqbool M, Bukhari B, Ara N, Hajam TA. Present status, standardization and safety issues with herbal drugs. International Journal of Research in Pharmaceutical Sciences and Technology. 2020 May 18;1(3):95-101.
18. Ara I, Maqbool M, Gani I. Reproductive Health of Women: implications and attributes. International Journal of Current Research in Physiology and Pharmacology. 2022 Nov 28:8-18.
19. Zehravi M, Maqbool R, Maqbool M, Ara I. To Identify Patterns of Drug Usage among Patients Who Seek Care in Psychiatry Outpatient Department of a Tertiary Care Hospital in Srinagar, Jammu and Kashmir, India. Journal of Pharmaceutical Research International. 2021 Jun 10;33(31A):135-40.
20. Maqbool M, Javed S, Bajwa AA. Assessment OF pain management IN postoperative cases using different scales and questionnaires. INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES. 2019 Jan 1;6(1):983-7.
21. Ara I, Maqbool M, Zehravi M. Psychic consequences of infertility on couples: A short commentary. Open Health. 2022 Jan 1;3(1):114-9.
22. Bashir R, Maqbool M, Ara I, Zehravi M. An In sight into Novel Drug Delivery System: In Situ Gels. CELLMED. 2021;11(1):6-1.
23. Zehravi M, Maqbool M, Ara I. Teenage menstrual dysfunction: an overview. International Journal of Adolescent Medicine and Health. 2022 Sep 19;35(1):15-9.
24. Ara I, Zehravi M, Maqbool M, Gani I. A review of recent developments and future challenges in the implementation of universal health coverage policy framework in some countries. Journal of Pharmaceutical Research & Reports. SRC/JPRSR-131. DOI: doi. org/10.47363/JPRSR/2022 (3). 2022;127.
25. Maqbool M, Shabbir W, Aamir S. Adverse events of blood transfusion and blood safety in clinical practice. Indo American Journal Of Pharmaceutical Sciences. 2018 Aug 1;5(8):8254-9.
26. Maqbool M, Naeem A, Aamer S. Diabetes mellitus and its various management strategies in practice. Indo American Journal of Pharmaceutical Sciences. 2018 Aug 1;5(8):8163-+.
27. Maqbool M, Tariq S, Amjad S. Prescribing practices in pediatrics and drug utilization studies promoting pediatric health. Indo American Journal of Pharmaceutical Sciences. 2018 Aug 1;5(8):8070-6.
28. Maqbool M, Ikram U, Anwar A. Adverse drug reaction monitoring and occurrence in drugs used in pulmonary disorders. Indo American Journal Of Pharmaceutical Sciences. 2018 Aug 1;5(8):8060-5.
29. Maqbool R, Maqbool M, Zehravi M, Ara I. Acute neurological conditions during pregnancy and their management: a review. International Journal of Adolescent Medicine and Health. 2021 Aug 23;33(6):357-66.
30. Zehravi M, Maqbool M, Ara I. An overview about safety surveillance of adverse drug reactions and pharmacovigilance in India. The Indian Journal of Nutrition and Dietetics. 2021 Jul:408-18.
31. Maqbool M, Zehravi M. Neuroprotective role of polyphenols in treatment of neurological disorders: A review. Interventional Pain Medicine and Neuromodulation. 2021 Dec 31;1(1).

32. Maqbool M, Ara I, Gani I. The Story of Polycystic Ovarian Syndrome: A Challenging Disorder with Numerous Consequences for Females of Reproductive Age. International Journal of Current Research in Physiology and Pharmacology. 2022 Nov 28:19-31.

33. Maqbool M, Gani I. Utilization of statins in reducing comorbidities of diabetes mellitus: A systematic review. Journal of Pharmacy Practice and Community Medicine. 2018;4(4).

34. Maqbool R, Maqbool M, Zehravi M, Ara I. Acute neurological conditions during pregnancy and their management: a review. International Journal of Adolescent Medicine and Health. 2021 Aug 23;33(6):357-66.

35. Maqbool M, Tariq S, Amjad S. Prescribing practices in pediatrics and drug utilization studies promoting pediatric health. Indo American Journal of Pharmaceutical Sciences. 2018 Aug 1;5(8):8070-6.

36. Maqbool M, Naeem A, Aamer S. Diabetes mellitus and its various management strategies in practice. Indo American Journal of Pharmaceutical Sciences. 2018 Aug 1;5(8):8163-+.

37. Maqbool M, Shabbir W, Aamir S. Adverse events of blood transfusion and blood safety in clinical practice. Indo American Journal Of Pharmaceutical Sciences. 2018 Aug 1;5(8):8254-9.

38. Qadrie Z, Maqbool M, Dar MA, Qadir A. Navigating challenges and maximizing potential: Handling complications and constraints in minimally invasive surgery. Open Health. 2025 Feb 5;6(1):20250059.

39. Oral O, Maqbool M, Thapa P, Tatlibal P, Enser M. The potential impact of weight control management on metabolic health during healthy aging. Global Translational Medicine. 2025 Jan 13:4815.

40. Maqbool M, Oral O. Implications of hypothyroidism in females of reproductive age: a review of current literature.