

A REVIEW ON CKD MANAGEMENT: CURRENT STRATEGIES AND EMERGING THERAPIES

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ABSTRACT

Chronic kidney disease (CKD) is a long-term, irreversible ailment marked by a steady decline in kidney function, impacting millions worldwide and posing a significant public health issue. This review explores current management approaches and new therapeutic developments for CKD. The disease is identified by a glomerular filtration rate (GFR) below 60 mL/min per 1.73 m² or indicators of kidney damage persisting for a minimum of three months, with classification determined by GFR and albuminuria levels as per KDIGO guidelines. The development of CKD is characterized by a range of glomerular and tubulointerstitial disorders, such as chronic glomerulonephritis, diabetic nephropathy, and hypertension. As the condition progresses through its four main stages—from reduced renal reserve to end-stage renal disease

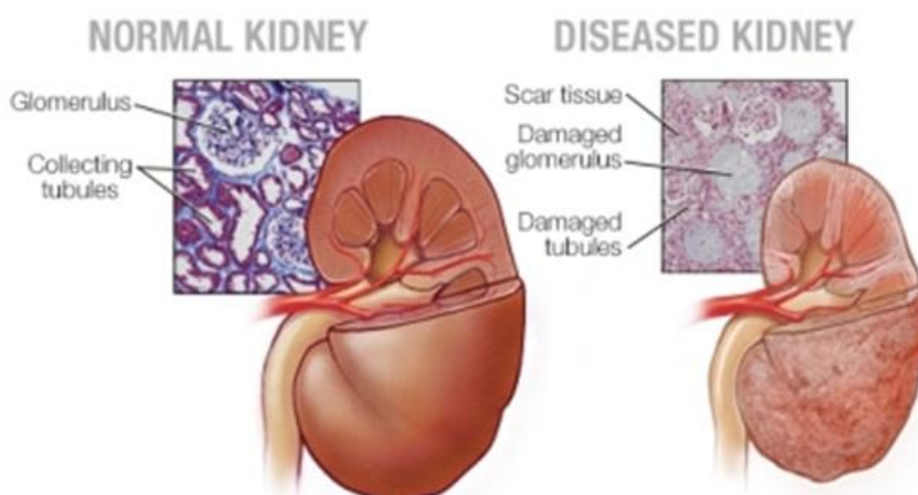
(ESRD)—patients encounter systemic issues like metabolic acidosis, hyperkalemia, anemia, and cardiovascular disease. Management aims to decelerate disease progression through non-drug approaches, such as limiting dietary sodium and protein, making lifestyle changes, and rigorously controlling glucose and blood pressure levels. For individuals advancing to ESRD, renal replacement options like hemodialysis, peritoneal dialysis, and kidney transplants are essential, with transplantation providing the most favorable long-term survival and quality of life. Additionally, the review emphasizes new regenerative treatments, particularly the use of

pluripotent stem cells (ESCs and iPSCs) to potentially slow disease progression through paracrine effects, although issues like tumor risks and long-term stability are still being actively researched.

INTRODUCTION

Chronic kidney disease (CKD) is a condition that progressively worsens, with kidney function declining regardless of the original cause of damage or external influences like infections, inflammation, or toxins. This disease poses a major public health challenge, impacting millions globally and resulting in higher rates of illness and death. CKD is marked by a slow deterioration of kidney function over time, which can ultimately progress to end-stage renal disease (ESRD), necessitating dialysis or a kidney transplant.^[1]

Chronic kidney disease (CKD) develops through various diverse disease mechanisms that lead to irreversible changes in the kidney's function and structure over an extended period, ranging from months to years. Diagnosing CKD involves confirming a persistent decline in kidney function along with structural damage to the kidneys. The most reliable measure of overall kidney function is the glomerular filtration rate (GFR), which represents the total volume of fluid filtered by all functioning nephrons within a given time frame.^[2]



Chronic kidney disease is generally characterized by a decline in kidney function, indicated by an estimated glomerular filtration rate (eGFR) below 60 mL/min per 1.73 m², or by signs of kidney damage such as albuminuria, hematuria, or abnormalities found through laboratory tests or imaging, persisting for a minimum of 3 months.^[3]

The 2012 KDIGO guidelines for CKD classification suggest identifying the cause of CKD and categorize the condition into six groups based on GFR, ranging from G1 to G5, with G3 further divided into 3a and 3b. Additionally, the guidelines incorporate staging based on three levels of albuminuria (A1, A2, and A3), with each CKD stage further divided according to the urinary albumin-creatinine ratio (ACR; mg/g or mg/mmol) measured in an early morning "spot" urine sample.

The 6 CKD categories, known as stages 1 through 5, are described below (stage 3 is separated into 3a and 3b):

1. G1: GFR 90 mL/min/1.73 m² and above with evidence of kidney disease, such as hematuria or proteinuria
2. G2: GFR 60 to 89 mL/min/1.73 m²
3. G3a: GFR 45 to 59 mL/min/1.73 m²
4. G3b: GFR 30 to 44 mL/min/1.73 m²
5. G4: GFR 15 to 29 mL/min/1.73 m²
6. G5: GFR less than 15 mL/min/1.73 m² or treatment by dialysis

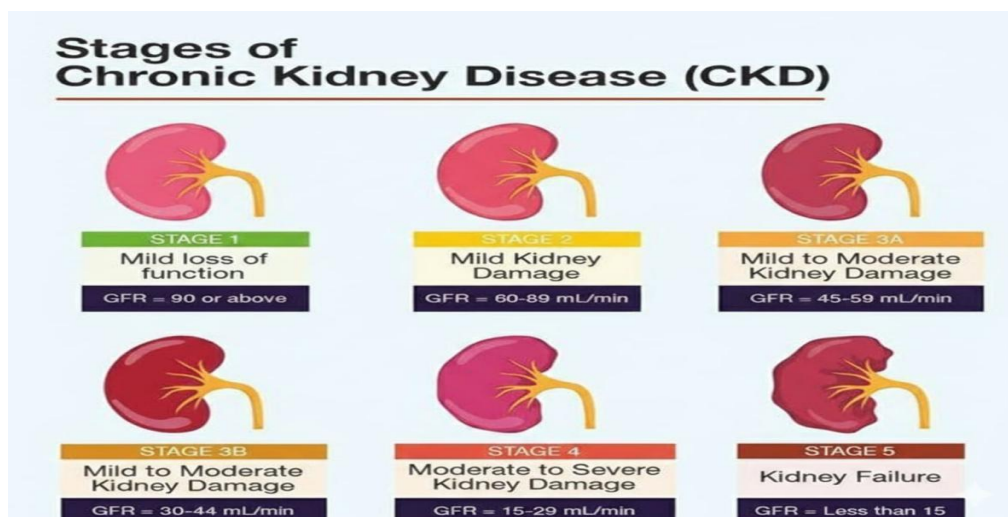
The 3 levels of albuminuria include an ACR:

1. A1: ACR less than 30 mg/g (<3.4 mg/mmol)
2. A2: ACR 30 to 299 mg/g (3.4-34 mg/mmol)
3. A3: ACR greater than 300 mg/g (>34 mg/mmol)

The enhanced classification of CKD has proven useful in pinpointing prognostic markers associated with reduced kidney function and elevated albuminuria. Nonetheless, a drawback of this classification system is the risk of overdiagnosing CKD, especially among older adults.^[4]

Table 0.1: KDIGO classification of the stages of chronic kidney disease.

Stage	GFR (ml/min/1.73m ²)	Terms
1	≥90	Normal or high
2	60-89	Mildly decreased
3a	45-59	Mildly to moderately decreased
3b	30-44	Moderately to severely decreased
4	15-29	Severely decreased
5	<15	Kidney failure



Etiopathogenesis

All chronic kidney diseases have the potential to progress to chronic renal failure (CRF). These diseases can typically be divided into two primary categories: those that result in glomerular damage and those that lead to tubulointerstitial damage. While this classification aids in research, the disease seldom remains restricted to just the glomeruli or the tubulointerstitial tissue. Ultimately, in the advanced stage of CRF, every part of the nephron is affected.

1. A variety of glomerular diseases associated with chronic renal failure (CRF) have their origins in immune mechanisms. The damage to the glomeruli disrupts the filtration process, resulting in nephrotic syndrome, which is characterized by proteinuria, hypoalbuminemia, and edema. Chronic glomerular diseases that lead to CRF are divided into two categories: primary and systemic.

i) Primary glomerular disease Chronic renal failure is primarily caused by chronic glomerulonephritis, which often begins with various forms of glomerulonephritis, including membranous glomerulonephritis, membranoproliferative glomerulonephritis, and anti-glomerular basement membrane nephritis.

ii) Systemic glomerular pathology refers to conditions that originate outside the kidneys but subsequently cause alterations in the nephrons. Notable examples include lupus nephritis, serum sickness nephritis, and diabetic nephropathy.

2. Diseases that affect tubulointerstitial tissues lead to changes in the absorption and secretion of vital substances, resulting in the production of large amounts of dilute urine.

Tubulointerstitial diseases can be classified into four categories based on their initial cause: vascular, infectious, toxic, and obstructive.

i. Vascular factors Chronic primary or essential hypertension leads to particular changes in the renal arteries and arterioles, referred to as nephrosclerosis (page 688). This condition causes a progressive obstruction of renal blood vessels, eventually leading to ischemia and tissue necrosis in the kidneys.

ii. Some toxic substances can lead to gradual damage to the tubules, ultimately resulting in chronic renal failure (CRF). A prevalent instance of this is the consumption of large amounts of pain relievers like phenacetin, aspirin, and acetaminophen, which can cause chronic analgesic nephritis. Additionally, prolonged exposure to substances such as lead, cadmium, and uranium can also result in CRF.

iii. Chronic obstruction in the urinary tract results in ongoing nephron damage due to fluid backpressure. Examples of such chronic issues include stones, blood clots, tumors, strictures, and an enlarged prostate.

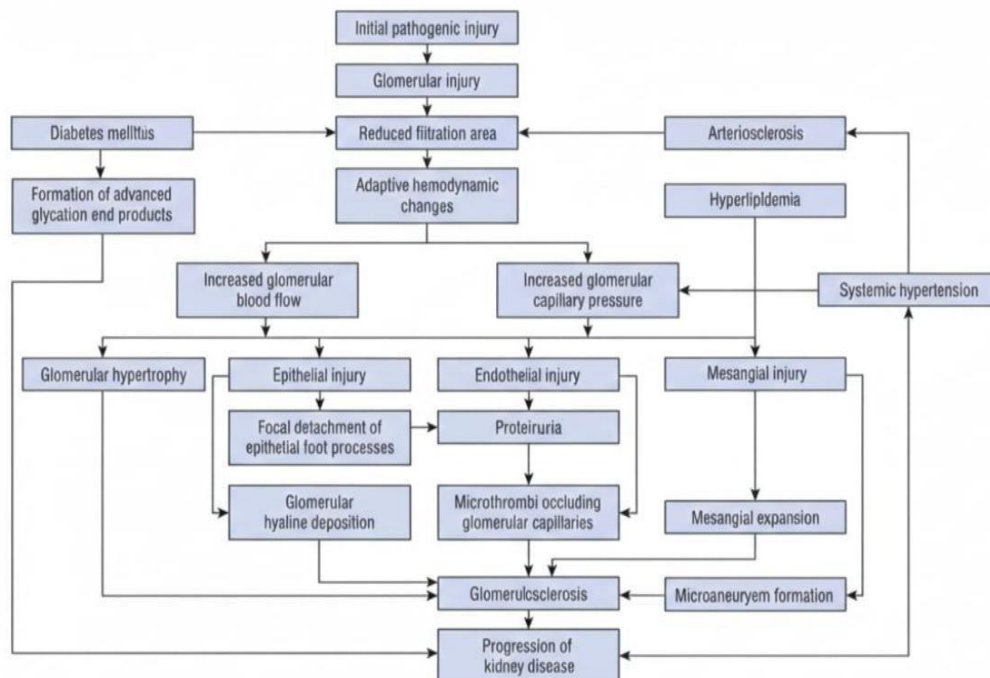


Fig 2.0 Proposed mechanism for progression of kidney disease.

Regardless of the initiating cause, CRF evolves progressively through 4 stages**✓ Decreased renal reserve**

At this point, the damage to the kidney tissue is minimal, and the kidneys continue to function properly. The glomerular filtration rate (GFR) is approximately 50% of the normal level, while blood urea nitrogen (BUN) and creatinine levels remain within normal ranges. Patients typically do not exhibit symptoms unless they are under stress.

✓ Renal insufficiency

At this point, approximately 75% of the functional kidney tissue has been lost. The glomerular filtration rate (GFR) is reduced to about 25% of its normal level, and there is an increase in blood urea nitrogen (BUN) and serum creatinine levels. Tubulointerstitial damage leads to increased urination and nighttime urination. A sudden stressor can trigger uremic syndrome.

✓ Renal failure

At this point, around 90% of the kidney's functional tissue has been destroyed. The glomerular filtration rate (GFR) is roughly 10% of what is considered normal. The tubular cells are largely non-functional. Consequently, the ability to regulate sodium and water is impaired, leading to edema, metabolic acidosis, hypocalcemia, and the signs and symptoms associated with uremia.

✓ End-stage kidney (chronic kidney disease)

At this stage, the GFR is reduced to less than 5% of its normal level, leading to a complicated clinical scenario of uraemic syndrome, characterized by worsening primary (renal) and secondary systemic (extra-renal) symptoms.^[5]

Some common symptoms and signs at these stages of CKD include

- Nausea
- Vomiting
- Loss of appetite
- Fatigue and weakness
- Sleep disturbance
- Oliguria
- Decreased mental sharpness
- Muscle cramps

- Swelling of feet and ankles
- Persistent pruritus
- Chest pain due to uremic pericarditis
- Shortness of breath due to pulmonary edema from fluid overload
- Hypertension.

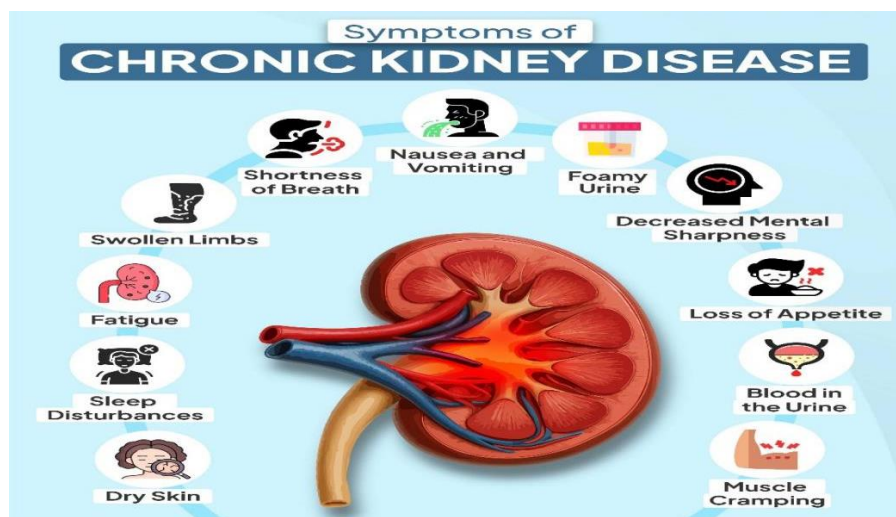
Differential Diagnosis

When assessing a patient for CKD, it is important to take into account other possible conditions that might exhibit similar symptoms and clinical signs. The differential diagnoses encompass:

1. Acute kidney injury
2. Alport syndrome
3. Antiglomerular basement membrane disease
4. Diabetic nephropathy
5. Multiple myeloma
6. Nephrolithiasis
7. Rapidly progressive glomerulonephritis
8. Renal artery stenosis

Physical examination is often not helpful, but patients may demonstrate the following symptoms

- Skin pigmentation
- Scratch marks from pruritus
- Pericardial friction rubs due to uremic pericarditis
- Uremic frost, where high levels of blood urea nitrogen (BUN) cause urea in sweat to crystallize into fine, white powder on the skin
- Hyperreflexia or muscle twitches
- Hypertensive fundal changes suggesting chronicity.^[6]



Non-pharmacological therapies in chronic kidney disease.

Table 1.0 Dietary and lifestyle modification for patients with CKD^[7]

Salt intake	❖ Lower salt intake to <90 mmol (<2 g) of sodium per day (corresponding to 5 g of sodium chloride) in adults, unless contraindicated. ❖ Restrict sodium intake in children with CKD who have hypertension (systolic and/or diastolic blood pressure >95th percentile) or prehypertension (systolic and/or diastolic blood pressure >90th percentile and <95th percentile), following the age-based recommended daily. Intake ❖ Supply free water and sodium to children with CKD and polyuria to avoid chronic intravascular depletion and to promote optimal growth
Protein intake	❖ Lower protein intake to 0.8 g/kg/day in adults with or without diabetes and GFR <30 mL/min/1.73 m² (G4-G5) ❖ Avoid high protein intake (>1.3 g/kg/day) in adults with CKD at risk of progression
Hyperuricemia	❖ Despite the relationship between hyperuricemia and incidence of CKD, there is insufficient evidence supporting or refuting the use of agents to lower serum uric acid concentrations in individuals with CKD and either symptomatic or asymptomatic hyperuricemia in order to delay CKD
Lifestyle	❖ Encourage patients with CKD to perform physical activity compatible with their cardiovascular health and tolerance (aiming for at least 30 min five times per week) ❖ Achieve a healthy weight (BMI 20-25, according to country-specific demographics) ❖ Stop smoking
Additional dietary advice	❖ Provide expert dietary advice and information in the context of an education program tailored to the severity of CKD and the need to intervene on salt, phosphate, potassium, and protein intake, when indicated

Treatment / Management

General Management

- Medication dosages need to be modified according to the patient's eGFR levels.
- When preparing for renal replacement therapy, it is important to arrange surgical consultations for the insertion of hemodialysis or peritoneal dialysis access and for transplantation if it is deemed suitable.

Treatment of Reversible Causes of Renal Failure

It is important to identify and manage potentially reversible causes of AKI, such as infections, medications, low blood pressure, and reduced blood volume. Before patients with CKD undergo intravenous contrast procedures, they should be thoroughly assessed, and alternative options should be prioritized. Furthermore, nephrotoxic substances, like aminoglycoside antibiotics and NSAIDs, should be avoided.

Slowing the Progression of Chronic Kidney Disease

It is important to address the factors that contribute to the advancement of CKD, such as high blood pressure, proteinuria, metabolic acidosis, and elevated lipid levels. Blood pressure should be controlled in line with established targets, and efforts should be made to decrease proteinuria to below 1 g/d if feasible.^[8]

Numerous studies have indicated that smoking is linked to a heightened risk of developing nephrosclerosis, and quitting smoking can slow the progression of CKD.^[9] Additionally, limiting protein intake has been found to decelerate CKD progression. Nonetheless, individuals with advanced CKD face the risk of malnutrition, highlighting the importance of dietician involvement.

Research has shown that using bicarbonate supplements to treat chronic metabolic acidosis can slow down the progression of CKD.^[10] Moreover, maintaining strict glucose levels in diabetic patients has proven effective in delaying the onset of albuminuria and preventing its progression to full-blown proteinuria.^[11]

Treatment of Reversible Causes of Renal Failure

Recognizing and addressing reversible causes of AKI, such as infections, medications, hypotension, and decreased blood volume, is crucial. Prior to intravenous contrast procedures in CKD patients, a comprehensive evaluation should be conducted, and alternative methods

should be considered. Additionally, nephrotoxic agents, including aminoglycoside antibiotics and NSAIDs, should be avoided.

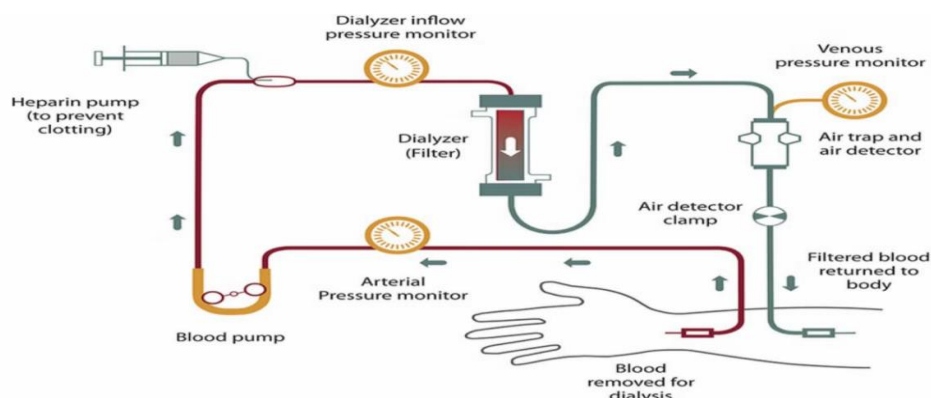
Slowing the Progression of Chronic Kidney Diseases

Addressing the elements that contribute to the progression of CKD is crucial, including factors like hypertension, proteinuria, metabolic acidosis, and high lipid levels. Blood pressure should be managed according to established guidelines, and efforts should be made to reduce proteinuria to less than 1 g/d if possible.^[8]

Research has consistently shown that smoking is associated with an increased risk of nephrosclerosis, and giving up smoking can help slow the progression of CKD.^[9] Furthermore, reducing protein consumption has been demonstrated to slow down the advancement of CKD. However, those with severe CKD are at risk of malnutrition, underscoring the need for dietician involvement.

Studies have indicated that bicarbonate supplements can be effective in slowing the advancement of chronic metabolic acidosis in CKD patients.^[10] Additionally, keeping glucose levels tightly controlled in individuals with diabetes has been shown to delay the onset of albuminuria and stop it from progressing to full proteinuria.^[11]

Dialysis replicates two essential kidney functions: the elimination of solutes and the removal of fluids. In hemodialysis, solutes are primarily removed through a process known as diffusion, where they passively move from the blood compartment to the dialysate compartment across a semipermeable membrane. The diffusion rate is influenced by various factors, including the molecular weight of the solutes, the permeability of the membrane, the rate of blood flow, the concentration gradient of solutes between the blood and dialysate, and the flow rate.



The Haemodialysis machine has three main components

- (1) the dialyzer (i.e., dialysis membrane).
- (2) a pump that regulates blood flow.
- (3) a dialysate solution delivery system at a constant rate up to approximately 500 mL/min.

Moreover, the machine is equipped with numerous safety mechanisms to guarantee the correct, secure, and dependable delivery of blood and dialysate to the filter, where the exchange of water and solutes occurs. These mechanisms include monitors to ensure that the pressures within the extracorporeal circuit remain within safe limits, a detector to identify any leakage of red blood cells from the blood compartment into the dialysate compartment, an air detector and shut-off device to prevent air embolism, a pump for delivering dialysate, a proportioning system to accurately dilute the dialysate concentrate, a heater to bring the dialysate to body temperature, an ultrafiltration controller for precise fluid removal regulation, and conductivity monitors to assess the ionic strength of the dialysate.

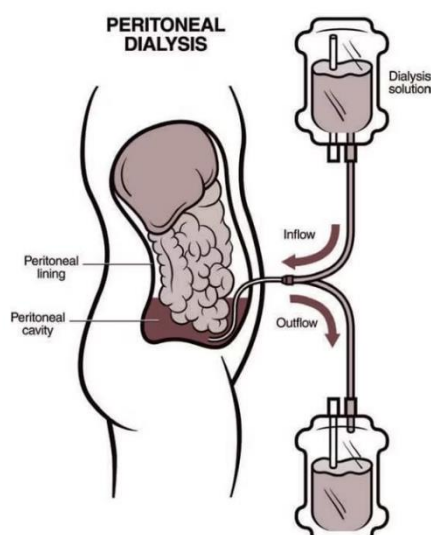
In most situations, the removal of solutes and fluids happens at the same time. However, when aggressive ultrafiltration is performed during standard hemodialysis, patients often experience symptoms like nausea, muscle cramps, and vomiting. Additionally, systemic vascular resistance might drop, leading to hypotension. By removing fluid alone without solute removal, osmotic changes are minimized. Conducting ultrafiltration separately can result in improved hemodynamic stability.

The elimination rate of smaller molecules is higher. Diffusion occurs more swiftly when there is a larger concentration gradient between the blood and the dialysate. The permeability of the membrane is influenced by specific characteristics, including pore size, charge, and quaternary conformation. Increased flow rates enhance solute removal, and the countercurrent flow of dialysate relative to blood flow optimizes the gradient across the dialysis membrane.

Peritoneal dialysis

Peritoneal dialysis (PD) is a gradual and ongoing process compared to hemodialysis, which helps lower the risk of heart-related issues and preserves remaining kidney function. Patients can perform PD at home, offering them a sense of autonomy and independence. Additionally, dietary restrictions on salt, potassium, protein, and fluids are less stringent with PD. For children with end-stage renal disease (ESRD), PD is preferred as it avoids frequent needle

insertions into blood vessels and, crucially, supports growth. Despite these benefits, there is still debate over PD's outcomes compared to hemodialysis. Depending on the study design and statistical methods used, PD has been associated with worse, similar, or better mortality rates than hemodialysis.



In PD treatment, the peritoneal membrane serves as the dialyzing surface. Consequently, it is important to regularly assess the function of the peritoneal membrane to prevent inadequate dialysis. PD should be considered the primary treatment option for all patients undergoing preemptive kidney transplantation and other home dialysis therapies, unless there is a significant contraindication.^[13]

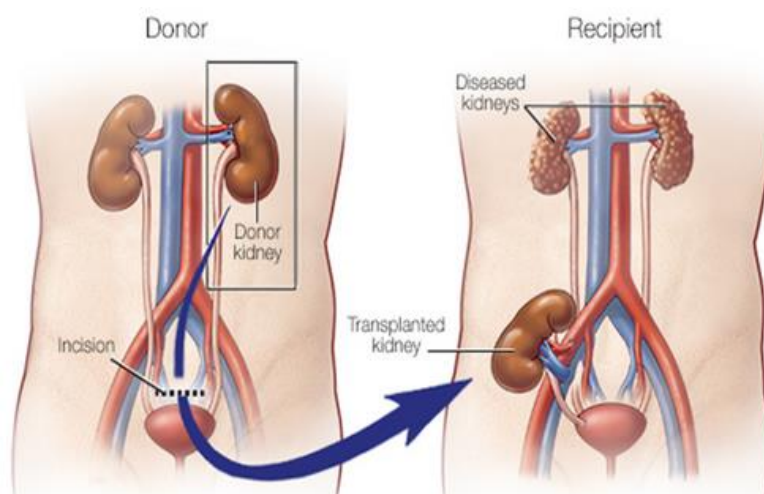
Renal Replacement Therapy

Patients can experience an optimal quality of life when a successful kidney transplant is carried out. Following the procedure, the restoration of normal kidney function allows them to be free from dietary and fluid limitations, enabling them to work and travel without restrictions, and addressing metabolic imbalances and anemia. Additionally, the long-term survival rate for kidney transplant recipients surpasses that of individuals undergoing hemodialysis.

Although kidney survival rates have significantly improved in recent times, post-transplant complications and long-term survival remain significant challenges for surgeons and nephrologists. Numerous factors affect the prognosis of transplantation, including ages of donor and recipient, race and sex, major histocompatibility complex (MHC), presensitization to human leukocyte antigen (HLA), kidney disease of recipient, condition and complication

before transplantation, compliance of recipient, cold ischemia time, number of nephrons of donor, and immunosuppressive strategy.

Identifying suitable candidates is crucial for kidney transplantation. Patients who wish to undergo transplantation should be listed as soon as dialysis begins. To ensure patients receive the highest quality and longest duration of life, it is vital to evaluate candidates early and, if feasible, proceed with preemptive transplantation when a living donor is available.^[14,15]



Complications

Systemic Complications of Chronic Kidney Disease

1. Imbalances in salt and fluid are frequently observed in CKD, particularly becoming more noticeable in stages 4 and 5.
2. Hypertension: In cases of CKD, hypertension can result from volume expansion, even though patients with CKD may not always exhibit edema as an indication of this expansion.^[16]
3. Hyperkalemia: In patients with CKD, hyperkalemia can develop, especially in those who are oliguric or have dysfunction in the distal renal tubules.
4. Metabolic acidosis is frequently observed in individuals with advanced CKD, as it results from the accumulation of acidic substances.
5. Hyperphosphatemia: This condition frequently arises in CKD patients because of a reduced phosphorous filtration rate.^[17]
6. Anemia: Anemia associated with CKD is generally normocytic and normochromic, arising from decreased erythropoietin production due to a reduction in functional kidney mass, irregular iron metabolism, and shorter red blood cell lifespan. Hemoglobin levels

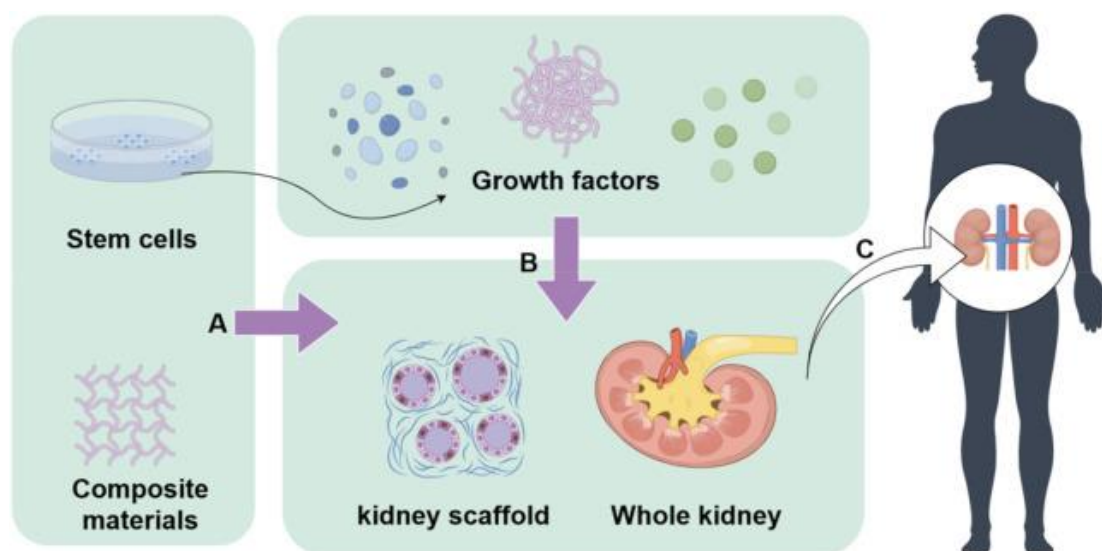
should be monitored annually in stage 3 CKD, every six months in stages 4 and 5, and on a monthly basis for patients undergoing dialysis.

7. Cardiovascular disease: The likelihood of developing CVD rises with the progression of CKD. Substantial evidence demonstrates a notable link between the thickness of epicardial adipose tissue and the occurrence of CVD events in individuals with CKD.^[18]
8. Complications of Renal Transplantation Posttransplant patients frequently experience infectious complications, making it essential to administer suitable prophylaxis and vaccinations. Additionally, other typical complications of CKD include hypertension, dyslipidemia, coronary artery disease due to new-onset diabetes mellitus, arrhythmias, and heart failure.^[19,20]

Current strategies

Pluripotent Stem cell Therapy in Experimental Chronic Kidney Disease

The potential of pluripotent stem cells in treating CKD is promising. One study found that when ESCs were transplanted near the damaged kidneys of rats that had undergone 5/6 nephrectomy, the progression of the disease was slowed due to the release of paracrine factors.^[21] In a similar model, another study noted that injecting undifferentiated iPSCs into the renal parenchyma improved kidney damage but also led to the development of Wilms' tumors.^[22]



When mesenchymal stem cells (MSCs) derived from induced pluripotent stem cells (iPSCs) were administered to mice with nephropathy caused by adriamycin (ADR), they helped preserve kidney function and exhibited anti-apoptotic effects on tubular cells as well as anti-

fibrotic properties.^[23] Our group's unpublished research revealed that renal progenitor cells (RPCs) derived from iPSCs, which had previously demonstrated efficacy in a cisplatin-induced acute kidney injury (AKI) model, also provided renal protection in an ADR nephropathy model when administered repeatedly. Although these iPSC-derived RPCs did not improve proteinuria or glomerulosclerosis, 14 days post-ADR administration, they did mitigate podocyte loss, decreased the number of glomeruli with synechiae, and reduced vascular rarefaction. Despite displaying a renal progenitor phenotype, we did not observe any renal engraftment or direct integration into glomerular and tubular structures, indicating that the beneficial effects of iPSC-derived RPCs were likely due to a paracrine influence on renal tissues.

Preliminary research indicates that enhancing the impact of iPSC-derived cell therapy is crucial for achieving significant clinical results. This enhancement might be accomplished by pinpointing iPSC-derived cells that exhibit the characteristics of glomerular precursor cells, which could then be evaluated in various CKD models to assess their long-term effectiveness and stability. Current investigations are centered on determining if iPSCs, sourced from individuals with chronic kidney diseases, could serve as an effective means for generating renal precursors capable of efficiently regenerating their own kidneys.^[24,25]

CONCLUSION

In conclusion, chronic kidney disease (CKD) poses a major global health issue, marked by the permanent deterioration of kidney function and structure. Treatment approaches emphasize identifying the root causes—whether they are glomerular or tubulointerstitial—and tackling reversible elements like infections or exposure to nephrotoxic drugs. Key to decelerating the disease's progression are non-pharmacological measures, such as reducing dietary salt and protein intake, managing weight, and quitting smoking, in addition to pharmacological management of hypertension and proteinuria.

As individuals progress to more advanced stages of the disease, it becomes crucial to prepare for renal replacement therapy. Hemodialysis and peritoneal dialysis serve as vital life-sustaining treatments, yet kidney transplantation is the ultimate solution for achieving optimal quality of life and long-term survival. Looking ahead, innovative therapies such as pluripotent stem cell research offer a promising experimental path for potentially regenerating kidney tissue and halting disease progression, although significant obstacles like the risk of tumor development need to be addressed. Ultimately, early detection and a multidisciplinary

management approach are essential for enhancing patient outcomes and minimizing the systemic complications associated with this progressive condition.

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