Review Article

Kidney Damage: A Comprehensive Review

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ABSTRACT

Kidneys are the filtering unit of body, filters extra water, wastes and toxins from blood and is removes them in the form of urine. Kidney injury refers to inefficiency of kidneys to filter the waste from the body. When kidneys fail to perform their normal function, toxins accumulate in the body that can lead to kidney failure, which can be fatal if untreated. Kidney injuries are of main two types based on their onset, Acute Kidney Injury (AKI) and Chronic Renal Failure (CRF). AKI occurs when the kidneys suddenly stops functioning properly, manifests as fluid retention causing swelling in lower extremities, decreased urine output although it remains occasionally normal, shortness of breath, fatigue, confusions, irregular heartbeat, etc. CKD occurs when the kidneys gradually stops functioning normally. Compared to CKD AKI complicates the course and worsens the condition of patients. In this review we provide the most recent updates in the definition, epidemiology, aetiology, risk factors, signs and symptoms, pathophysiology, diagnosis, staging, complications and treatment of AKI and CKD.

Keywords: Acute Kidney Injury (AKI), Chronic Kidney Disease (CKD), RIFLE criteria, AKIN criteria, KDIGO classification of CKD.

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INTRODUCTION

The term kidney or renal injury indicates the inability of the kidneys to perform its excretory function, leading to retention of nitrogenous waste products in the blood.

Roles of the kidney are as follows

- Excretion of nitrogenous waste (urea, amino acids and ammonia).
- Electrolyte and volume regulation.
- Synthesis of various hormones (Ex; erythropoietin, calcitonin, renin).
- Metabolism of low molecular weight proteins, such as insulin.
- Elimination of exogenous molecules like drugs.
- Regulates acid-base balance in the body.

Definitions

KDIGO (Kidney Disease Improving Global Outcomes) defines kidney injuries as following; [1]

- AKI: Increase in SCr by 50% within 7 days or increase in SCr by 0.3 mg/dl (26.5µmol/l) within 2 days, or oliguria with no structural damage.
- CKD: GFR <60 ml/min/1.73 m^2 for >3 months with kidney damage persisting for >3 months.
- AKD: AKI, or GFR <60 ml/min/1.73 m^2 for <3 months or decrease in GFR by ≥35% or increase in SCr by >50% for <3 months with kidney damage persisting for <3 months.

Acute Kidney Injury

ARF is a syndrome in which GFR declines very rapidly (hours-days) and is usually reversible.

According to the Kidney Disease Improving Global Outcome (KDIGO) criteria proposed in 2012, AKI was defined based on the following criteria's; [2]

- Increase in sCr level of 0.3 mg/dl in 48 hours
- Increase in sCr to 1.5 times to the baseline within last 7 days, or
- If the urine output is <0.5 ml/kg/hr for 6 hours.

Lately the term AKI has been replaced by ARF (Acute Renal Failure) or also called as ARD (Acute Renal Damage) because, AKI denoted the entire clinical spectrum from a mild increase in sCr levels to overt renal failure. [3]

Chronic kidney disease (CKD)

CKD or CRF (Chronic Renal Failure) is defined as a persistent impairment of kidney function, in other words, abnormally elevated sCr for >3 months or calculated GFR <60 ml/ min/ 1.73m^2.

It frequently involves a progressive loss of kidney function necessitating renal replacement therapy (dialysis or transplantation). When a patient needs renal replacement therapy, the condition is called ESRD (End-Stage Renal Disease). [2]

Etiology

Acute Kidney Injury [4]

- **Pre-renal causes:** accounts for approximately 60% of kidney damage cases

  Pre-renal causes include Volume contraction (Ex; sepsis, hemorrhage), Hypotension, Severe organ failure such as liver failure or heart failure, drugs like NSAIDs (non-steroidal anti-inflammatory drugs), Angiotensin Receptor Blockers (ARB) and Angiotensin-Converting Enzyme Inhibitors (ACEI), and Cyclosporine.

- **Intra-renal causes:** accounts for approximately 35% of kidney damage cases

  Intra-renal causes include Acute tubular necrosis (from prolonged prerenal failure, radiographic contrast material, drugs like aminoglycosides, or nephrotoxic substances), acute interstitial nephritis (drug induced), connective tissue disorders (vasculitis), fat embolus, arteriolar insults, rhabdomyolysis, intrarenal deposition (seen in tumor-lysis syndrome, multiple myeloma-Bence-Jones proteins and increased uric acid production).

- **Post-renal causes:** accounts for approximately 5% of kidney damage cases

  Post-renal causes include Intrinsic obstruction (due to calculus, tumor, clot, stricture), extrinsic compression (due to prostatic hypertrophy, carcinoma), decreased function (in conditions like neurogenic bladder).

Chronic Kidney Damage [5]

Etiological factors for chronic kidney damage include.

- Diabetes mellitus, especially type-2 DM, is the most frequent cause of ESRD
- Hypertension is the second most leading cause of CKD
- Polycystic kidney diseases
- Glomerulonephritis
- Recurrent kidney infections/pyelonephritis
- Renal vascular diseases
- Vesicoureteral reflux (VUR), a condition in which urine backups into the kidneys
- Other known causes like nephrolithiasis (Kidney stone), prolonged obstruction of the urinary tract
- Unknown etiology (idiopathic)

Epidemiology

The incidence of AKI has been cited as 1% on hospital admission, 2-5% during hospitalization, and in as many as 37% of cases treated in ICUs (intensive care units), and in 4-15% of patients after cardiovascular surgery. [6, 7, 8]

Overall, the incidence of AKI has been estimated to be 209 cases per million populations per year, with 36% of patients with AKI requiring dialysis or renal replacement therapy. [8]

The prevalence and incidence of CRF in the United States are uncertain. The third National Health and Nutrition Examination Survey (NHANES III) shows that nearly 2 million people in the United States have a sCr ≥2 mg/dl. [9]

CRF (Chronic renal failure) is known to be more common in men than in women. This gender disparity extends to end-stage renal disease (ESRD).

About 100,000 people develop ESRD every year in the United States. [8, 9]

Rates of ESRD vary with race. Both the incidence and prevalence of ESRD are 3-4 times higher in blacks than in whites. [9]

Risk Factors [10 - 13]

- Older age
- Prior exposure to potential nephrotoxins (Ex: nonsteroidal anti-inflammatory drugs [NSAIDs], lithium, angiotensin converting inhibitors, angiotensin-II receptor blockers, contrast, phosphate-based bowel preparations, herbal remedies containing aristolochic acid, antibiotics such as gentamicin, and chemotherapeutic agents)
- History of nephrolithiasis, recurrent urinary tract infections, obstruction of lower urinary tract, hypoalbuminemia, hyperuricemia
- Presence of comorbidities (Ex: hypertension, diabetes mellitus, COPD, chronic liver failure, metabolic syndromes like obesity, reduced kidney mass, previous episodes of acute kidney injury, shock, sepsis, anaemia, and autoimmune diseases)
- HIV infection
- Family history of kidney disease, and other known genetic risk factors like sickle cell trait, autosomal dominant polycystic kidney disease

Signs and Symptoms

Patient with Acute kidney injury may primarily present with decreased urine output (occasionally normal), fluid retention manifesting as swelling in lower extremities, shortness of breath, fatigue, confusion, nausea, weakness, irregular heartbeat, chest pain, seizures or coma in severe cases. Chronic kidney disease is generally identified by routine screening of serum chemistry profile and urine analysis. CKD patients may present with symptoms like

gross hematuria, foamy urine (indicating albuminuria), nocturia, flank pain, or decreased urine output. If CKD is advanced, patients may present with pallor, skin excoriations, asterixis, myoclonic jerks, altered mental status, pericardial rub, fatigue, poor appetite, nausea, vomiting, metallic taste, unintentional weight loss, pruritus, dyspnea, and peripheral edema. [11, 14]

Pathophysiology

Renal failure pathophysiology may be described by various sequences of events which takes place during acute insult or in short period in the setting of AKI and also gradually over a period in cases of CKD.

AKI can broadly be classified into 3 groups: [15]

- Prerenal azotemia (decrease in renal blood flow): Prerenal AKI occurs secondary to either an absolute reduction in extracellular fluid volume or a reduction in circulating volume despite a normal total fluid volume, Ex; in heart failure, advanced cirrhosis and sepsis. Normally kidney's auto-regulatory mechanism maintains intra-capillary pressure during initial phase by causing constriction of efferent arterioles and dilation of afferent arterioles. When prerenal conditions become severe, renal adaptive mechanisms fail to compensate unmasking the rapid fall in GFR and increase in BUN (Blood Urea Nitrogen) and sCr levels.
- Renal azotemia (Intrinsic renal parenchymal diseases): Intrinsic disorders can be sub-divided into those involving the vasculature, glomeruli or tubulointerstitium respectively.
- Post renal azotemia: obstruction of urine outflow or micturition.

The pathophysiology of CRF is mainly associated to specific initiating mechanisms. Over the course of time-adaptive physiology plays a role leading to compensatory hypertrophy and hyper filtration of remaining viable nephrons. As the renal damage continues, sub sequentially it leads to histopathologic changes which include distortion of glomerular architecture, abnormal podocyte function, and disruption of filtration and leads to sclerosis [16].

Diagnosis

The initial laboratory evaluation includes measurement of serum creatinine levels, Glomerular Filtration Rate (GFR) and urine output. [17] Based on widely accepted guidelines and criteria's kidney injury is defined as;

In 2002, Acute Dialysis Quality Initiative (ADQI) was formed with the primary intention of developing concordant and evidence-based guidelines for treatment and prevention of AKI. ADQI generated a uniformly accepted definition for AKI, RIFLE (Risk, Injury, Failure, Loss, End-stage kidney disease) criteria. RIFLE defines AKI based on variability of GFR and sCr levels as [18, 19, 20, 21] (Table 1);

Table 1: Rifle Criteria

Criteria	Category
Risk (R)	Decrease in GFR by >25% or increase in sCr level by 1.5 times
Injury (I)	Decrease in GFR by >50% or increase in sCr level by 2.0 times
Failure (F)	Decrease in GFR by >75% or increase in sCr level by 3.0 times
Loss (L)	Persistent renal failure or complete loss of kidney function for >4 weeks
End-stage kidney disease (E)	Complete loss of kidney function for >3 months

In September 2004, Acute Kidney Injury Network (AKIN) was formed. AKIN advised the term AKI to be used to represent full spectrum of renal injury, from mild to severe with end-stage kidney disease.

AKIN proposed the criteria to define AKI as [18, 19, 20, 21]

- Increase in Serum Creatinine (sCr) by ≥0.3 mg/dl (≥26.5 µmol/l) within 48hrs; or
- Increase in Serum Creatinine to ≥1.5 times baseline, within last 7days; or
- Urine volume <0.5 ml/kg/hr for 6hrs.

In 2012, KDIGO released a clinical guideline for AKI based on the previously proposed RIFLE and AKIN criteria's. KDIGO defines AKI as any of the following; [22]

- Increase in Serum Creatinine (sCr) by ≥0.3 mg/dl (≥26.5 µmol/l) within 48hrs; or
- Increase in Serum Creatinine to ≥1.5 times baseline, within last 7days; or
- Urine volume <0.5 ml/kg/hr for 6hrs

KDIGO has recommended a staging system for AKI severity[17, 22, 23] (Table 2).

Table 2: Staging the severity of AKI based on KIDIGO guidelines

Stage	Serum Creatinine (sCr)	Urine output
1	Increase in 1.5-1.9 times baseline Or sCr ≥0.3 mg/dl	<0.5 ml/kg/hr for 6hrs
2	Increase in 2-2.9 times baseline	<0.5 ml/kg/hr for 12hrs
3	Increase in 3 times baseline Or sCr ≥4mg/dl or Initiation of renal replacement therapy	<0.3 ml/kg/hr for 24hrs Or Anuria for ≥12hrs

KDIGO defines CKD as structural and functional abnormalities of renal system persisting for ≥43 months, with implications of health as one or more of the following

- GFR <60 ml/min/1.73 m^2
- Albuminuria (i.e., urine albumin ≥30 mg/day or urine albumin-to-creatinine ratio [ACR] ≥30 mg/g)
- Abnormalities in urine sedimentation, histology, or imaging studies suggests about kidney damage
- Renal tubular disorders; or
- History of kidney transplantation

Based on the National Kidney Foundation Developed (NKFD) criteria, as part of its Kidney Disease Outcomes Quality Initiative severity of CKD can be stratified as [24, 25, 26] (Table 3).

Table 3: Staging the severity of CKD

Stages	Description	e-GFR (ml/min/1.73m²)
1	Kidney damage with normal or ↑GFR	≥90
2	Kidney damage with mild ↓GFR	60-89
3a	Mild-moderate ↓GFR	59-45
3b	Moderate-severe ↓GFR	44-30
4	Severe ↓GFR	15-29
5	Kidney failure	<15 (or dialysis)
e-GFR (estimated Glomerular Filtration Rate)		

If the duration of kidney disease is unclear, repeated assessments are performed to differentiate CKD from acute kidney injury (i.e., change in kidney function occurring within 2–7 days) and acute kidney disease (kidney damage or decreased kidney function present for ≤3 months).

In certain circumstances, it is necessary to use adjunctive methods to diagnose kidney injury, especially when creatinine and urine output values cannot be interpreted accurately, often observed in critically ill patients with fluid overload, muscle wasting, sepsis, and reduced circulation may completely mask the diagnosis of AKI. New biomarkers have been identified to accomplish these situations; biomarkers for AKI can be stratified as markers reflecting [27, 28];

- Glomerular filtration (Ex: serum cystatin C)
- Glomerular integrity (Ex: albuminuria and proteinuria)
- Tubular stress (Ex: insulin-like growth factor binding protein 7 (IGFBP-7), Tissue inhibitor metalloproteinase 2 (TIMP2))
- Tubular damage (Ex: neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), N-acetyl-βD-glucosaminidase (NAG), Liver fatty acid-binding protein (L-FAB))
- Intra-renal inflammation (Ex: interleukin-18)

Other laboratory examinations include assessment of serum electrolyte concentrations, complete blood count, liver function tests, glucose level, bone profile, microscopic examination. Chest x-ray can be evident in finding the potential cause like pneumonia or vasculitis and is also useful in evaluation of volume overload condition. [3]

Imaging studies like ultrasound examination of kidneys, renal ultrasound Doppler, computerized tomography, renal angiography, voiding cystourethrography and renal scans are useful in establishing accurate diagnosis [29].

Treatment/Management [30]

Treatment choice for renal failure may vary and mainly depends on the cause of failure. Broadly the treatment choices are divided into 2 groups: treating the causes of kidney failure in acute states versus replacing the renal function in acute or chronic conditions. The summary of renal failure treatment is given below:

Acute Renal Failure

- The mainstay in the treatment of ARF is treating the underlying cause and associated complications.
- Patients with oliguria should have a fluid restriction of about 400 ml (unless if there is a signs of volume overload or depletion).
- In case of oliguria patients if overload and no volume are noted, a fluid challenge can be appropriate with the diligent monitoring for volume overload.
- In the case of hyperkalemia with ECG changes, glucose, sodium bicarbonate, and IV calcium, along with insulin should be given. And can be supplemented with the polystyrene sulfonate, which removes potassium from the body. Hemodialysis is an emergency method for the removal.
- If acidosis: Serum bicarbonate IV or oral, versus urgent/emergency dialysis based on the clinical situation
- If obstructive etiology present treat accordingly and or if bladder outlet obstruction secondary to prostatic hypertrophy patients may get benefited from Flomax or other selective alpha blockers.

General Measures

- First things, always first review the drug list.
- Stop the drugs which cause nephrotoxicity and renally adjust the dose of others. Many of the supplements have not been approved by the FDA and it can be nephrotoxic.

- Always record in's and out's.
- Monitor the weights daily
- Watch for the complications, such as pulmonary edema, hyperkalemia, and acidosis before starting the dialysis.
- Ensure good cardiac output and subsequent renal blood flow is maintained.
- Pay attention for the diet: total caloric intake should be 35-50 kcal/kg/day to avoid catabolism. Potassium intake is restricted to 40 mEq/day; phosphorus restricted to 800 mg/day. If it becomes high, and it should be treated with calcium carbonate or other phosphate binder. Magnesium compounds should be avoided.
- And treat the infections aggressively.

Immediate Dialysis Indications

- Acidosis
- Severe hyperkalemia
- Volume overload refractory to conservative therapy
- Encephalopathy
- Uremic pericarditis
- Alcohol and drug intoxications

Chronic Renal Failure

- Optimize control of the specific causes of CKD like diabetes mellitus and hypertension should be done.
- Measure sequentially and plot the rate of decline in GFR in all the patients.
- Rule out uncontrolled hypertension, extracellular fluid volume depletion, new obstructive uropathy, urinary tract infection, exposure to nephrotoxic agent (contrast dye or NSAIDs), reactivation or flare of the original disease like vasculitis or lupus.
- Interventions to slow the progression of CKD.
- Reduce intra-glomerular filtration.
- Reduce proteinuria; effective medications include ARB/ACE.
- Strict glycemic control should be followed.
- Prevent and treat CKD complications.
- Discuss renal replacement therapy (RRT) with the patients appropriately and start timely.
- Periodically review the medications and avoid the medications which cause nephrotoxicity. Renally excreted medications should be dosed appropriately.
- Patients with CKD should be referred to a nephrologist when GFR is <30 ml/min as this provides enough time for adequate preparation for kidney replacement therapy.

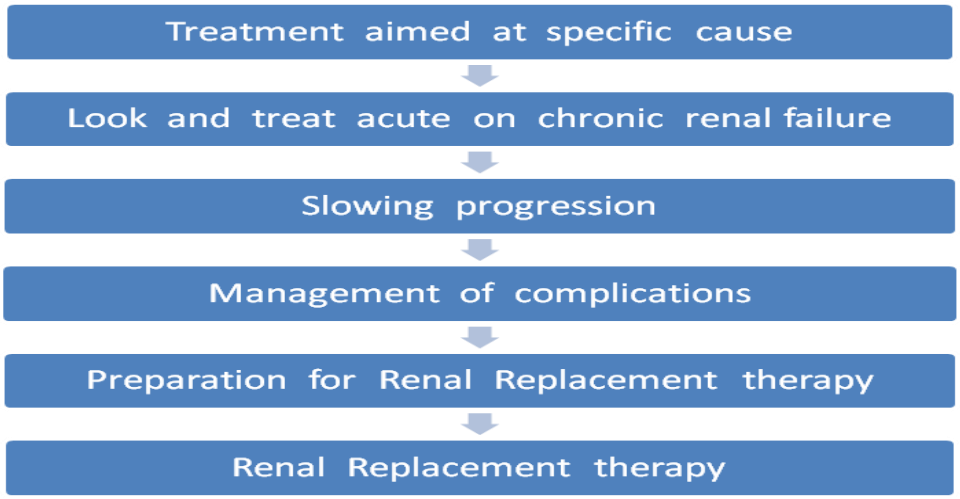

Figure 2: Management of CKD

Complications [30]

- Hyperkalemia
- Volume overload
- Acidosis
- Hyponatremia
- Anemia
- Calcium and phosphate imbalance

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