Renal failure

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kidneys

- The kidneys play a central role in excretion of many metabolic breakdown products, including ammonia and urea from protein, creatinine from muscle, uric acid from nucleic acids, drugs and toxins.
- They achieve this by making large volumes of an ultrafltrate of plasma (125mL/min, 180L/24 hrs) at the glomerulus, and selectively reabsorbing components of this ultrafltrate at points along the nephron
- Each kidney contains approximately 1 million individual functional units, called nephrons.
- Under normal circumstances, approximately 99% of the 180L of glomerular filtrate that is produced each day is reabsorbed in the tubules

Glomerular filtration rate

- The glomerular filtration rate (GFR) is the sum of the ultrafiltration rates from plasma into the Bowman's space in each nephron and is a measure of renal excretory function
- The normal range of GFR, is 100–130 average of 125 mL/min in men
- and 90–120 ml/min in women younger than the age of 40.

1.Acute kidney injury (AKI)

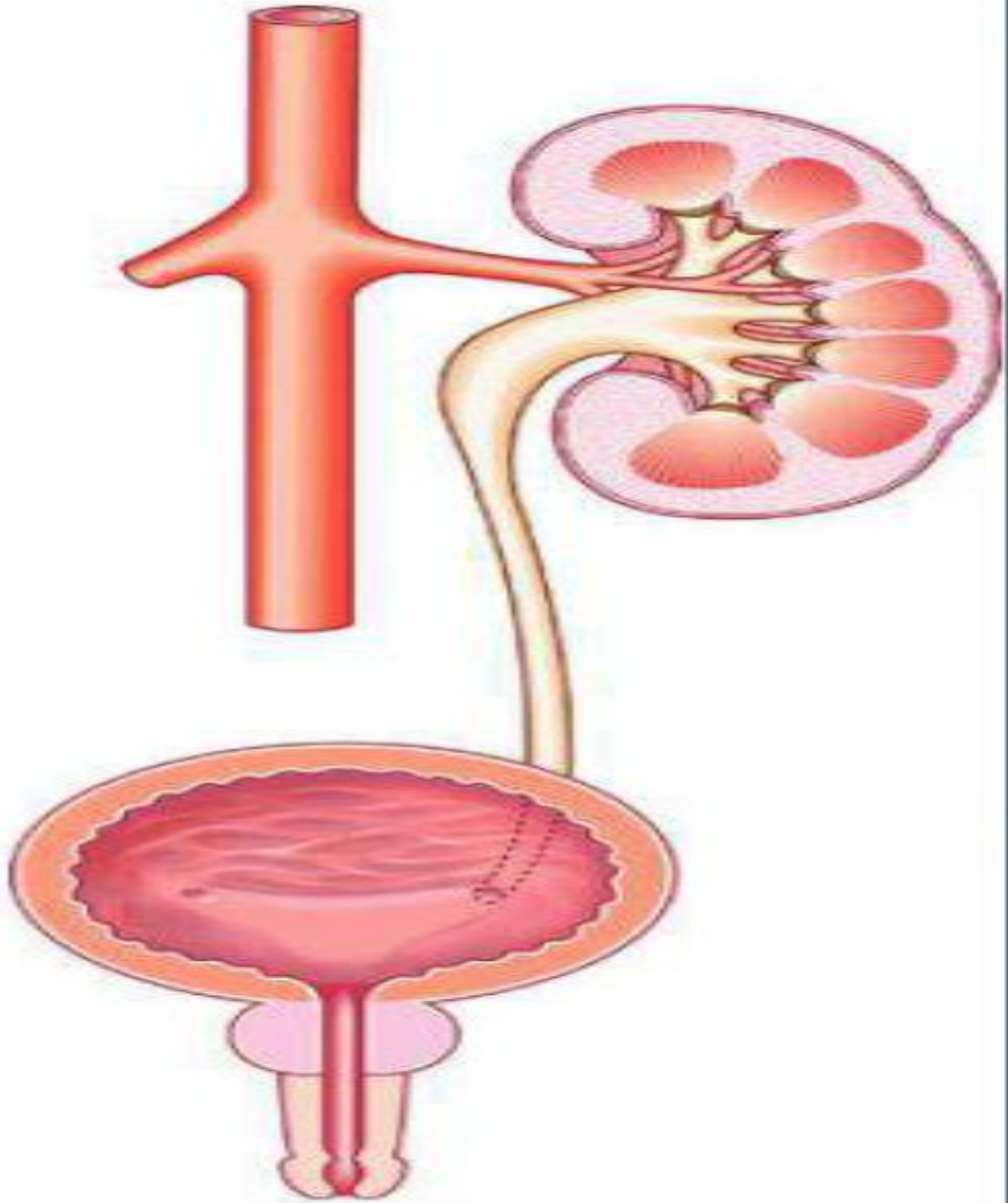
(AKI) is a sudden and often reversible loss of renal function, which develops over days or weeks and is often accompanied by a reduction in urine volume.

Approximately 7% of all hospitalized patients and 20% of acutely ill patients develop AKI

In uncomplicated AKI mortality is low, however when it is associated with sepsis and multiple organ failure mortality is 50%–70% and the outcome is usually determined by the severity of the underlying disorder and other complications, rather than by kidney injury itself

Pathophysiology

- ‘Pre-renal, when perfusion to the kidney is reduced
- ‘Renal’, when the primary insult affects the kidney itself
- ‘post-renal’, when there is an obstruction to urine flow at any point from the tubule to the urethra.



PRE-RENAL

Impaired perfusion:
- Cardiac failure
- Sepsis
- Blood loss
- Dehydration
- Vascular occlusion

RENAL

Glomerulonephritis
Small-vessel vasculitis
Acute tubular necrosis
- Drugs
- Toxins
- Prolonged hypotension

Interstitial nephritis
- Drugs
- Toxins
- Inflammatory disease
- Infection, including COVID-19

POST-RENAL

Urinary calculi (bilateral)
Retroperitoneal fibrosis
Benign prostatic hypertrophy
Bladder cancer
Prostate cancer
Cervical cancer
Urethral stricture/valves
Meatal stenosis/phimosis

Clinical features

- .
- Early recognition of AKI, is required to prevent rapid progression of renal injury and to facilitate recovery as the damage may be potentially reversible if detected at an early stage. **Pre renal**
- Low BP relative to normal for the patient (including postural drop)
- Tachycardia
- Weight decrease
- Dry mucous membranes
- Decreased skin turgor
- JVP not visible even when lying down.

- nausea or vomiting
- shortness of breath
- confusion • tiredness • reduction in urine output
- water retention

- Renal AKI
  - Vital signs , Fluid assessment, Oedema
  - Purpuric rash, uveitis, arthritis
  - Fever
  - Rash
- **Post renal**
  - Rectal examination (prostate and anal tone)
  - Distended bladder
  - Pelvic mass

Diagnosis

- **Pre-renal**
- Fractional excretion Na < 1%
- High serum urea: creatinine ratio
- Urinalysis

- **Renal**
- Proteinuria, haematuria
- Red cell casts, dysmorphic red cells
- Leucocyturia
- White cell casts
- Minimal proteinuria
- **Post-renal**
- Urinalysis frequently normal (may reveal haematuria depending on cause)
- Renal ultrasound (hydronephrosis

Management of acute kidney injury

- **Assess fluid status** as this will determine fluid prescription:
- If hypovolaemic: optimize systemic hemodynamic status with fluid
- Once euvolaemic, match fluid intake to urine output plus an additional 500mL/24 hrs to cover insensible losses
- If fluid-overloaded, prescribe diuretics (loop diuretics at high dose will often be required); if the response is unsatisfactory, dialysis may be required
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Cont.

- **Treat underlying cause**
- If K+ > 6.5 mmol/L and ECG changes of hyperkalaemia are present administer calcium gluconate to stabilise myocardium, lower potassium by oral potassium exchange resin to prevent potassium absorption, or administering intravenous glucose/insulin or sodium bicarbonate to move potassium intracellularly.
- These are holding measures until a definitive method of removing potassium is achieved (restoration of renal function or dialysis)

Cont.

- Discontinue potentially nephrotoxic drugs and reduce doses of therapeutic drugs according to level of renal function
- Ensure adequate nutritional support
- Consider proton pump inhibitors to reduce the risk of upper gastrointestinal bleeding
- Screen for intercurrent infections and treat promptly if present

Recovery from AKI

- Most cases of AKI will recover after the insult resolves but recovery may be impaired in those with pre-existing CKD or following a prolonged, severe or irreversible insult.
- Recovery is heralded by a gradual return of urine output and a steady reduction in serum creatinine.
- There is often a diuretic phase in which urine output increases rapidly and remains excessive for several days before returning to normal

2.Chronic kidney disease

- Chronic kidney disease (CKD) refers to an irreversible deterioration in renal function that usually develops over a period of years.
- Initially, it manifests only as a biochemical abnormality but, eventually, loss of the excretory, metabolic, and endocrine functions of the kidney leads to the clinical symptoms and signs of renal failure, collectively referred to as uremia.
- When death is likely without RRT (CKD stage 5), it is called end-stage renal disease (ESRD)

Epidemiology

- The social and economic consequences of CKD are considerable.
- In many countries, estimates of the prevalence of CKD stages 3–5 (eGFR <60 mL/min) are around 5%–7%, mostly affecting people aged 65 years and above.

- clinical features markers of CKD such as anemia, elevated PTH, and small kidneys observed on imaging

Common causes of chronic kidney disease

- Diabetes mellitus 20%–45% Large racial and geographical differences
- Interstitial diseases 20%–30% Drug-induced, reflux nephropathy
- Glomerular diseases 10%–20% IgA nephropathy is most common
- Hypertension 5%–20% Causality controversial, much may be secondary to another primary renal disease

Cont.

- Systemic infammatory diseases 5%–10% Systemic lupus erythematosus, vasculitis
- Renovascular disease 5% Mostly atheromatous, may be more common
- Congenital and inherited 10% Polycystic kidney disease,
- Unknown 5%–10

investigations in chronic kidney disease

Initial tests
Creatinine, eGFR
Urinalysis and quantification of proteinuria
Electrolytes
Calcium, phosphate, parathyroid hormone and 25(OH)D
Albumin
Full blood count (± Fe, ferritin, folate, B_{12})
Lipids, glucose ± HbA_{1c}
Renal ultrasound
Hepatitis and HIV serology
Other tests

Management

- The aims of management in CKD are to:
- **monitor renal function**
- Renal function should therefore be monitored every 6 months in patients with stage 3 CKD, but more frequently in patients who are deteriorating rapidly or have stage 4 or 5 CKD
- **prevent or slow further renal damage**
- Therapies directed towards the primary cause of CKD should be employed where possible; tight blood pressure control is applicable to CKD regardless of cause, however, and reducing proteinuria is a key target in those with glomerular disease.

Maintenance of fluid and electrolyte balance

- Urea is a key product of protein degradation and accumulates with progressive CKD.
- All patients with stages 4 and 5 CKD should be given dietetic advice aimed at preventing excessive consumption of protein.
- . Potassium often accumulates in patients with advanced CKD, who
- should be provided with dietary advice to reduce daily potassium intake to below 70 mmol
- Consideration should be given to stopping or reducing drugs that elevate potassium, such as potassium-sparing diuretics and ACE inhibitors/ARBs

Cont.

- **limit complications of renal failure**
- Anaemia is common in patients with CKD and contributes to many of the non-specific symptoms, including fatigue and shortness of breath.
- Once iron deficiency and other causes of anaemia have been excluded or corrected, recombinant human erythropoietin is very effective in correcting the anaemia of CKD and improving symptoms
- treat risk factors for cardiovascular disease
- prepare for RRT, if appropriate

Renal replacement therapy

- Renal replacement therapy (RRT) may be required on a temporary basis in patients with AKI or on a permanent basis for those with advanced CKD.
- Since the advent of long-term RRT in the 1960s, the number ofpatients with ESRD who are kept alive by dialysis and transplantation has increased considerably

1.Hemodialysis

- Haemodialysis is the most common form of dialysis employed in ESRD and is also used in AKI.
- Haemodialysis involves gaining access to the circulation, either through a central venous catheter or an arteriovenous

fistula or graft.

2.Peritoneal dialysis

- Peritoneal dialysis is principally used in the treatment of CKD, though it may occasionally be employed in AKI.
- It requires the insertion of a permanent Silastic catheter into the peritoneal cavity.

3. Renal transplantation

- Renal transplantation offers the best chance of long-term survival in ESRD and is the most cost-effective treatment.
- All patients with ESRD should be considered for transplantation but many are not suitable due to a combination of comorbidity and advanced age (although no absolute age limit applies).
- Active malignancy, vasculitis and cardiovascular comorbidity are common contraindications to transplantation, with risk of recurrence of the original renal disease.

Activity

- How do you deal with uraemic patients during anesthesia?

Thank you