**SNS COLLEGE OF ALLIED HEALTH SCIENCES**
SNS Kalvi Nagar, Coimbatore - 35
Affiliated to Dr MGR Medical University, Chennai

**DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE TECHNOLOGY**

**COURSE NAME: PATHOLOGY II**

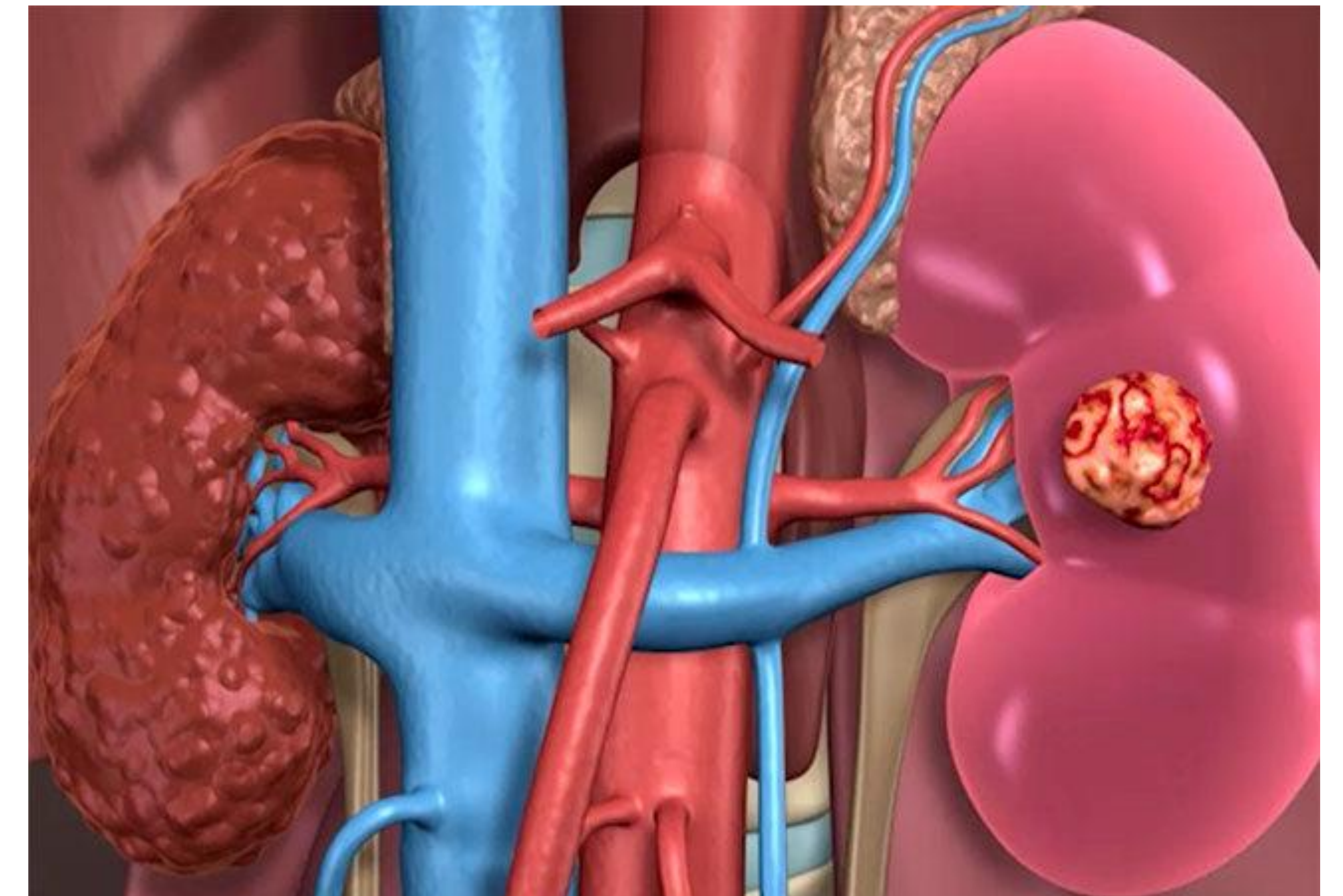
**II YEAR**

UNIT III : PATHOLOGY OF KIDNEY

TOPIC 3 : CHRONIC KIDNEY FAILURE (CKF)

# Chronic Kidney Failure

- Chronic renal failure is a syndrome characterised by progressive and irreversible deterioration of renal function
- It is due to slow destruction of renal parenchyma, eventually terminating in death when sufficient number of nephrons have been damaged.

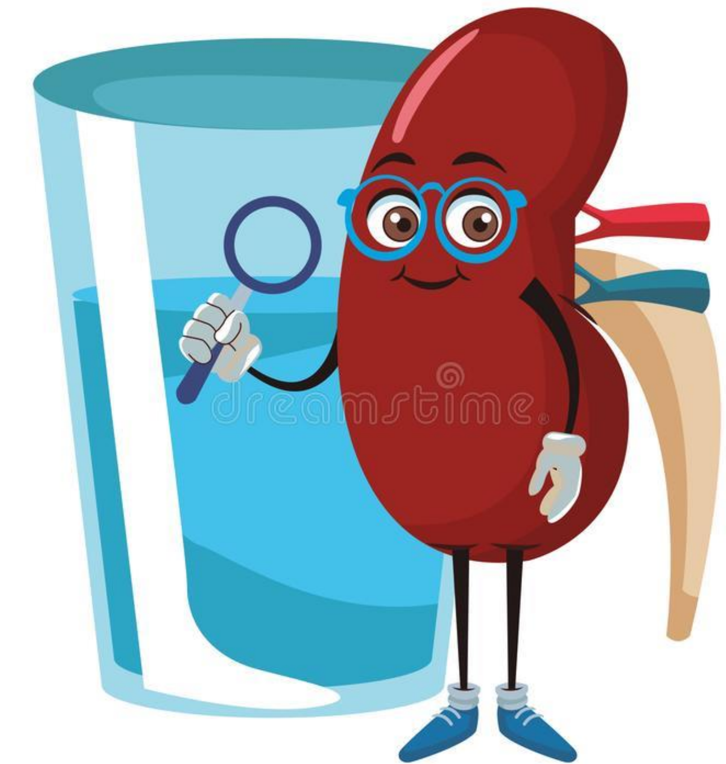


# Epidemiology

- According to Nephrology Dialysis Transplantation there are ~7.85 million CRF patients in India.

**Etiologically,**

- diabetes (41%)
- hypertension (22%)
- chronic glomerular nephritis (16%)
- chronic interstitial disease (5.4%)
- ischaemic nephropathy (5.4%)
- obstructive uropathy (2.7%)
- miscellaneous (2.7%) and unknown cause (5.4%) constituted the spectrum



# Etiopathogenesis

- All chronic nephropathies can lead to CRF.

Classification of CKF is of two major conditions

**Diseases causing glomerular pathology**

*Primary glomerular pathology* -- > chronic glomerulonephritis

*Systemic glomerular pathology*

- systemic lupus erythematosus
- Diabetic nephropathy

**Diseases causing tubulointerstitial pathology**

*Vascular* -- > hypertension

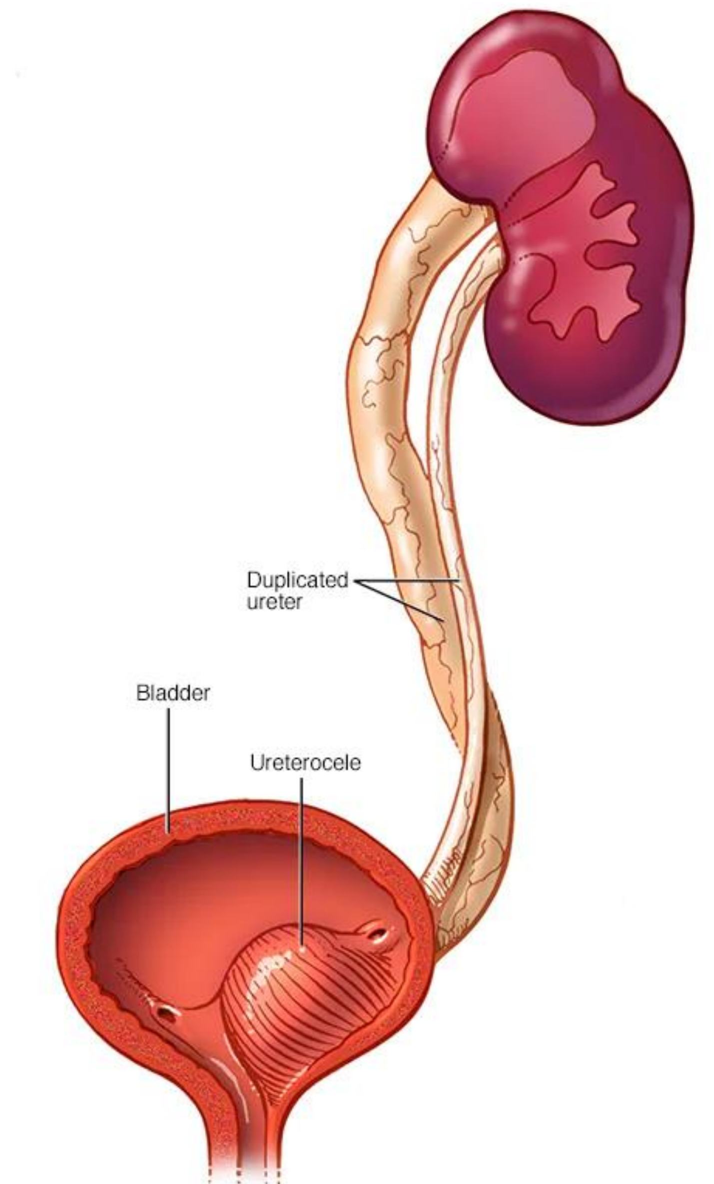
*Infectious* -- > chronic pyelonephritis

*Toxic* -- > intake of high doses of analgesics

*Obstructive* -- > stones, blood clots, tumours

# Leading Cause of CKD

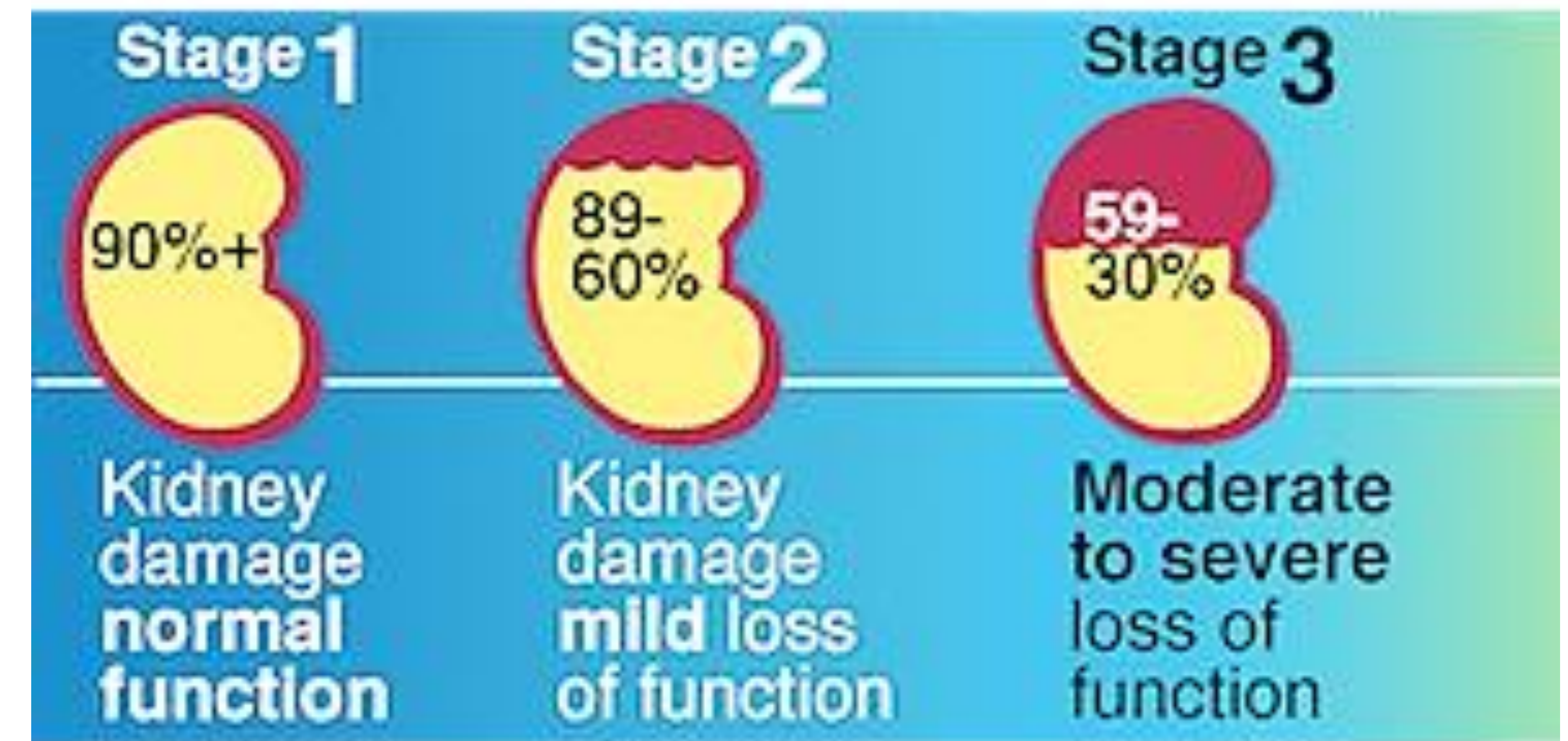
- Diabetes
- Hypertension
- Obstructed urine flow
- Kidney diseases
- Kidney artery stenosis
- Certain toxins - including fuels, lead etc.
- Fetal developmental problem
- Malaria and yellow fever
- Some medications - for example, NSAIDs
- Illegal substance abuse - such as heroin or cocaine.
- Injury - a sharp blow or physical injury to the kidney



# Stages of Chronic Renal Failure

***Decreased renal reserve***

- Minimal damage to renal parenchyma
- GFR is about 50%
- BUN and creatinine values are normal




***Renal insufficiency***

- 75% destruction of renal parenchyma
- GFR is about 25%
- Elevation in BUN and serum creatinine
- Polyuria and nocturia occur due to tubulointerstitial damage

# Stages of Chronic Renal Failure

***Renal failure***

- 90% destruction of renal parenchyma
- GFR is approximately 10%
- Tubular cells are essentially nonfunctional
- Patient enters into oedema, metabolic acidosis, hypocalcaemia & mild uremia

***End-stage kidney***

GFR is 5%

- Uremic syndrome occurs with progressive primary (renal) and secondary systemic (extra-renal) symptoms

# Stages of Chronic Renal Failure

**Stage** 1 = with normal or high GFR (GFR > 90 mL/min)

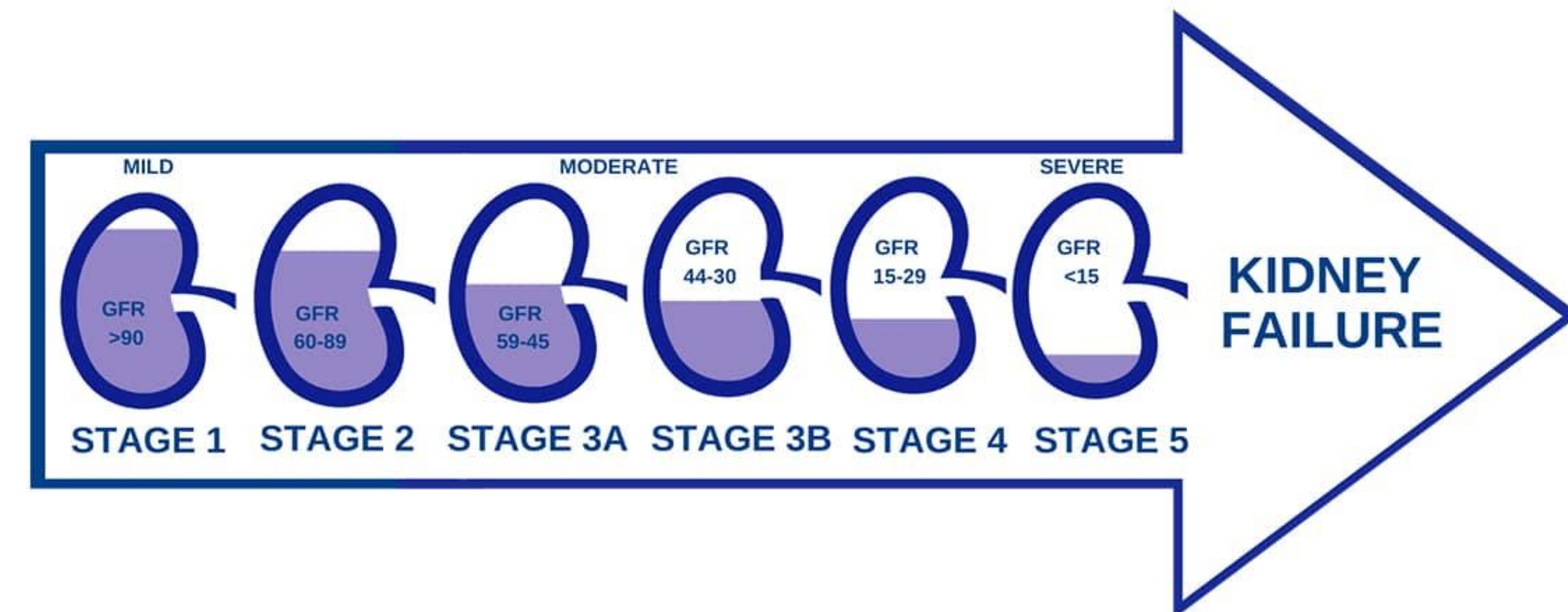
**Stage** 2 = Mild **CKD** (GFR = 60-89 mL/min)

**Stage** 3A = Moderate **CKD** (GFR = 45-59 mL/min)

**Stage** 3B = Moderate **CKD** (GFR = 30-44 mL/min)

**Stage** 4 = Severe **CKD** (GFR = 15-29 mL/min)

**Stage 5** = End **Stage CKD** (GFR <15 mL/min)

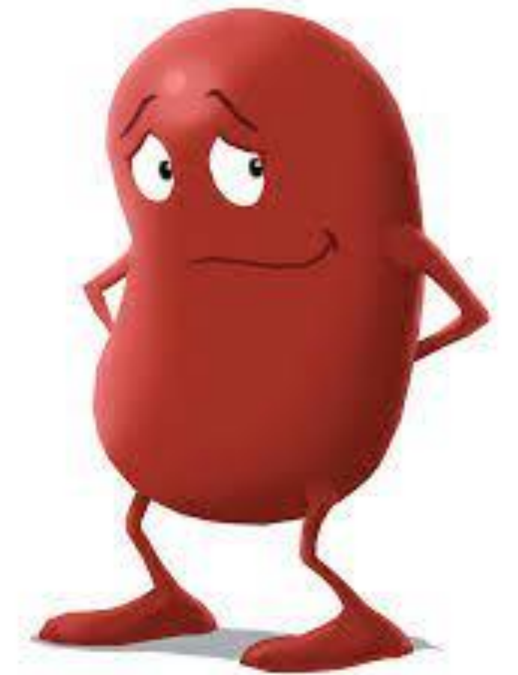


# Pathophysiology

Compensatory hypertrophy of surviving nephrons

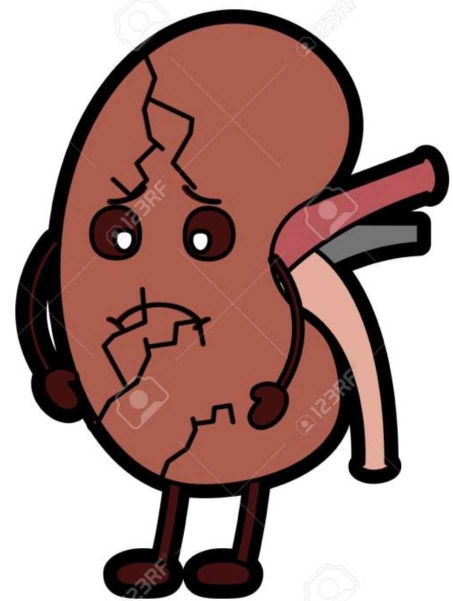
→ adaptive hyper filtration & hypertrophy.

→ Loss of excretory function → Decreased ph, k+, nitrogenous waste excretion.



→ Loss of non-excretory renal function. → Like failure to produce erythropoietin & to convert inactive form of calcium

→ sclerosis of remaining nephrons, & total function loss.



# Clinical Manifestations

## *Primary uraemic (renal) manifestations*

- **Metabolic acidosis -** Excess of hydrogen ions occurs, while bicarbonate level declines in the blood, resulting in metabolic acidosis.
- **Hyperkalaemia -** A decreased GFR results in excessive accumulation of potassium in the blood
- **Sodium and water imbalance –** sodium and water cannot pass sufficiently into Bowman's capsule leading to their retention
- **Hyperuricaemia -** Uric acid crystals may be deposited in joints and soft tissues resulting in gout
- **Azotaemia -** biochemical abnormality, because of elevation

# Clinical Manifestations

***Secondary uraemic (extra-renal) manifestations***

- **Anaemia -** Decreased production of erythropoietin
- **Integumentary system -** Deposit of urinary pigment (sallow-yellow colour)
- **Cardiovascular system -** congestive heart failure (hypervolemia)
- **Respiratory system -** pulmonary congestion and pulmonary oedema
- **Digestive system -** Azotaemia directly induces mucosal ulcerations
- **Skeletal system -** *renal osteodystrophy*

- ***Osteomalacia*** – *deficiency of vitamin D (Less deposition of calcium)*
- ***Osteitis fibrosa*** – *elevated levels of parathromone,* deposits of excess calcium salts in joints and soft tissues and weakening of bones

# Complications

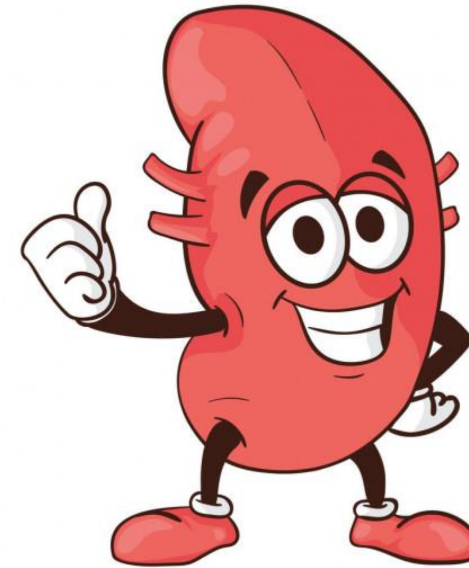
- Fluid retention
- Hyperkalemia
- Cardiovascular disease
- Weak bones and an increased risk of bone fractures
- Anemia
- erectile dysfunction or reduced fertility.
- Damage to your central nervous system.
- Decreased immune response.
- Pregnancy complications that carry risks for the mother and the developing fetus.
- Irreversible damage to the kidneys (end-stage kidney disease)

# Diagnosis

- **Urine Tests** – urinalysis, 24 hrs urine tests, GFR
- **Blood Tests** – Creatinine and Urea (BUN), Acid Base Balance, Erythropoitin
- **Other Tests** – Renal Biopsy, Abdominal CT Scan, Abdominal MRI

# Management

## Pharmacological Management



- Antihypertensive and Cardiovascular Agents
- Anti-seizure Agents
- Erythropoietin
- Antidiuretics
- Antacids - Hyperphosphatemia and hypocalcemia are treated with aluminum-based antacids that bind dietary phosphorus in the GI tract.

## Renal Replacement Therapy

- Dialysis
- Renal Transplantation

# THANK YOU

References:


- Text book of Pathology Harsh Mohan
- Textbook of Pathology for Allied Health Sciences, Ramadas Nayak