ACUTE RENAL FAILURE CHRONIC RENAL FAILURE (PATHOLOGY)

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ACUTE RENAL FAILURE

- Acute renal failure is a syndrome characterized by rapid onset of renal dysfunction chiefly oliguria or anuria and sudden increase in metabolic waste products (urea and creatinine) in the blood with consequent development of uraemia.

Etiopathogenesis

1. Pre renal causes

Pre renal causes are those which cause sudden **decrease in blood flow to the nephron.Renal ischaemia** ultimately results in functional disorders or depression of GFR or both. These cause include **inadequate cardiac out put and hypovolaemia or vascular disease**.

2. Intra renal cause

Intra renal disease is characterized by disease of renal tissue itself. These include

- Vascular disease of the arteries and arterioles within the kidney
- Disease of glomeruli
- Acute tubular necrosis due to ischaemia or the effect of nephrotoxin
- pyelonephritis

3.Post renal causes

Caused by **obstruction to flow of urine** anywhere along the renal tract distal to the opening of the collecting ducts. This may be caused by **a mass within the lumen** or from wall of tract or from **external compression** anywhere along the tract- ureter, bladder neck or urethra.

Clinical features

- One of the following 3 pattern occur

1. Syndrome of acute nephritis

- Mild proteinuria
- Haematuria
- Oedema
- Mild hypertension
- Fluid retention is due to both diminished GFR and increased salt and water reabsorption in distal nephron.

2. Syndrome accompanying tubular pathology

When ARF is caused by destruction of the tubular cells of the nephron as occurs in acute tubular necrosis, the disease typically progresses through **3 characteristic stages from oliguria to diuresis to recovery**

a.Oliguric phase

- This phase lasts from 7 to 10days
- Characterized by by urinary output of less than 400ml per day
- Accumulation of waste products of protein metabolism in blood cause azotaemia,metabolic acidosis, hyperkalaemia, hypervolaemia

b. Diuretic phase

with the onset of healing of the tubules ,there is improvement in urinary output.

c.Phase of recovery

full recovery with healing of tubular epithelial cells occurs in about half the cases while others terminate in death.

3.Pre renal syndrome

Due to decreased renal blood flow there is decrease in GFR causing oliguria,azotaemia, and possible fluid retention and oedema.

Chronic Renal Failure

- Chronic renal failure is characterized by **progressive and irreversible deterioration** of renal function due to slow destruction of renal parenchyma eventually terminating in death when sufficient number of nephrons have been damaged.Development of biochemical azotaemia and clinical uraemia syndrome occurs.

Etipathogenesis

- All chronic nephropathies can lead to CRF and can be classified into 2 major groups

- Those causing glomerular pathology
- Those causing tubulointerstitial pathology

1. Disease causing glomerular pathology

These are covered under 2 headings

a)Primary glomerular pathology

- The major cause of CRF is chronic glomerulonephritis usually initiated by various types of glomerulonephritis such as membranous glomerulonephritis, membranoproliferative glomerulonephritis,

b) Systemic glomerular pathology

Certain conditions originate outside the renal system but induce changes in nephron secondarily.major examples of this type are systemic lupus erythematosus,serum sickness nephritis and diabetes nephropathy.

2.Disease causing tubulointerstitial pathology

It can be categorized according to initiating etiology into 4

a) Vascular causes

Long standing primary or essential hypertension produces characterisic changes in renal arteries and arterioles reffered to as nephrosclerosis.nephrosclerosis causes progressive necrosis of renal tissue.

b) Infectious cause

good example of chronic renal infection causing CRF is chronic pyelonephritis

c) Toxic causes

some toxic substances induce slow tubular injury eventually culminating in CRF.

eg.high doses of analgesics aspirin, acetaminophen

d) Obstructive causes

chronic obstruction in the urinary tract leads to progressive damage in nephron.

eg. Stones , blood clots, tumors, enlarged prostate

Clinical features

CRF evolves progressively through 4 stages

1. **Decreased renal reserve-**

- in this stage damage to renal parenchyma remain marginal and kidney remains functional
- GFR about 50%
- BUN and creatinine values are normal
- Patient usually asymptomatic

2. Renal insufficiency

- In this stage 75% renal parenchyma has been destroyed
- GFR is 25% normal
- Elevated BUN and serum creatinine

3.Renal failure

- 90% functional renal tissue damaged
- GFR 10% normal
- Tubular cells are destroyed resulting in oedema, metabolic acidosis,hypocalcaemia and signs and symptoms of uraemia

4. End stage kidney

- GFR at this stage is less than 5% of normal
- Complex clinical pictures of uraemic syndrome.

Uraemic syndrome described under 2 headings

1.Primary uraemic manifestation

2.Secondary (extra renal) manifestations

- Primary uraemic manifestation

1.Metabolic acidosis

2. Hyperkalaemia –

3. Sodium and water imbalance due to decrease GFR

4. Hyperuricaemia - due to decreased GFR increased uric acid in blood

5. Azotaemia

Secondary uraemic manifestations

- Anaemia
- Integumentary system – deposit of urinary pigment urochrome in skin cause sallow-yellow colour
- Cardiovascular system- fluid retention cause increased work load on heart results in Congestive cardiac failure

- Respiratory system – hypervolaemia and heart failure cause pulmonary congestion and pulmonary oedema
- Digestive system

  Azotaemia directly induce mucosal ulceration in the lining of stomach and intestine

- Skeletal system

Osteomalacia

Osteitis fibrosa due to elevated parathormone

THANK YOU