

Renal Diseases-4

Dr.Nayel Abdelhamid Zaki

**Professor of Internal Medicine and
Nephrology**

Tubulo-interstitial Diseases

2. Interstitial nephritis

Chronic interstitial nephritis

- *Etiology:*

1. Reflux nephropathy
2. Drugs : especially NSAIDS (analgesic nephropathy)
3. Diabetes Mellitus
4. Chronic glomerular diseases
5. Inherited diseases as Sickle cell disease, Wilson's disease.
6. Systemic diseases as sarcoidosis, Sjogren syndrome, SLE.
7. Toxins as lead, cadmium.
8. Tumors as myeloma

Tubulo-interstitial Diseases

2. Interstitial nephritis

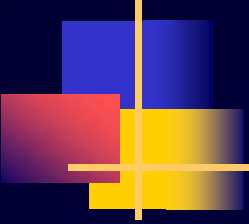
Chronic interstitial nephritis

- *Clinical features:*

1. Chronic renal failure, hypertension, small kidneys.
2. Electrolyte disturbances (hyperkalemia, acidosis)
3. Some patients presents with polyuria, hypotension with risk of development of acute renal failure on top.

- *Management*

1. Conservative management of chronic renal failure
2. Correction of acidosis or electrolyte abnormality as hyperkalemia.



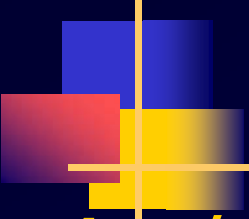
Chronic interstitial nephritis

Analgesic nephropathy;

A type of CIN & papillary necrosis occur as a result of chronic prolonged consumption of analgesics.

Pathology;

- Diffuse interstitial fibrosis& tubular atrophy. Acute papillary necrosis is common.
- Predispose to uroepithelial carcinoma (R. pelvis, ureter and urinary bladder)

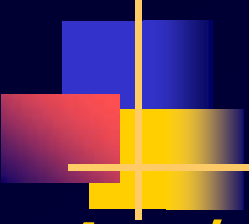


Chronic interstitial nephritis

Analgesic nephropathy;

Clinical features;

- History of drug intake.
- Asymptomatic hematuria, Anemia
- Hypertension
- UT infection
- Salt & water wasting/acidosis (if the damage is mainly tubular)
- Renal impairment / failure
- UT obstruction/R. colic (passage of fragments of necrotic papillae).



Chronic interstitial nephritis

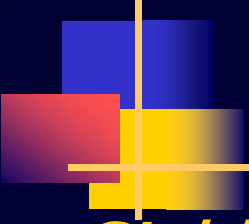
Analgesic nephropathy;

Investigations;

- Biochemical evidence of tubular dysfunction
- IVU; the appearance of the papillae is characteristic.

Management

- Discontinue drugs
- Maintain fluid intake, correction of acidosis, ttt of hypertension, prompt management of UT infection.
- When renal function is severely impaired → dialysis

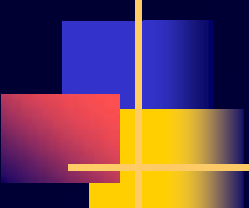


Chronic interstitial nephritis

Sickle cell nephropathy;

As a complication of microvascular occlusion;
Sickling can occur (commonly in vasa recta)
because of hypoxia and hypertonicity →

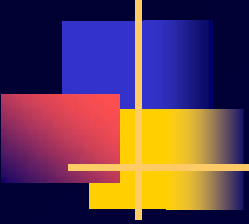
- loss of urinary concentrating ability and polyuria.
- Distal renal tubular acidosis and hyperkalemia
- Papillary necrosis can occur
- Minority develop end stage renal failure



Chronic interstitial nephritis

Reflux nephropathy;

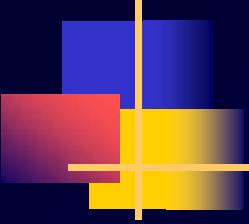
- CIN resulting from urinary tract infection associated with vesico-ureteric reflux.
- VUR is often congenital, usually diminish or disappear as the child grow. It can be due to obstructive lesions at the bladder neck.
- Recurrent UT infections lead to renal scarring.
- Renal damage is aggravated by UT obstruction or stasis (e.g. pregnancy).
- Changes may be unilateral or bilateral with any degree of severity. gross scarring → small sized kidney & narrowing of the cortex and medulla.



Chronic interstitial nephritis

Reflux nephropathy;

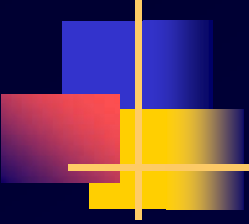
- *Clinical features;*
 1. In many cases no symptoms
 2. Hypertension, proteinuria on routine examination.
 3. Symptoms of UT infection; frequency of micturition, dysuria, aching lumbar pain.
 4. Symptoms of renal calculi.
 5. Uremia; weakness, lassitude,... →



Chronic interstitial nephritis

Reflux nephropathy;

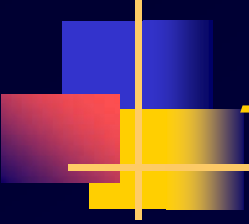
- *Investigations;*
 1. IVU; the kidney is reduced in size, localized contraction of renal substance with clubbing of adjacent calyces.
 2. Urine culture (organisms are commonly E. coli, proteus, pseudomonas or others).
 3. Investigations to detect the cause of obstruction.
 4. Assessment of renal functions.



Chronic interstitial nephritis

Reflux nephropathy;

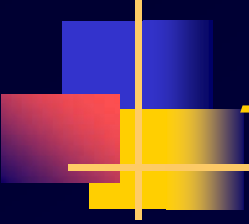
- *Management;*
 1. Management of infection.
 2. Correction of UT abnormalities.
 3. Nephrectomy (unilateral) if pyonephrosis develop with persistent pain, infection or malignant hypertension.
 4. Management of chronic renal failure.



Isolated defects of tubular function

Renal tubular acidosis;

- Failure of either reabsorption of bicarbonate in the proximal tubule or acidification of urine in the distal tubule with little or overall reduction in renal function.
- In both types the *etiology* can be;
 1. Gene defect
 2. Causes of interstitial nephritis

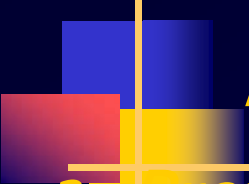


Isolated defects of tubular function

Renal tubular acidosis;

1. Distal renal tubular acidosis

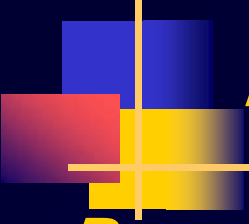
- The ability to form a highly acidic urine is lost, urine PH can't be reduced below 5.3 even in the presence of systemic acidosis.
- *Presented with*
 1. Acidosis
 2. Hypokalemia
 3. Hypercalciuria, hyperphosphaturia, renal stones & osteomalacia.
 4. Children may present with polyuria and thirst



Renal tubular acidosis

2. Proximal renal tubular acidosis

- Proximal tubular Na^+/H^+ exchange is impaired, resulting in decreased bicarbonate reabsorption & large losses of bicarbonate in urine.
- May occur as an isolated defect or as a part of Fanconi's syndrome.
- *Fanconi's syndrome*; generalized proximal tubular dysfunction with hypophosphatemia, glycosuria, aminoaciduria and proximal tubular acidosis.
- *Causes;*
 1. Causes of interstitial nephritis
 2. Some congenital metabolic disorders as Wilson's disease, cystinosis, hereditary fructose intolerance



Renal vascular diseases

Renal artery stenosis

- Well known cause of secondary hypertension and renal failure
- *Pathology;*
 1. Atheromatous narrowing
 2. In young patients: fibromuscular dysplasia (congenital band of fibrous tissue around the artery).
- The affected kidney will have reduced GFR, the unaffected kidney usually shows changes of nephrosclerosis.
- *Management;*
- Medical ttt (antihypertensive: avoid ACEI), Lipid lowering therapy)
- Angioplasty or resection anastomosis.