Nephritic syndrome

- Mild proteinuria
- HTN
- Oliguria
- Hematuria
- Azotemia

Causes:

I - 1° glomerular disease
* PSGN
* IgA nephropathy
* Rapid progressive GN
* Membranoproliferative GN
* Focal & diffuse proliferative GN

II Systemic
* SLE
* PAN
* Wegener granulomatosis
* Cryoglobulinemia

PSGN

Morphology
- light microscopy: Hypercellular
- EM & subendothelial humps
- IF: Starry sky

Causes → flea bitten

IgA nephropathy (Berger's)

Morphology & IgA1 deposition in mesangium

RPGN

- Crescent formation [fibrin + leukocytes + parietal epithelial cells]

→ Good pasture's (1, 2, 3, 4)
RBGN-I
HS-II
α3
type IV-collagen.

Alport syndrome
α5-type IV

RBGN (EM) → α3 - rupture - RBGN - casts.

Nephrotic syndrom

- massive proteinuria
- edema
- Hypoalbunemia
- lipiduria
- Thrombotic events.

Causes:

I 1° Glomerular diseases
- minimal change disease — Effacement of foot processes.
- Focal segmental glomerulosclerosis
- membranous GN — subepithelial deposits → Spike pattern of BM
- Membranoproliferative GN

II Systemic diseases
- SLE
- Amyloid
- DM

III Infection
Viral
Bacterial
Protozoal
Mycoplasm

IV NS
- Drug
- Bee bites

V malignancy

VI Pregnancy
- toxaemia

- Diabetic nephropathy

Clinical syndromes
* Asymptomatic proteinuria
* Nephrotic syndrome
* Progressive renal failure & hypertension

- Renal complication of DM

Pathology :- Diabetic glomerulosclerosis → nodular
→ diffuse.

Vascular lesions

Diabetic pyelonephritis with papillary necrosis

Tubular lesions (Armani - Ebstein lesions)

Diabetic glomerulosclerosis

pathogenesis

hyperglycemia → glycosylation of glomerular matrix → Advance glycated end products

↓

renal hyperperfusion ← glomerular hypertension ← Used resistance to filtration

↓

leakage of plasma proteins into the mesangial areas & glomerular BM

↓

thickening of hyperperfused areas

↓

glomerulosclerosis

— In addition cellular infiltration also

* Diffuse GS - m/c
  - involvement of all parts of glomeruli
  - thickening of GBM & ↑ mesangial matrix
  - Capsular hyaline drops & fibrin caps (+)

* Nodular GS ↑ also known as Kimmelstiel - Wilson (KW)
  → PAS +ve & contains lipid & fibrin

Vascular lesions
- Atheroma of renal arteries
- Hyaline arteriosclerosis

Diabetic pyelonephritis
- susceptible to bacterial infection

Tubular lesions
- Armani ebstein lesion.
- extensive glycogen deposits appears as vacuoles

NEPHRITIC SYNDROME : Clinical syndrome characterized by acute onset of hematuria, oliguria, azotemia, hypertension & mild to moderate proteinuria due to inflammatory damage to glomeruli

Clinical manifestations

- Haematuria
- Proteinuria
- Hypertension
- Oedema
- Oliguria
- Azotemia

Pathogenesis:

Immune complexes / Abs deposit in glomeruli → activates complement → neutrophil infiltration → glomerular capillary wall injury → ↓ GFR → oliguria & fluid retention

Ethology / Causes

- PRIMARY GLOMERULAR DISEASES
  - Acute GN / Post-streptococcal GN (infection related GN) - m/c cause in children
  - IgA nephropathy - m/c common cause in adults
  - Rapidly progressive GN
  - Membranoproliferative GN

- SYSTEMIC DISEASES
  - SLE
  - Wegener granulomatosis
  - Cryoglobulinemia
  - Polyarteritis nodosa (PAN)

i) A/C GN / Post-streptococcal GN

- 2-14 years of age (m/c in children)
- Onset of disease is sudden after 2-4 weeks of streptococcal infection

ETIOPATHOGENESIS :

- 12, 4, 1 strains of streptococcus
- The glomerulus lesions appear to result from deposition of immune complexes.

Renal carcinoma

ETIOLOGY & PATHOGENESIS

1) TOBACCO
   - major RF
2) Genetic factors
   - 1st degree relatives of RCC
   - Hereditary clear cell RCC : AD inheritance
     Papillary RCC - lies in MET gene
     Chromophobe RCC - multiple loss of whole chromosome
3) Cystic diseases of kidneys
   - Polycystic & multicystic
4) Other RF
   - heavy metals
   - Oestrogen therapy
   - Obesity
   - Analgesic nephropathy
   - Hypertension

CLASSIFICATION

- Based on cytogenetics & molecular criteria
- Major histologic type : Clear cell type, Papillary type, Chromophobe type, & collecting duct carcinoma

PATHOLOGY

Gross : Arises from poles of kidney as solitary & unilateral tumor
   - Large, golden yellow & circumscribed.

C/S : large areas of ischaemic necrosis, cystic change & foci of haemorrhages

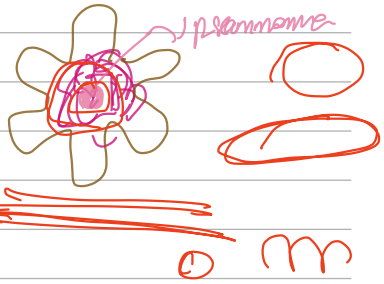
Histologically :


i) Clear cell type RCC

- Clear cytoplasm of tumor cells → due to removal of glycogen & lipid
- Tumor cells have a variety of patterns → solid, trabecular, acinar, tubular.

ii) Papillary type RCC

- Arranged in papillary pattern over fibrovascular stalks.
- Core may show psammoma bodies & foamy macrophages.



iii) Chromophobe type RCC

- Thought to arise from the intercalated cells of renal cortex
- Pale tan to mahogany-brown appearance

- tumor cells are large with well defined cell borders, abundant cytoplasm which varies from eosinophilic to clear in nature.
- Characteristic feature: Perinuclear halllow

iv) Collecting duct carcinoma

- Arises centrally
- Tubular / tubulo-papillary pattern with irregular infiltrating glands.
- Cells exhibit high grade nuclear features with eosinophilic cytoplasm

C/F:

- gross hematuria, flank pain & palpable abdominal mass

WILMS TUMOUR / NEPHROBLASTOMA

- Embryonic tumor derived from primitive renal epithelial & mesenchymal components.

ETIOPATHOGENESIS:

* Higher incidence seen in monozygotic twins & family history
* Association with other congenital anomalies (esp. genitourinary triad)
* Few other malignancies - eg. osteosarcoma, retinoblastoid, neuroblastoma etc.

GENETIC PATHOGENESIS:

* Wilms tumor associated gene - WT1 gene → mutation → tumor.
  WT1 gene → protein synthesis for the dev. of kidneys & gonads.
* Due to common genetic mutation → individuals with gonadal dysgenesis
* Interaction of WT1 gene protein with other genes - eg. β-catenin

PATHOLOGY:

- Gross: Large, spheroidal, solitary, unilateral
  C/S: soft, fish-flesh-like, foci of necrosis & haemorrhage.

M/S:

- 3 types of cellular components: Blastemal, epithelial, mesenchymal
  3 components + → triphasic
  2 " + → Biphasic
  1 " + → monophasic

* Blastemal component - Undifferentiated small round cells with scanty cytoplasm

* epithelial component - absorptive tubules & poorly formed glomerular str
* mesenchymal " - smooth & skeletal muscle, cartilage, bone, fat cells may be seen

C/F → palpable abdominal mass in a child
haematuria
pain, fever, HT