

Renal Pathology / 431

مجموعتي شخصي لكتاب، لاصواب

كتاب - Notes + Robins + Handout

دعواتي لكم بالتوفيق (=)

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Renal Pathology.

I Quick introduction.

- Nephron: Functional unit.
- Capillary is lined from ^{inside: endothelial.} _{outside: visceral.}
- Two types of nephrons → (i) Cortex. (ii) Juxtamedullary.
- Interstitium: Connective Tissue lies between glomeruli & tubules.
- Glomeruli stained by Silver Stain, it shows component clearly.
- Foot Processes seen only in Electron Microscope, which is used in: (i) Renal. (ii) Derm. (iii) Tumors.
- Matrix: Connective tissue (Parenchyma).

II Congenital & cystic diseases.

- * Renal agenesis: Sometimes one kidney or both are absent. if it effect one side, the patient can live.
- * Cystic Renal Dysplasia: As we know, dysplasia means Pre-cancerous lesion. But in this case it is not Pre-malignant. it is just a denomination.
 - Abnormal Parenchyma & Cartilage, usually the child comes with infection.
 - Commonest cystic Renal disease in children.
- * Acquired renal cyst: - It is present normally, sometimes as a one or two cysts which is called "leave-me-alone cysts" because it do nothing. It comes in variable sizes.
- * Autosomal Dominant (AD) Polycystic: Patients compatible with life, they can live until adulthood.
- * PKD-1, the defective gene is on the key of the disease. is hypertension or the short arm of Ch 16, which encode abdominal mass.
a "Polycystine-1 Protein". ← It is inherited on Ch 4 or Ch 16.
- * PKD-2, the defective gene that resides on Ch 4, which encode a "Polycystine-2 Protein".
Pathogenesis: next Page. (15% of cases) (85% of cases)

شرح المرض في نموذج Polycystin تؤدي إلى تكوين الكيسات (cysts) للمرضى، مما يؤدي إلى تضخم الكلى (enlargement of the kidneys) وتؤدي إلى تفتت الخلية (cell-matrix) وظهورها ككيسات (cysts).

* symptoms: - very large kidneys (4 kg), Palpable abdominally & signs. - Flank Pain.

- Intermittent gross hematuria

* The disease usually does not produce symptoms until the fourth decade.

* Autosomal Recessive "childhood" Polycystic: - very large numbers of cysts.

- child comes with renal failure, it is inherited on Ch.6 → mutation in PKHD1 gene, coding for Fibrocystin Protein.

- Liver always affected, Pts die quickly either from hepatic or renal failure, and if survive infancy they will develop liver cirrhosis.

No need for biopsy. ← * Acute tubular necrosis: - The most common cause of acute renal failure.

- Oliguria: less than 400 ml/day.

→ we divide the causes into two major categories.

Hypovolemic (ischemia)	Toxic
• major surgical operation	• Heavy metals (mercury).
• Trauma	• Antibiotics (Gentamicin).
• Burn	• Pesticide.
• Cesarean section	• Ethylene glycol (antifreeze).
• septicemia caused by gram -ve due to shock.	

* The clinical course may be divided into:

i) initiation Phase: lasting about 36 hours, Oliguria, in lab investigations a) \downarrow GFR. b) \uparrow urea, \uparrow creatinine. c) HYP (kalemia). (best e.g. Pregnancy).

ii) maintenance Phase: from 2nd to 6th day, oliguria, uremia, if not treated or didn't use dialysis for 2 weeks (not forever) Patient could die.

(iii) Recovery Phase: Polyuria (more than 3L/day),

Pathology - 2nd lecture

- Tubulo-interstitial diseases caused by infection
 - if it is not treated, Patient will develop septicemia.
 - we use strep-test to indicate leukocyturia, & also indicate sugar, PH, Blood, whether the urine alkaline or acid, Ketone bodies (in diabetic Patients, it indicate Ketoacids).
- Two routes for UTI to occur:

- 1- Hematogenous spread of the infection, e.g. TB, Staphylococcus, and it could be a complication of bacterial endocarditis.
- 2- Ascending route: lead to stagnation of urine which will lead to increase of bacteria. Like:
 - i) Calculi in Pelvi-uretric junction.
 - ii) Mal formation.
 - iii) Strictures (fibrosis) in urethra.
 - iv) Tumor (Sarcoma).
 - v) Pregnancy (Hormons, Anatomy structure)
 - vi) Enlarged Prostate.
 - vii) Vesicouretral reflux (in Pediatric; they will come with fever, \downarrow H_2O , & Vomiting).

abnormal narrowing

- Etiology: we can summarize it in the word (KEEPS)
 - K: Klebsiella E: E. Coli E: Enterobacter P: Proteus S: Serratia
 - Mainly gram -ve.
 - instrumentation is very common e.g (catheterization)

- Pyelonephritis: Cortical abscesses, under the microscope there's a lot of Polymorphonuclear cells, leukocytes interstitium is very edematous, neutrophil attack the tubules (tubulitis).

• Chronic Pyelonephritis:

- Acute Pyelonephritis could lead to Chronic Pyelonephritis with hypertension & Renal failure.

* Grossly: Both kidneys have fibrous scar in cortex.

- Calyces are blunted (flat) at cortic-calyceal region.

* Signs & symptoms

* Disturb electrolytes.

* Problems in GFR.

* Hematuria.

* Burning micturition.

* Dysuria.

• It needs biopsy, so when we look under the microscope:

* Interstitial fibrosis.

* Thyroidization.

* Diffuse & global Malinization.

* Chronic inflammatory cells in interstitium.

* Glomeruli are affected due to ischemia, which will lead to end stage kidney.

• Renal TB: Caseous area & granuloma.

• Staghorn calculi: To know it:

i) UTI: using intra venous Pyelogram.

if the stones are small, the symptoms will appear in more severity.

ii) Renal colic: Exaggerating Pain, usually in flank area. It could go to testis due to innervation.

and obstruction

iii) Hydronephrosis: Abnormal dilation of calyces and pelvis.

Types of calculi: 80% of cases are calcium calculi.

i) Calcium calculi.

Hypercalcaemia

Caused by:

1- Hyperthyroidism.

2- Malignancy.

3- other causes: sarcoidosis, Vitamin D intoxication.

- Radioopaque. - alkaline urine.

- Associated with hypercalcaemia, which is caused

by: a) ↑ intestinal absorption of calcium.

b) ↑ Renal excretion of calcium.

← c) Hypercalcaemia.

ii) Ammonium magnesium Phosphate calculi

- second most common form of stones.
- alkaline urine, caused by *Proteus vulgaris* or *staph*.
- Radiolucent.
- They form large staghorn (Struvite) calculi.

iii) Uric acid stones:

- it could be secondary to gout.
- It could be in an amazement picture especially in patient who are treated with cytotoxic drugs like Leukemia, Carcinoma patient.
- Hyperuricemia.

iv) Cystine Calculi

- Acidic urine
- metabolic error → in PEDI, we must investigate.
- Associated with cystinuria or aminoaciduria.

• Drugs-induced Interstitial Nephritis:

- Any drug that can cause reaction:
 - * Antibiotic → methicillin, Ampicillin, Gentamicin.
 - * NSAID → Phenylbutazone.
 - * Cimetidine → reduce acidity of the stone.
 - * Certain anti-hypertensive agents.
- Many features of the disease suggest an immune mechanism, so serum IgE are increased in some persons suggesting type I hypersensitivity.
- Granulomas, small ones without caseation → suggesting type IV hypersensitivity, synthesized T lymphocytes.
 - * obese patient, hypertensive, and diabetic.
- She developed an osteoarthritis and took NSAID → The drug stimulated type I or 2.
- Sometimes it cause necrotizing Papillitis.

* Renal Papillary necrosis could happen either by: metabolite drug (Phenacetin) that cause injury to cells, or by Aspirin by inhibiting Prostaglandin and PGE₂ synthesis.

the Papilla to ischemia by preventing its vasodilator effect
* How to treat such effect? (drug-induced nephritis in general)?

→ stop the medication, give substitute.

• Cystitis.

- One of the major causes in males is Prostatic hyperplasia.

- It could be cancer, too, so we must take it as a serious case.

* Trabeculation of the bladder wall, which its smooth muscle fibers hypertrophied due to the pressure during micturition.