Pathology

Vascular Diseases (Lec 8)

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1-Nephrosclerosis

"benign" because renal function is minimally affected or proceeds to chronic kidney injury slowly.

▪ Sclerosis of small renal arteries & arterioles that is strongly ass/wi hypertension. (Benign not malignant)

▪ aging / HTN / DM ⟶ ↑ the incidence & severity

▪ Affected vessels have thickened walls &consequently narrowed lumens → focal parenchymal ischemia.

leads combinations of
1-interstitial fibrosis
2-tubular atrophy
3-focal global
4-glomerulosclerosis.

2-Malignant Hypertension

=Malignant Nephrosclerosis

▪ It may present with severe acute kidney injury and renal failure.

▪ A blood pressure usually greater than 200/120 mm Hg. (occurs in only about 5% of hypertensive individuals)

↳ Far less common than essential hypertension

▪ arise de novo (without preexisting HTN) or appear suddenly (individual with mild HTN)

•Clinical:

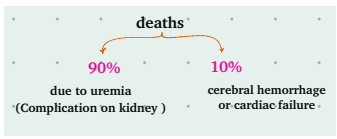
-characterized by papilledema, encephalopathy, cardiovascular abnormalities, & renal failure.

-the early symptoms are related to increased intracranial pressure: headache, nausea, vomiting, & visual impairment.

- Acute kidney injury develops.

- A true medical emergency→ requires prompt & aggressive anti-hypertensive therapy before irreversible renal lesions develop.

- ~ 50% survive at least 5 years.



• Pathogenesis:

hemodynamic changes,aging, genetic defects

↓

1. Medial and intimal thickening

↓

2. Hyalinization of arteriolar walls

caused by →
-extravasation of plasma proteins through injured endothelium
-increased deposition of basement membrane matrix

• Pathogenesis:

-The fundamental lesion in malignant nephrosclerosis is vascular injury:

1. Long-standing hypertension

↓

↑ permeability of the vessels to fibrinogen & other plasma proteins, endothelial injury, & platelet deposition

↓ leads to

fibrinoid necrosis of arterioles & small arteries & intravascular thrombosis.

2. Mitogenic factors from platelets (e.g., platelet-derived growth factor), plasma, & other cell cause hyperplasia of the intimal smooth muscles of vessels

↓

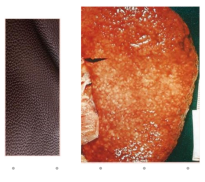
hyperplastic arteriosclerosis

↓

Luminal narrowing → kidneys are markedly ischemic → further elevation of blood pressure via the renin-angiotensin system

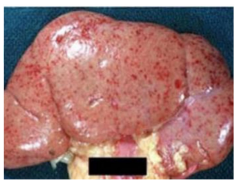
• Morphology -Gross

There is patchy ischemic atrophy with focal loss of renal parenchyma that gives the surface of the kidney the characteristic granular appearance, resembles grain leather.



• Morphology -Gross

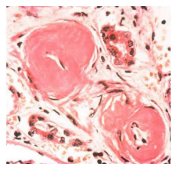
Small, pinpoint petechial hemorrhages may appear on the cortical surface rupture of arterioles or glomerularcapillaries, giving the kidney a peculiar“flea-bitten" appearance.



• Morphology -LM

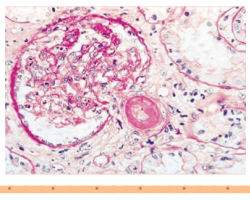
The most prominent change is hyaline thickening of the walls of the arterioles → hyaline arteriolosclerosis.
A homogeneous, pink hyaline thickening, at the expense of the vessel lumina, with loss of underlying cellular details

Homogeneous eosinophilic material

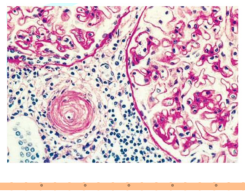


• Morphology -LM

Damage to the small vessels is manifested as fibrinoid necrosis of the arterioles..



In interlobular arteries & larger arterioles, proliferation of intimal cells after acute injury produces an onion-skin appearance (derived from the concentric arrangement of cells). Hyperplastic arteriolosclerosis causes marked narrowing to the point of total obliteration.



Chronic Kidney Disease

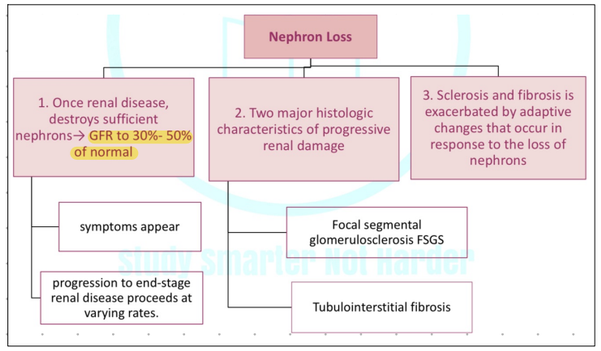
▪ A broad term that describes the final common pathway of progressive nephron loss resulting from any type of kidney disease.

▪ Alterations in the function of remaining intact nephrons are ultimately maladaptive and cause further scarring.

▪ Eventually results in an end-stage kidney; sclerosed glomeruli, tubules, interstitium and vessels, regardless of the anatomic site of the original injury.

▪ Unless the disorder is treated with dialysis or transplantation, death from uremia, electrolyte disturbances, or other complications of ends stage renal disease results.

• Nephron Loss :



▪ Process is initiated by adaptive change in the relatively unaffected glomeruli.

▪ Compensatory hypertrophy

of these glomeruli to maintain renal function
→ ass/w hemodynamic changes;
increases in single-nephron GFR, blood flow
transcapillary pressure (capillary/glomerular hypertension)
→ often with systemic hypertension.

▪ Alterations- maladaptive → further endothelial & podocyte injury:
1. Increased glomerular permeability to proteins
2. Accumulation of proteins & lipids in the mesangial matrix.
3. Capillary obliteration,
4. Increased deposition of mesangial matrix
5. Segmental or global sclerosis of glomeruli.
6. Further reduction of nephron mass

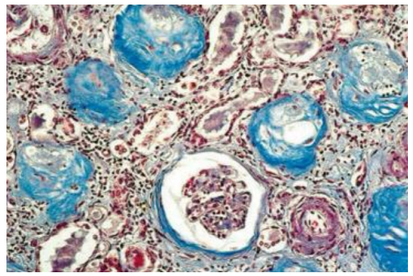
▪ Initiating a vicious cycle of progressive glomerulosclerosis.

• Morphology -LM:

Advanced scarring to complete sclerosis of the glomeruli. Obliteration of the glomeruli is the end point→ impossible to ascertain from the nature of the initial lesion. Also marked interstitial fibrosis.

(A Masson's trichrome stain)

مخصصة تصبغ الfibrosis في(Liver & kidney)



• Clinical :

- asymptomatic develop insidiously and discovered late in course.

- first detected by the discovery of proteinuria, hypertension, or azotemia on routine medical examination

- In patients with glomerular disease resulting in nephrotic syndrome, as the glomeruli undergo sclerotic changes & nephron loss, the avenue for protein loss is progressively lessened, & the nephrotic syndrome becomes less severe with advanced disease.

- Hypertension is very common.

- Without treatment, the prognosis is poor →
progression to uremia and death is the rule.
(The rate is extremely variable).