Benign Nephrosclerosis

- Definition: renal changes in benign hypertension
- It is always associated with hyaline arteriolosclerosis.
- Mild benign nephrosclerosis is present at autopsy in many persons > 60 years of age.
- The frequency and severity of the lesions are increased when hypertension or diabetes mellitus are present.
Pathogenesis

- many renal diseases cause hypertension which in turn is associated with benign nephrosclerosis.
- often seen superimposed on other primary kidney diseases.
Morphology

- the kidneys are symmetrically atrophic, each weighing 110 to 130 gm, with a surface of diffuse, fine granularity that resembles grain leather.
- the basic change is a homogeneous, pink hyaline thickening of the walls of small arteries and arterioles = hyaline arteriolosclerosis.
- This leads to decrease in vessel lumina with loss of underlying cellular detail → markedly decreased blood flow through the affected vessels → produces ischemia in the organ
- All structures of the kidney show ischemic atrophy→
  - glomerular tufts may become globally sclerosed.
  - Diffuse tubular atrophy and interstitial fibrosis are present
Benign nephrosclerosis. Arterioles with hyaline deposition, marked thickening of the walls and a narrowed lumen.
Clinical Course

• rarely causes severe damage to the kidney except in susceptible populations, such as African Americans.

• all persons with this lesion usually show some functional impairment, such as loss of concentrating ability or a variably diminished GFR.

• A mild degree of proteinuria.
Malignant Hypertension and Malignant Nephrosclerosis

- only 5% of HTN cases.
- It may arise de novo or it may appear suddenly in a person who had mild hypertension.

**Pathogenesis**
- vascular damage to the kidneys.
- injury to the arteriolar walls.
- The result is increased permeability of the small vessels to fibrinogen and other plasma proteins, endothelial injury, and platelet deposition.
- fibrinoid necrosis of arterioles and small arteries and intravascular thrombosis.
- The consequences of the markedly elevated blood pressure on the blood vessels throughout the body are known as **malignant arteriolosclerosis**, and the renal disorder is referred to as **malignant nephrosclerosis**.
• Mitogenic factors from platelets (e.g., PDGF) and plasma cause intimal smooth hyperplasia of vessels, resulting in the *hyperplastic arteriolosclerosis* typical of malignant hypertension and of morphologically similar thrombotic microangiopathies

• The kidneys become markedly ischemic.

• Renin-angiotensin system is stimulated.

• Angiotensin II causes intrarenal vasoconstriction → renal ischemia → renin secretion.

• Aldosterone levels are also elevated → salt retention →↑Bp
Morphology

• The kidney is normal-slightly shrunken

• **pinpoint petechial hemorrhages** on the cortical surface from rupture of arterioles or glomerular capillaries giving the kidney a peculiar, *flea-bitten appearance.*

• **fibrinoid necrosis** of the arterioles.

• In the interlobular arteries and larger arterioles, proliferation of intimal cells produces an onion-skin appearance.

• This lesion, called **hyperplastic arteriolosclerosis**, causes marked narrowing of arterioles and small arteries to the point of total obliteration.

• **Necrosis may also involve glomeruli** with microthrombi within the glomeruli as well as necrotic arterioles.
Malignant hypertension.

Fibrinoid necrosis of afferent arteriole (PAS stain).
Malignant hypertension

Hyperplastic arteriolosclerosis (onion-skin lesion).
Clinical Course

- malignant hypertension is characterized by:
  - 1-diastolic pressures > 120 mm Hg,
  - 2-papilledema
  - 3-encephalopathy
  - 4-cardiovascular abnormalities
  - 5-renal failure
• increased intracranial pressure ➔ headache, nausea, vomiting, and visual impairment, particularly the development of scotomatas, or spots before the eyes.
• marked proteinuria and microscopic or macroscopic hematuria
• The syndrome is a true medical emergency that requires prompt and aggressive antihypertensive therapy before irreversible renal lesions develop.
• About 50% of patients survive at least 5 years.
• 90% of deaths are caused by uremia.
• 10% by cerebral hemorrhage or cardiac failure