



Hypertension

By

Dr/ Maisa Hashem

Assistant professor of Pathology

Learning objectives

After the lecture, students should be able to:

- Define systemic hypertension.
- Enumerate its causes and mention its types.
- Describe the effects of systemic hypertension, particularly on the vessels, heart, kidney and brain.
- list causes of death in patients affected with systemic hypertension.
- Define 2ry hypertension and list its causes.

Definition of Hypertension:

- Hypertension means **persistent** elevation of the systolic and diastolic blood pressure above 140/90 mmHg due to increased peripheral resistance in the arterioles.

Types of systemic hypertension:

- Primary (essential) hypertension: common (90-95%), and it is subdivided to two types; benign essential hypertension and malignant essential hypertension.
- Secondary hypertension: Due to underlying cause(s). It is also classified into benign and malignant.

Primary (essential) hypertension

Aetiology:

- Genetic factors: it often runs in family.
- High sodium intake.
- Reduced renal sodium excretion.
- Environmental factors as stress, obesity, smoking, physical inactivity and heavy consumption of salts.

Benign essential hypertension

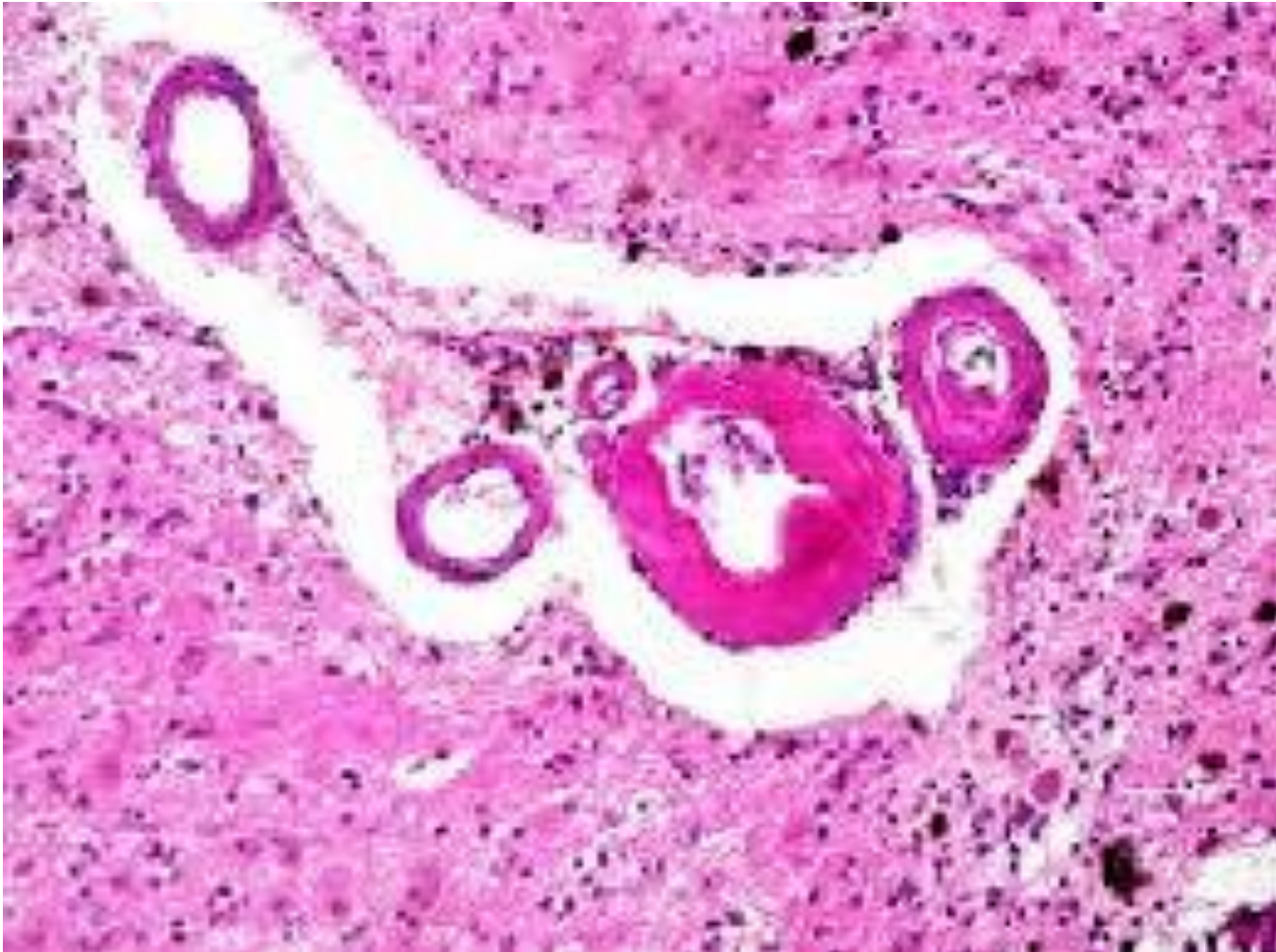
It is a common disease which occur above the age of 40 years. It is characterized by a slowly progressive increase in the blood pressure and a long course of the disease for more than 30 years.

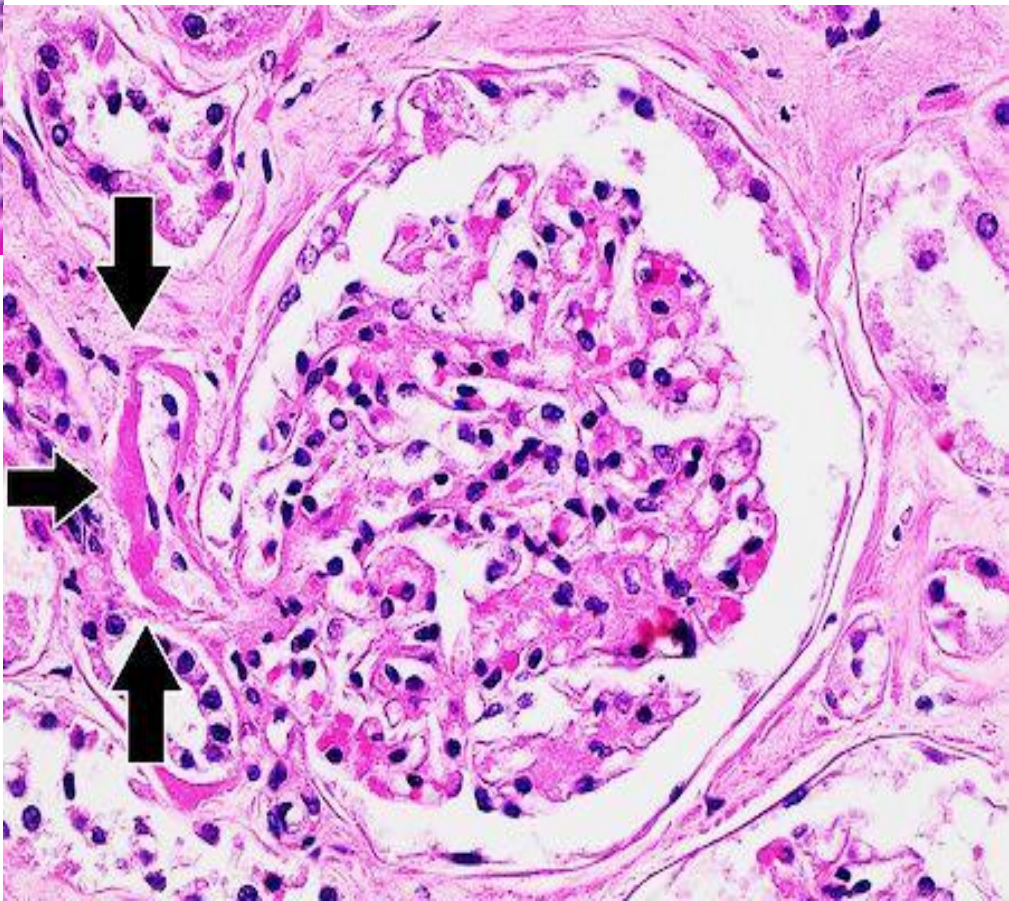
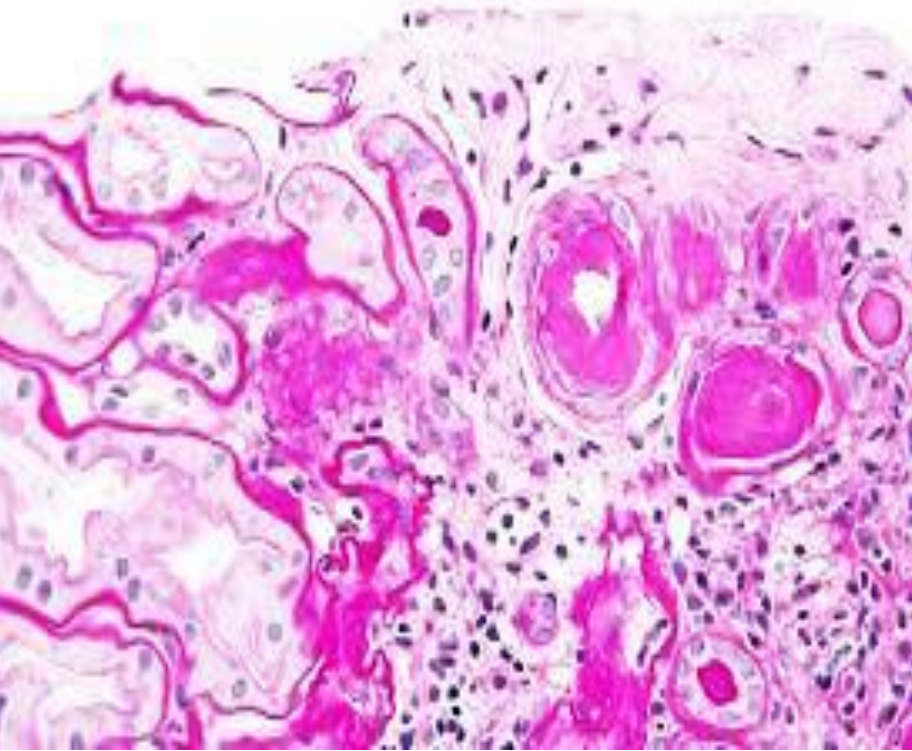
Pathological features

I- Vascular lesions (benign arteriolosclerosis):

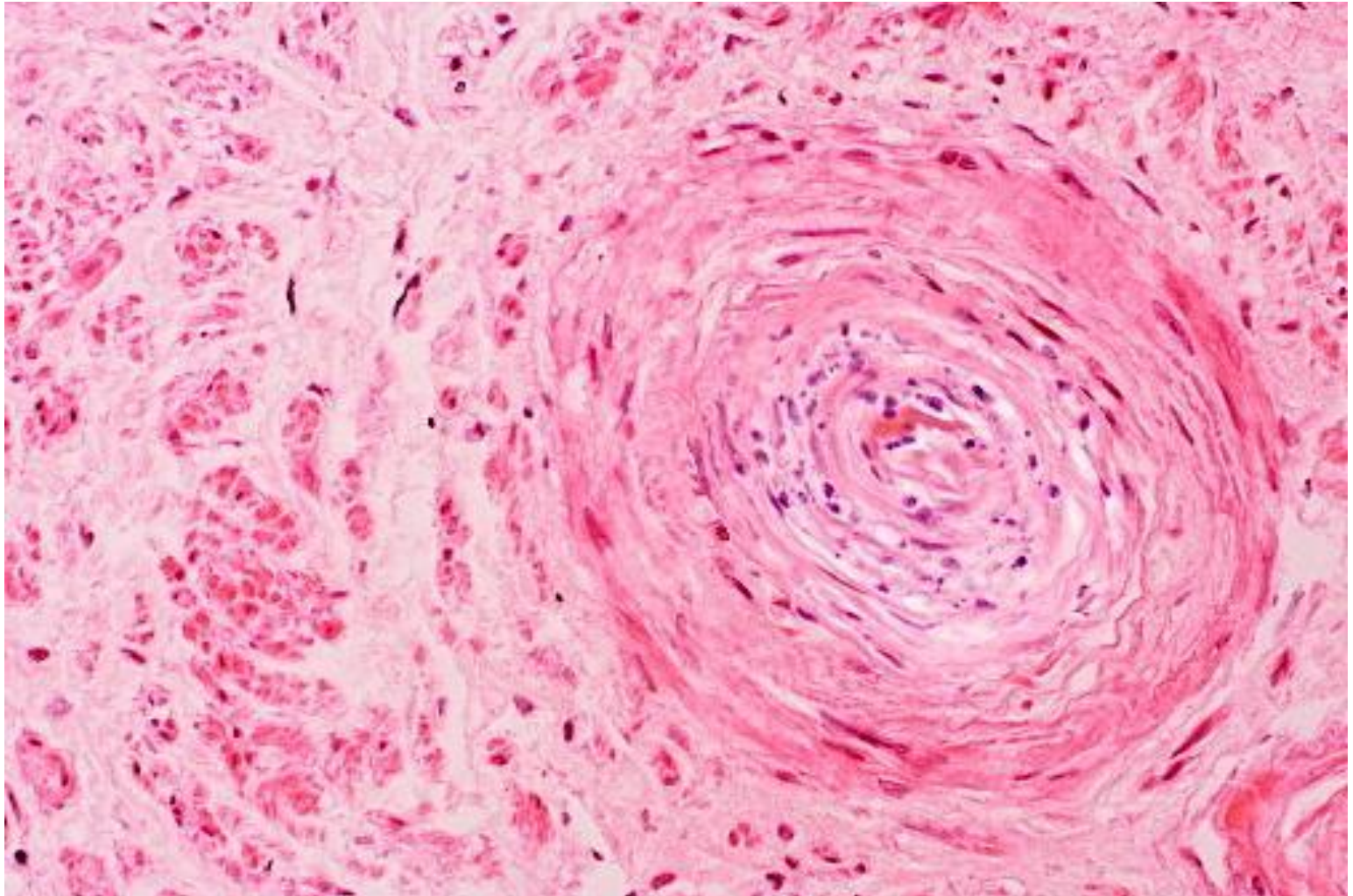
affects small arteries and arterioles less than 100 micron in diameter. The changes occur in the vessels of any structure, especially in the kidney, brain and retina. The lesions take a patchy distribution.

Hyalinosis: a pathological thickening of small arteries and arterioles, characterized by the deposition of pink, amorphous plasma proteins within vessel walls, leading to thickening of the wall and narrowing of the lumen.





Elastosis: hyperplasia and splitting of the internal elastic lamina into several layers. Elastosis affects the larger arterioles and small arteries.



II- Kidney lesions (Benign Nephrosclerosis):

Gross picture:

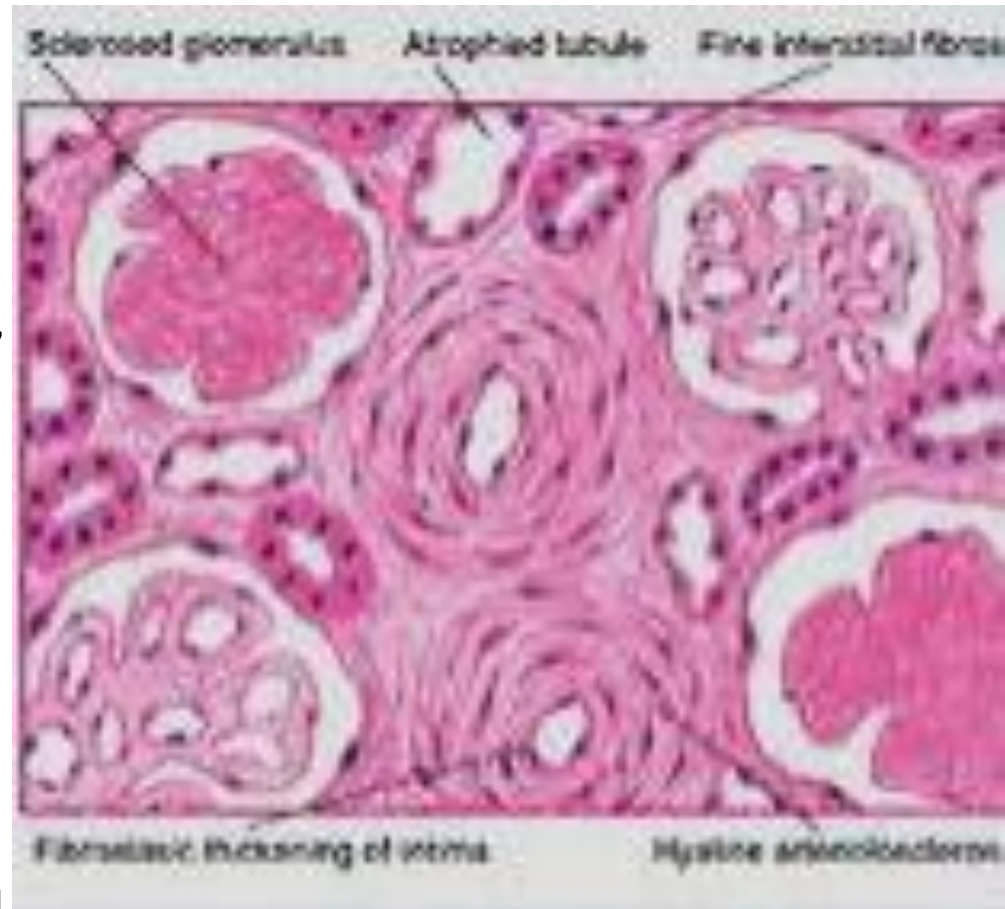
- In long standing cases of hypertension; both kidneys are small, contracted and firm (**primary contracted kidney**).
- The capsule is adherent.
- The outer surface shows fine granules and shows small retention cysts.
- The cut surface shows irregular atrophy and fibrosis of the cortex with loss of demarcation between cortex and medulla.
- The arterioles are thick walled and prominent.
- The perinephric fat is relatively increased.

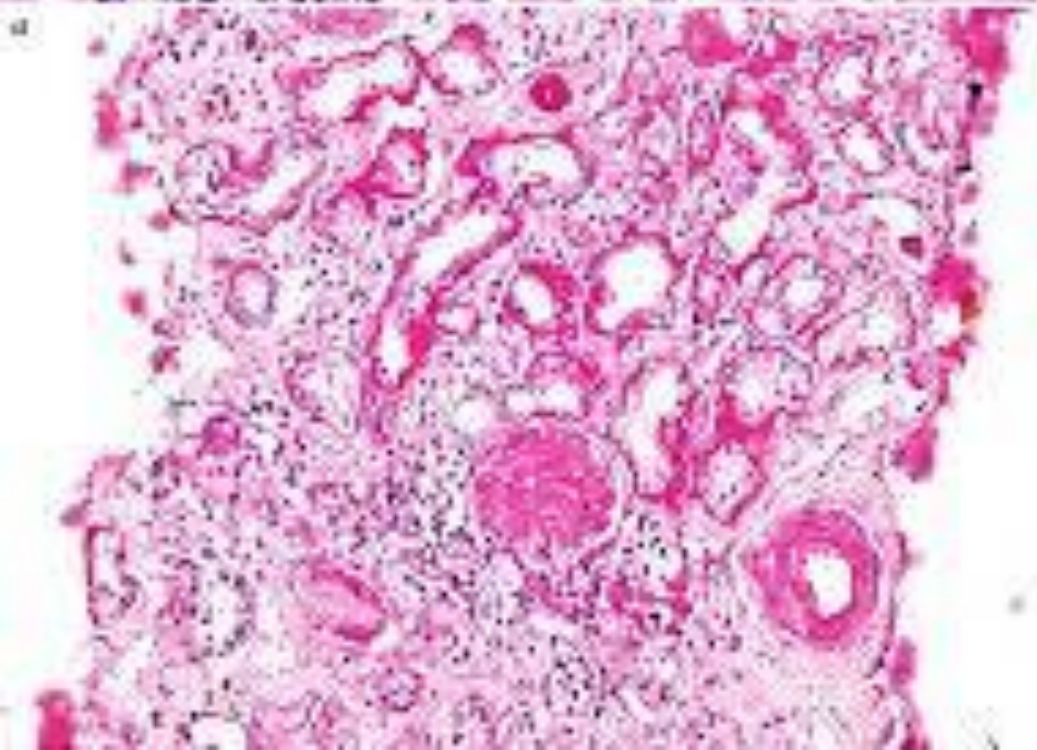
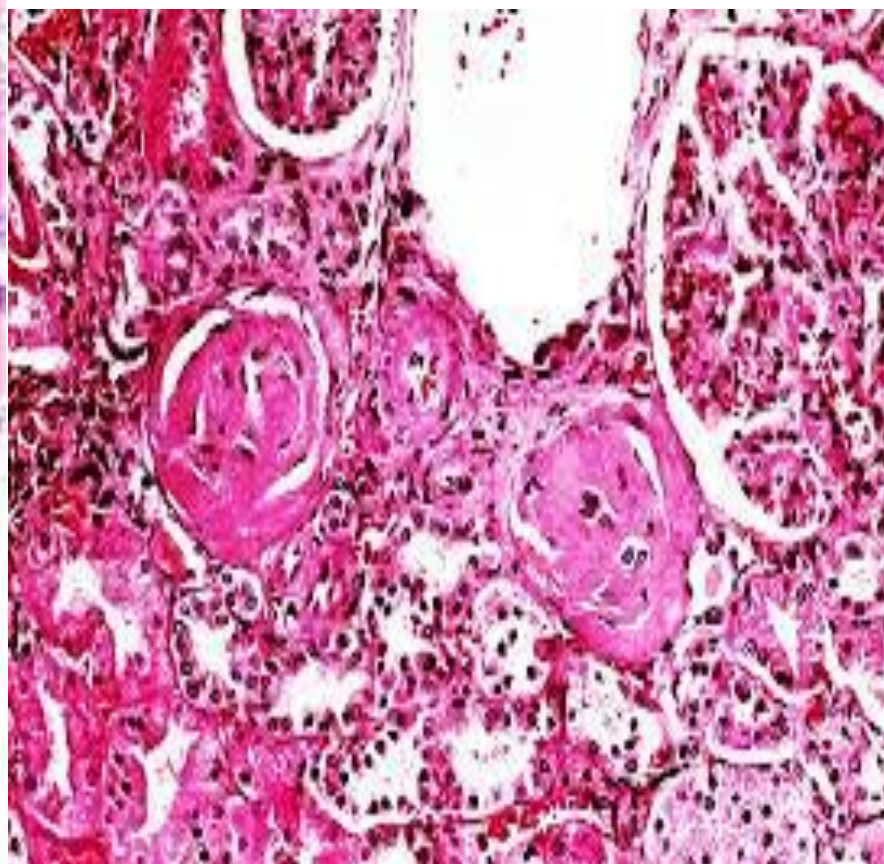
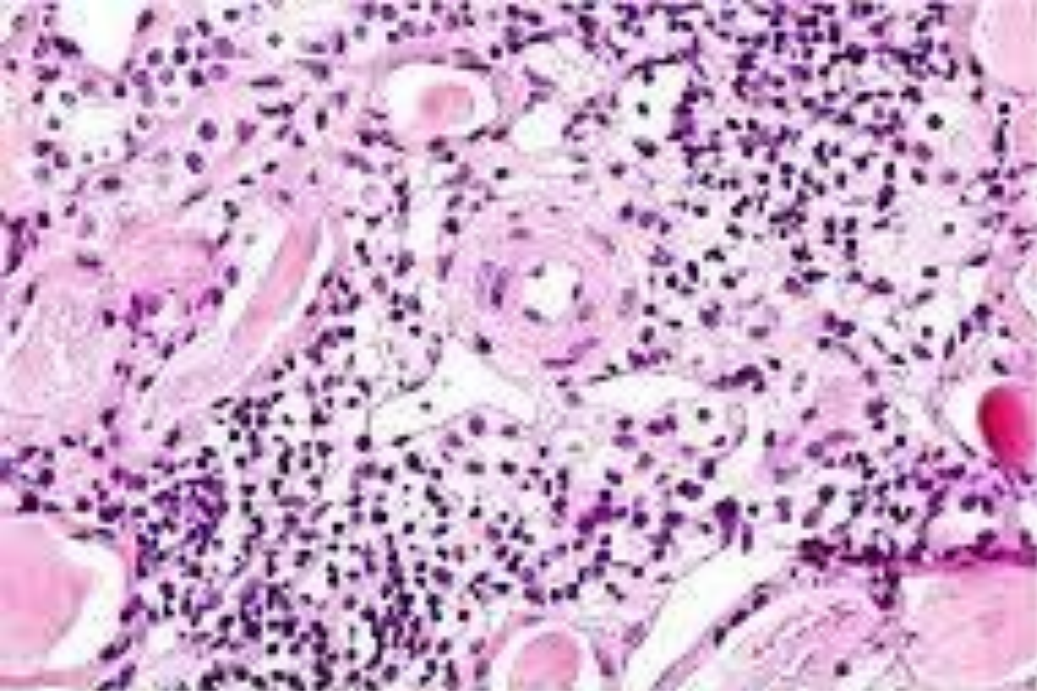


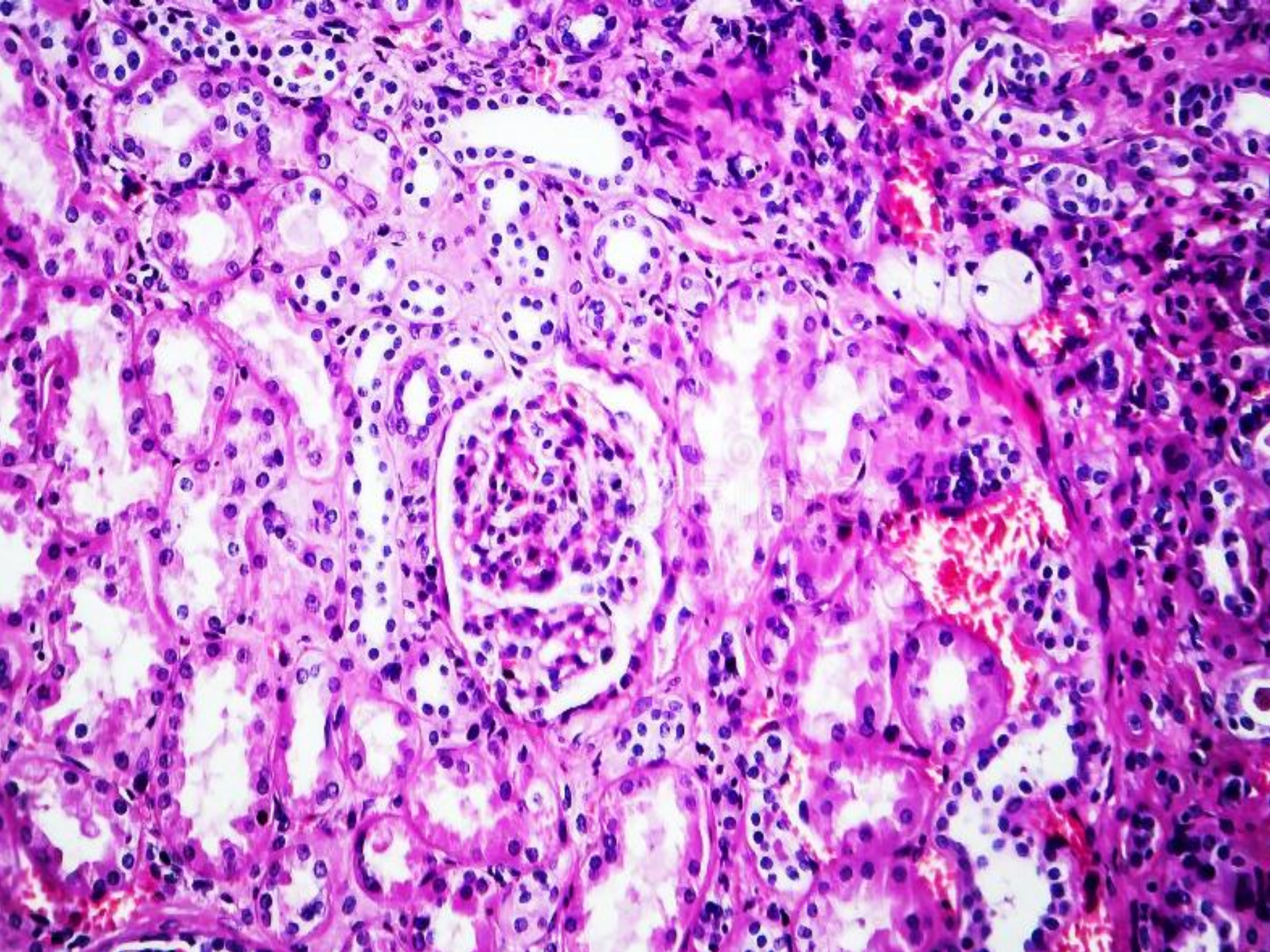


Microscopic picture:

- Afferent and efferent arterioles show hyalinosis and elastosis.
- The glomeruli shows thickening of the glomerular basement membrane, followed by atrophy, fibrosis and hyalinosis of the whole glomerulus.
- The tubules attached to the fibrosed glomeruli undergo atrophy and fibrosis. While, tubules attached to the functioning glomeruli show compensatory dilatation and may form small retention cysts.
- The interstitial tissue is thickened and shows lymphocytic infiltration.

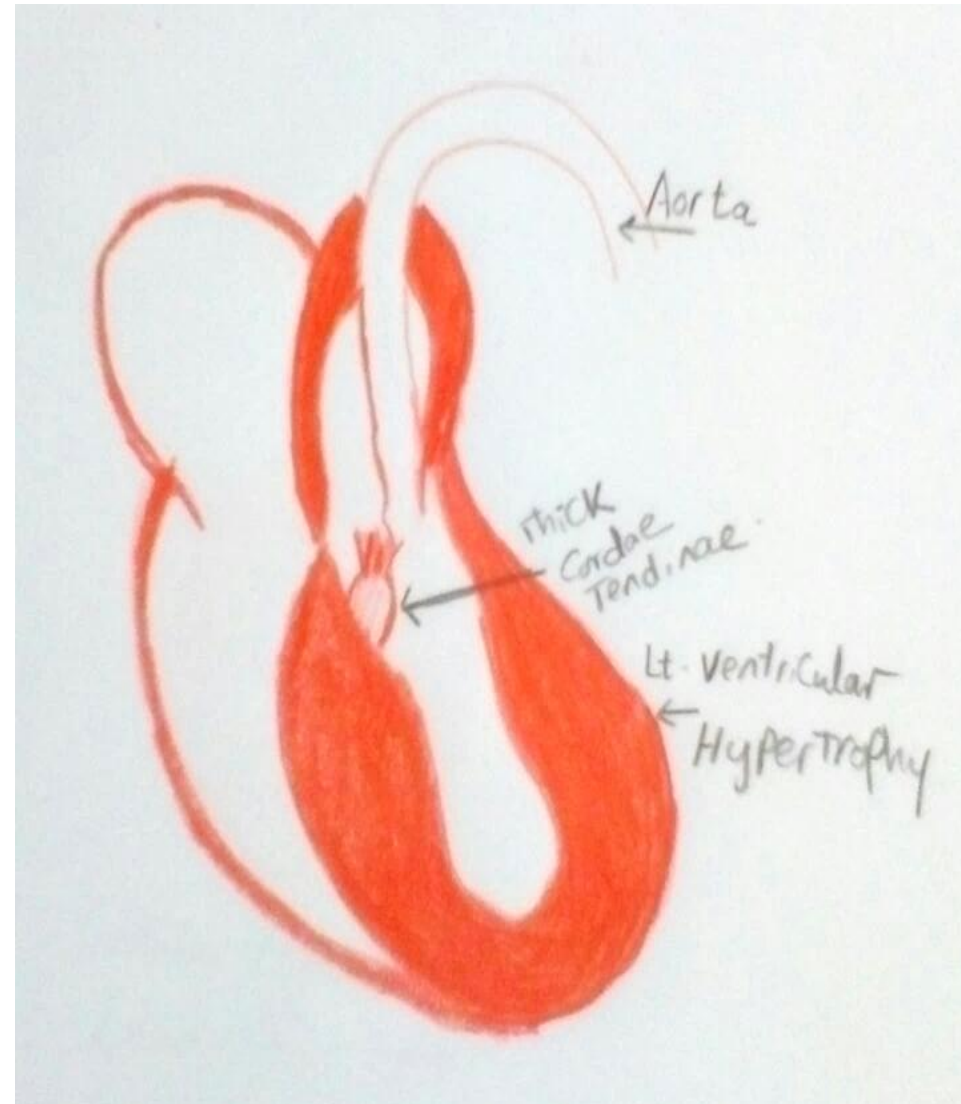






III- Cardiac lesions (hypertensive cardiomyopathy):

Constricting hypertrophy of the left ventricle due to high resistance caused by elevated blood pressure, the myocardium become thickened (2cm or more), the papillary muscles show hypertrophy and the septum is thickened and pulge into the right ventricular cavity. Next; the left atrium undergo hypertrophy. The hypertrophy enables the heart to remain in a state of compensation, eventually decompensation occurs and the cavities of the left side dilate.



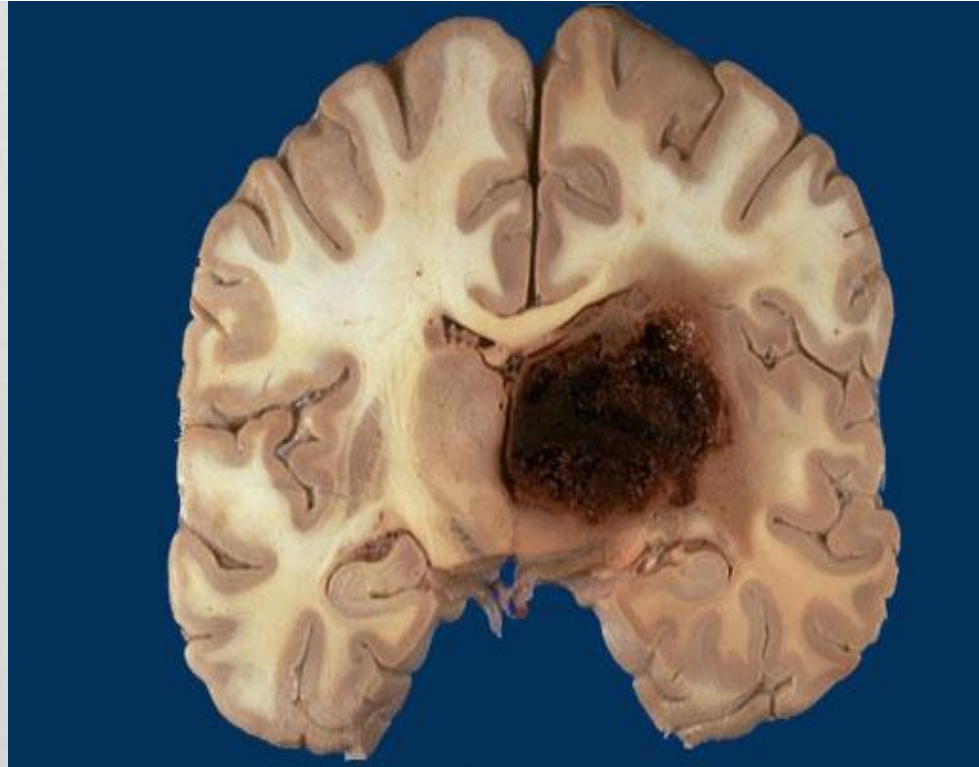
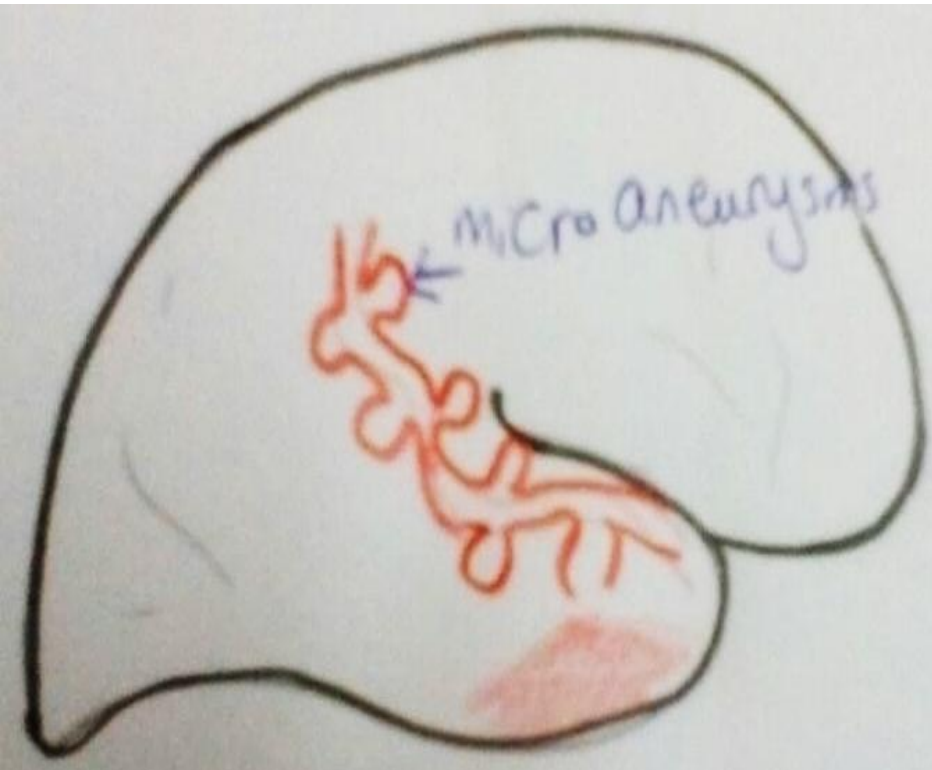


Seen in cross section, the heart is markedly enlarged due to a very thickened (hypertrophied) left ventricle in a patient with long-standing hypertension.

IV- Retinal changes:

- Retinal hemorrhage.
- Retinal exudate.

V- Cerebral lesions: micro-aneurysms occur in the small cerebral arteries which may rupture causing massive cerebral hemorrhage.



Causes of Death of Benign Essential Hypertension

- Left or right side heart failure (60%).
- Cerebral hemorrhage (30%).
- Chronic renal failure (10%).

*Malignant (Accelerated) essential
hypertension:*

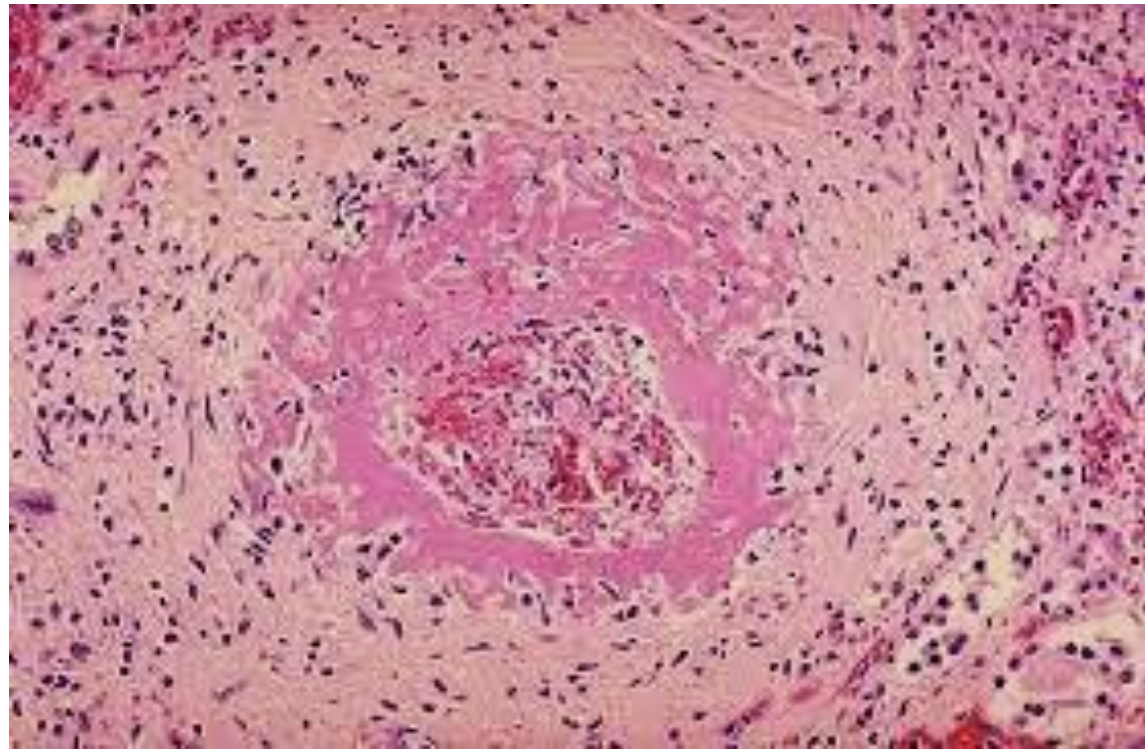
It is a rare disease affecting mainly young adults. It is characterized by a rapid progressive increase in blood pressure and short fatal course.

Pathological features of malignant hypertension:

1- Vascular lesions (malignant arteriolosclerosis):

- Arteriolar hyalinosis and elastosis are less marked than in benign hypertension due to short course of the disease.
- Cellular hyperplasia: concentric hyperplasia of the subendothelial connective tissue and the smooth muscles of the media of the small arteries and large arterioles. The vessel become narrow and its wall is thickened and **shows onion skin appearance**.

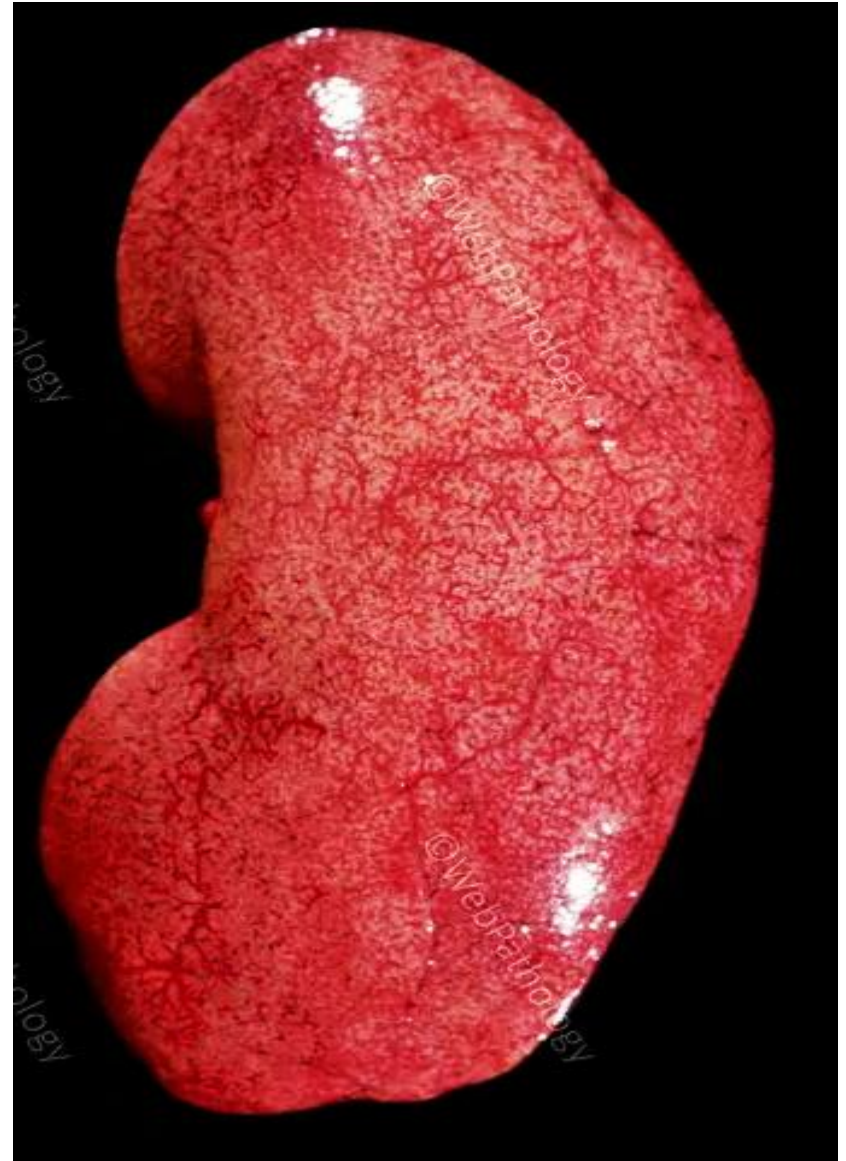
- Acute arteriolar necrosis: fibrinoid necrosis in the wall of the arterioles and small arteries mainly in the kidney, brain and retina. The vessel wall appear thickened, necrotic and infiltrated by fibrin, RBCs and polymorphs. The lumen is reduced and may show thrombosis. Hemorrhage may occur.



2- Kidney lesions (malignant nephrosclerosis):

Gross picture: both kidneys are normal in size. The capsule strips easily. The outer surface is smooth and shows focal hemorrhage. The vessels are prominent, thick and narrow.

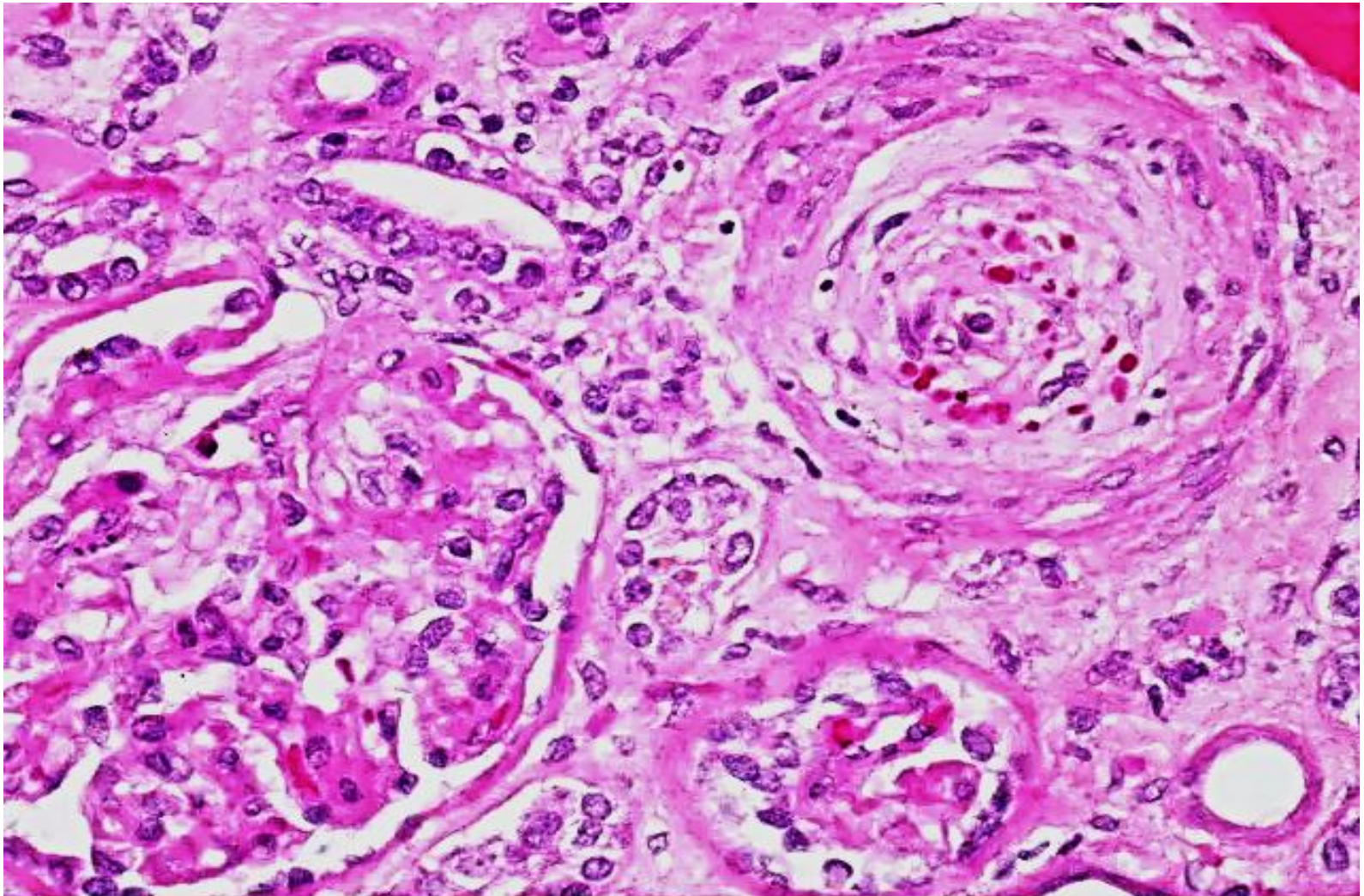
If malignant hypertension occurs on top of pre-existing benign hypertension; the kidneys are reduced in size, with granular outer surface and shows focal hemorrhage on the surface.





Malignant Nephrosclerosis: Grossly, the kidneys have a **mottled appearance** due to **petechial hemorrhages** on the surface as well as in the cortex. See close up in the next image.

Microscopic picture: necrosis of the afferent arterioles and glomerular capillaries. Hemorrhage in Bowman's capsule. Cellular hyperplasia in small arteries. Glomerular hyalinosis and tubular atrophy or dilatation.



3- Cardiac lesions: slight hypertrophy of the left ventricle.

4- Retinal lesions: retinal hemorrhage and retinal exudate.

5- Cerebral lesions: arteriolar necrosis causing massive cerebral hemorrhage.

Causes of death in malignant/ accelerated essential hypertension:

- Acute renal failure in 95% of cases.
- Cerebral hemorrhage.
- Left sided heart failure.

Causes of Secondary hypertension

1: Renal causes:

- Chronic renal disease.
- Polycystic kidney.
- Renal artery stenosis.
- Renin-producing tumors.

2: Endocrine causes:

- Pheochromocytoma.
- Acromegaly.
- Hypothyroidism (myxoedema).
- Hyperthyroidism (thyrotoxicosis).
- Pregnancy induced.
- Adrenocortical hyperfunction (as Cushing syndrome).

3: Cardiovascular causes:

- Coarctation of Aorta.
- Polyarteritis nodosa.
- Increased cardiac output.

4: Neurological causes:

- Increased intracranial tension.
- Acute stress, including surgery.



Thank You