

Hypertension

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Classification

- Primary/Essential hypertension:
 - 90-95% of cases.
 - Unknown etiology
 - Associated with my risk factors
- Secondary hypertension
 - Secondary to underlying organic pathology
 - 5-10%
 - Most renal pathology – e.g glomerulonephritis
 - Endocrine causes – e.g. hyperthyroidism, pheochromocytoma
 - Neurogenic – e.g. acute stress such surgery, ICP

Essential Hypertension

- Many risk factors interact and play role in its pathogenesis.
- Determinants of essential hypertension:
 - Family history – high risk in those with +ve Hx
- Environmental factors:
 - Dietary sodium intake: high risk with high intake
 - Stress – high stress related to high risk
 - Other factors: obesity, smoking, physical inactivity

Target Organs for Complications

- CVS (Heart and Blood Vessels – big & small vessels)
- The kidneys – essentially it is a vascular pathology
- Nervous system
- The Eyes

Effects On CVS

- Heart: Ventricular hypertrophy, dysfunction and failure.
- Conduction: Arrhythmias
- Small vessels: Coronary artery disease, Acute MI
- Large vessels: Arterial aneurysm, dissection, and rupture.

Vascular Pathology

- Hypertension accelerates atherogenesis
- Induces hyaline arteriosclerosis
 - common in old age. Common in diabetes.
- Induces hyperplastic arteriosclerosis
 - Related to more acute or severe hypertension
 - Characteristic in malignant hypertension (but not limited to it)

Effects on The Kidneys

- Glomerular sclerosis leading to impaired kidney function and finally end stage kidney disease.
- Ischemic kidney disease especially when renal artery stenosis is the cause of HTN

Nervous System

- Acute event: Stroke, intracerebral and subarachnoid hemorrhage.
- Chronic hypertension: Cerebral atrophy and dementia

Pathological changes in stroke

Massive hypertensive hemorrhage into a lateral ventricle



Hypertensive hemorrhage in the pons



The Eyes

- Retinopathy: retinal hemorrhages and impaired vision.
- Vitreous hemorrhage, retinal detachment
- Neuropathy of the nerves leading to extraocular muscle paralysis and dysfunction

Results of Essential hypertension

- Eye – hypertensive retinopathy
- Cardiac :
 - LVH & cardiac failure
 - IHD
 - Stroke: hemorrhagic stroke
 - Conduction abnormalities
- Kidney – hypertensive nephropathy. Essentially vascular changes.

Hypertensive Retinopathy

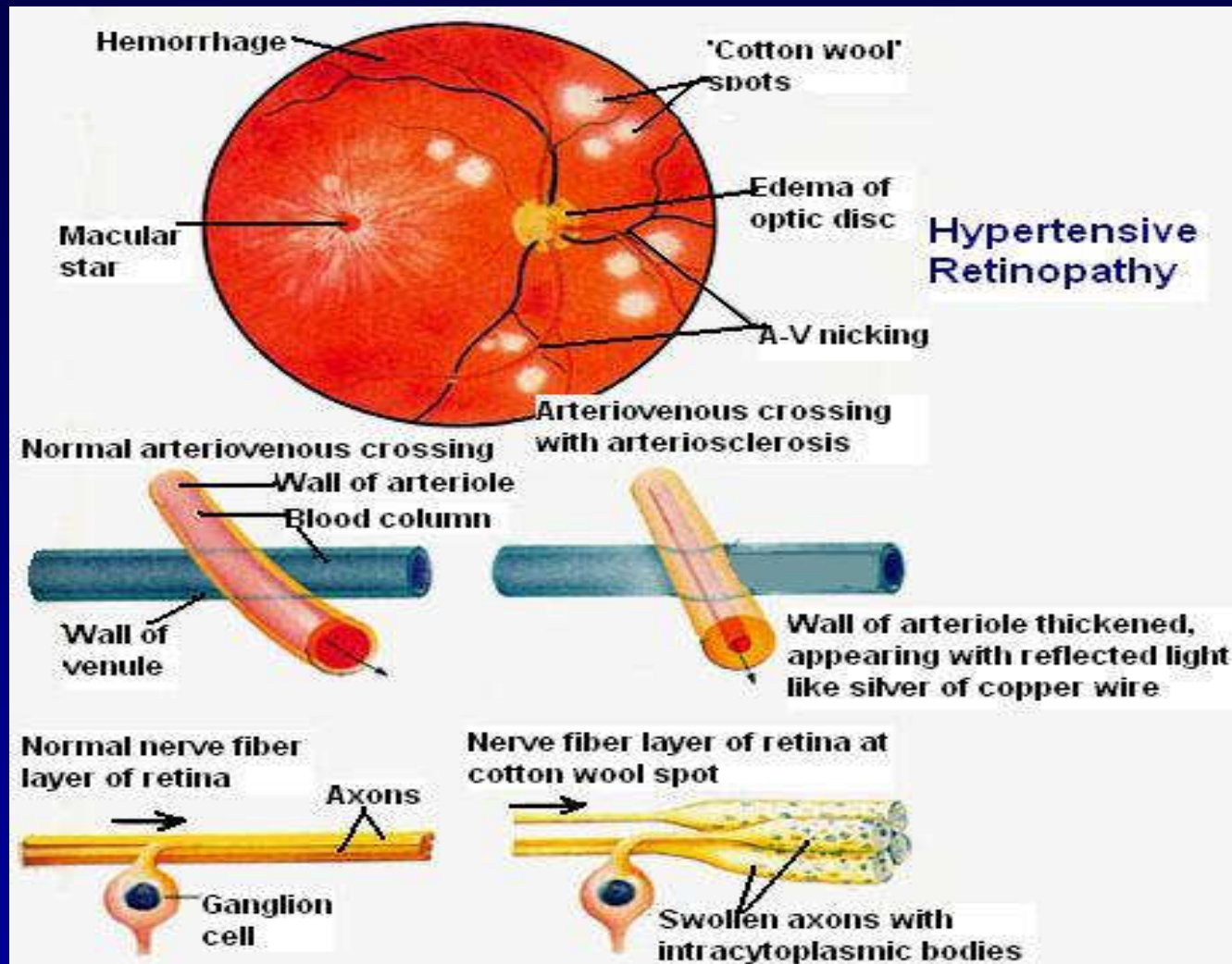
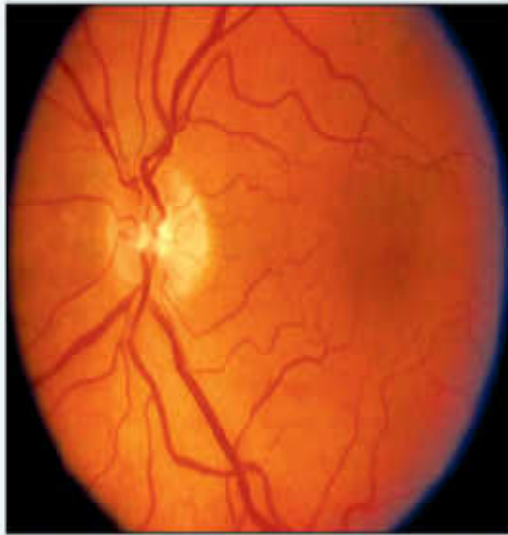


Image from: www.pathology-india.com

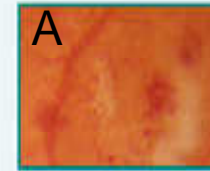
Retina Normal and Hypertensive Retinopathy



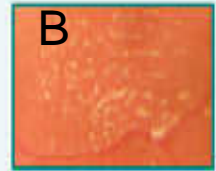
Normal Retina



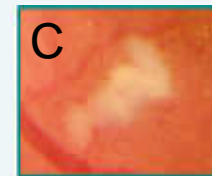
Hypertensive Retinopathy



A



B



C

A: Hemorrhages

B: Exudates (Fatty Deposits)

C: Cotton Wool Spots (Micro Strokes)

Classification of Hypertensive Retinopathy

Grade	Classification
Grade I	Mild generalized retinal arteriolar narrowing or sclerosis
Grade II	Definite focal narrowing and arteriovenous crossings Moderate to marked sclerosis of the retinal arterioles Exaggerated arterial light reflex
Grade III	Retinal hemorrhages, exudates and cotton wool spots Sclerosis and spastic lesions of retinal arterioles
Grade IV	Severe grade III and papilledema

Hypertensive Retinopathy - Grade 4

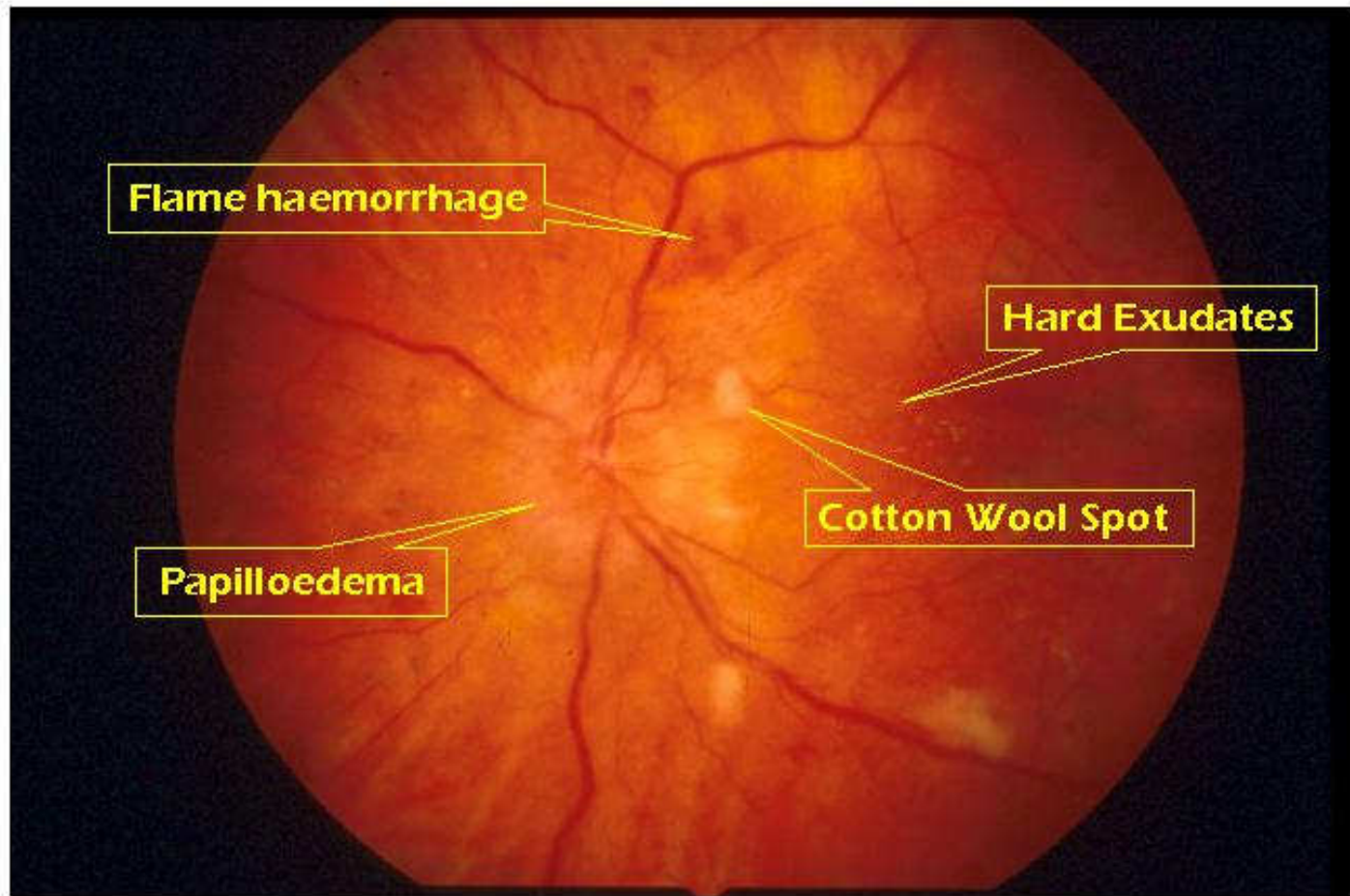
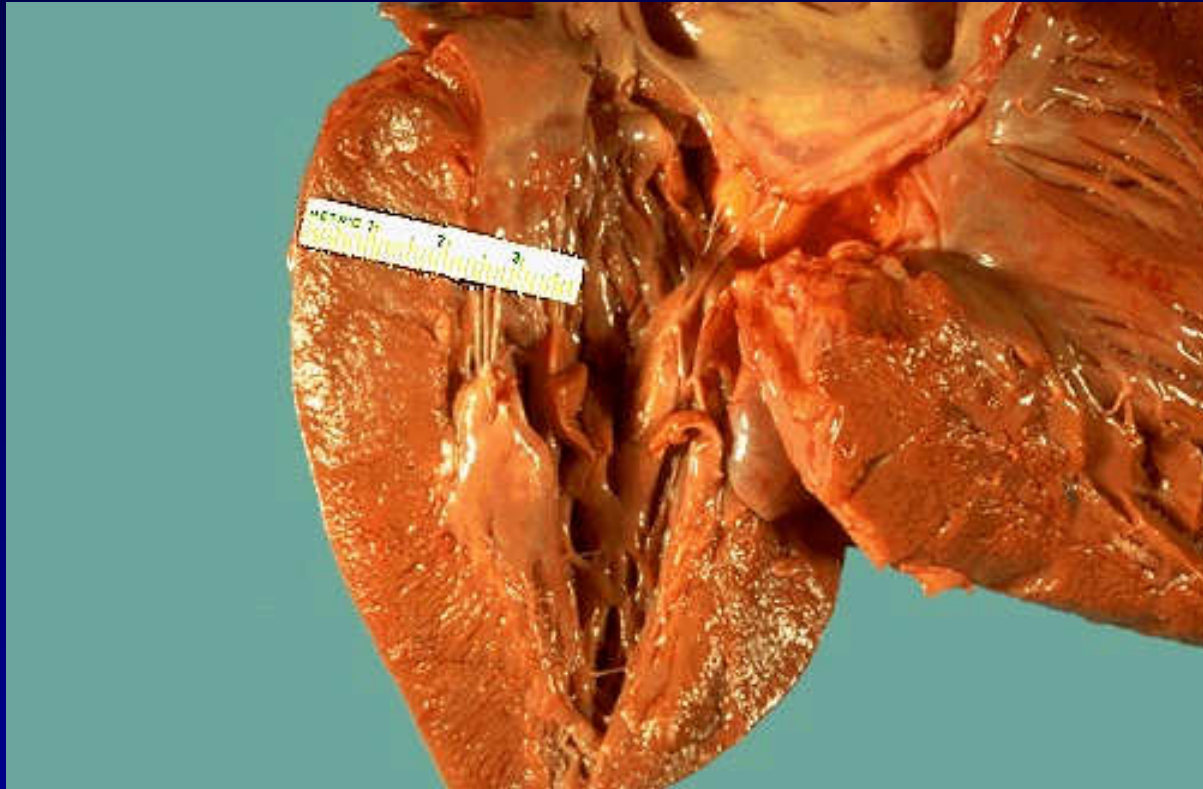


Image from: www.theeyeppractice.com.au

Cardiac changes: LVH



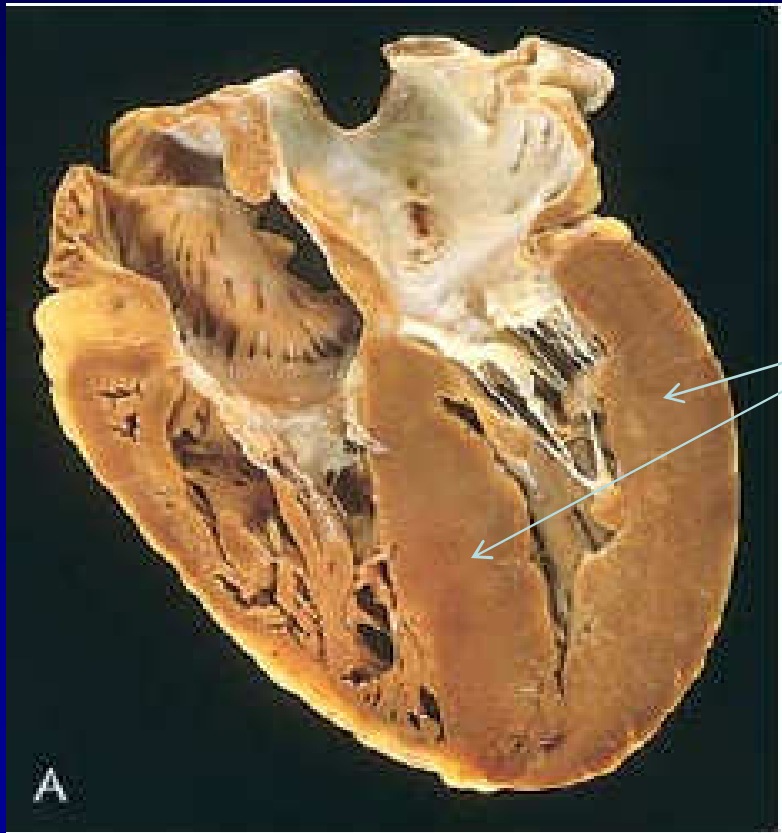
This left ventricle is very thickened (slightly over 2 cm in thickness), but the rest of the heart is not greatly enlarged. This is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy.

Image from: <http://library.med.utah.edu/>



The left ventricle is markedly thickened in this patient with severe hypertension that was untreated for many years. The myocardial fibers have undergone hypertrophy.

LVH



- Pressure hypertrophy due to ventricular outflow obstruction.
- Left ventricle wall grossly enlarged.

Pressure hypertrophied heart



Normal heart



Dilated heart



B

Increased mass with thin walls



Ref: interactive pathology manual, UTAS.

Hypertensive vascular changes

- 2 forms of small blood vessel diseases:
 - Hyaline arteriosclerosis
 - Hyperplastic arteriosclerosis
- Large vessel diseases:
 - Aortic dissection & Aneurysm
 - Cerebrovascular haemorrhage

Aortic Dissection - Macro

Right carotid artery
compressed by blood
dissecting upwards



Image from: <http://library.med.utah.edu/WebPath>

Aortic Dissection - Micro

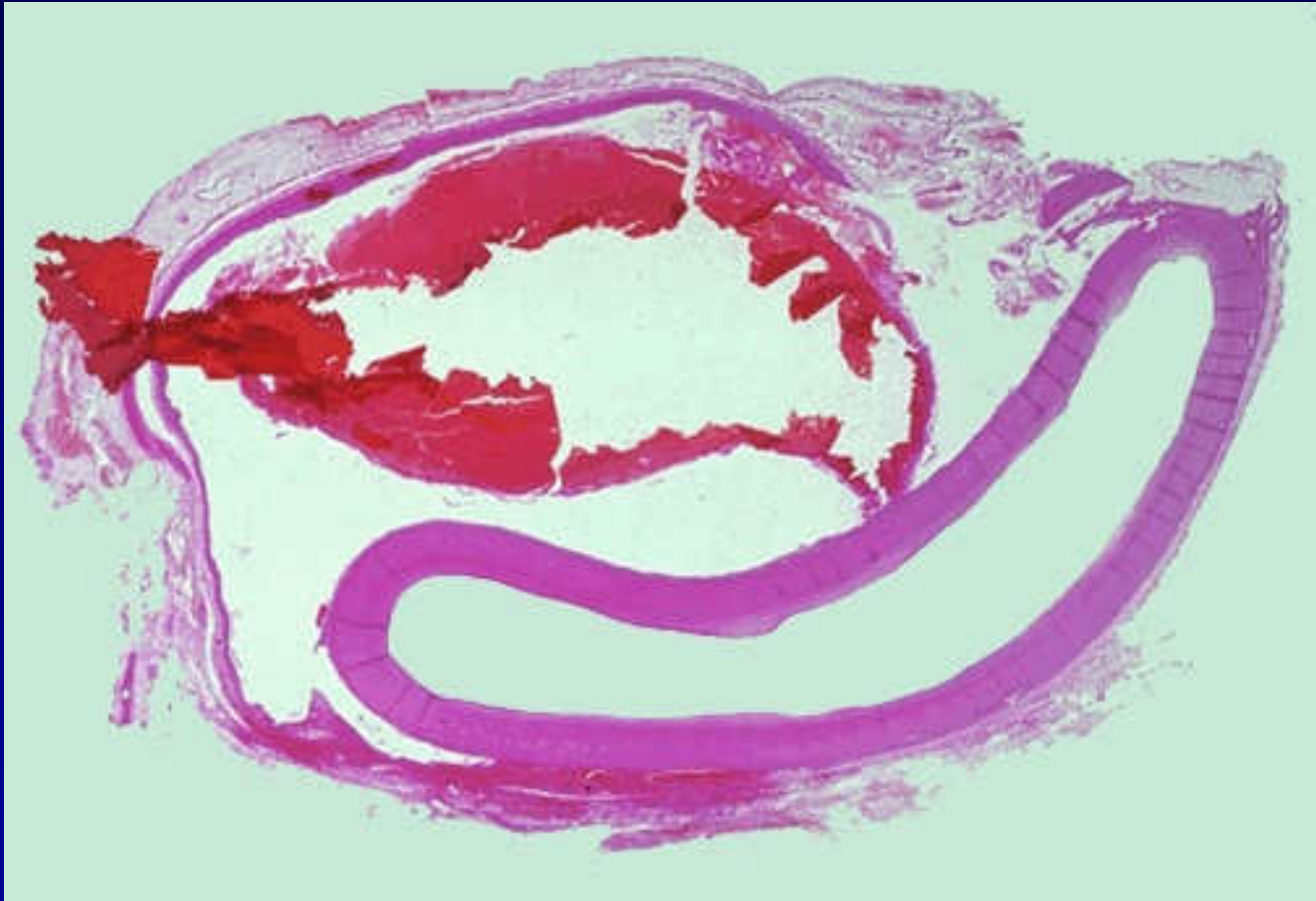


Image from: www.library.med.utah.edu/WebPath

Aneurysm



Aortic
aneurysm
with
organised
blood clot

Image from: www.quizelet.com

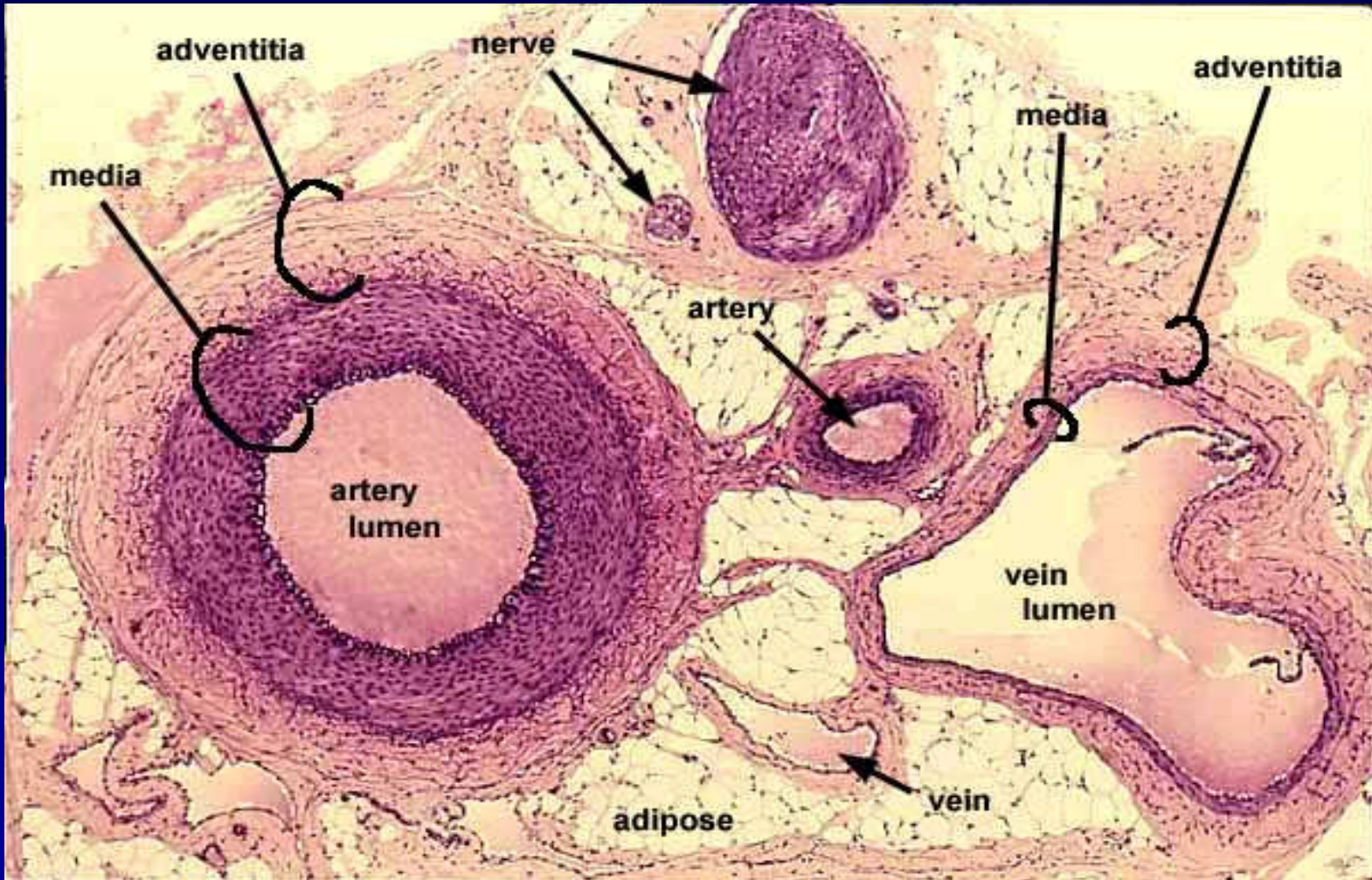
Atherosclerosis

- Most frequent cause of vascular disease worldwide.
- Characterised by: fibrous plaques or atheromas in intima or arteries affecting coronary arteries, carotid arteries, circle of Willies, large vessels of lower limbs, renal and mesenteric arteries.
- Plaques have a central core of cholesterol & cholesterol esters, lipid-laden macrophages or foam cells, calcium and necrotic debris.
- Core covered by a subendothelial fibrous cap made up of smooth muscles, foam cells, fibrin and coagulation proteins, collagen, elastin, glycosaminoglycans, proteoglycans and ECM.

Atherosclerosis

- Plaques maybe complicated by: Ulceration, haemorrhage into plaque or calcification of plaque, thrombus formation at the site causing obstruction to blood flow or embolization of an overlying thrombus/plaque material.
- Consequences of atherosclerosis: IHD, MI, stroke, ischaemic bowel disease, peripheral vascular occlusive disease & hypertension.

Normal Histology Review



Ref: Wikipedia

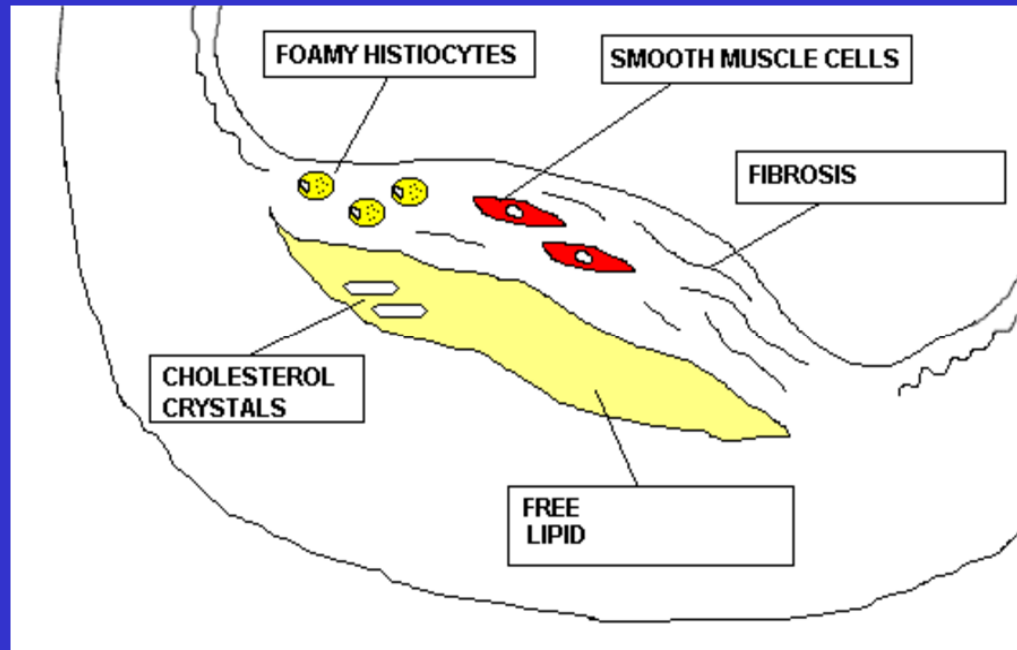


ATHEROMA: MORPHOLOGY and EFFECTS

Brian Angus

Pathology
Department

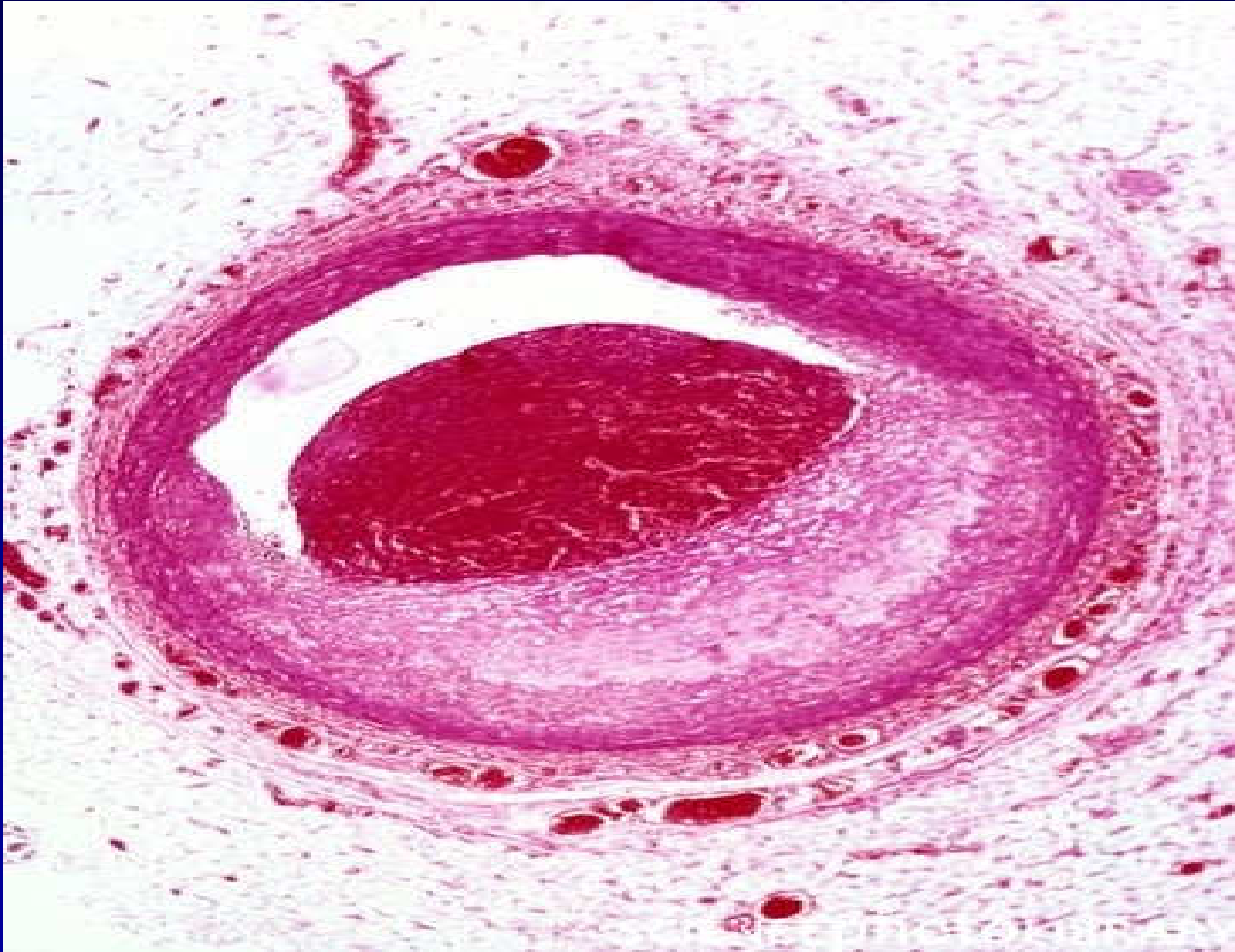
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Atheroma with intraluminal thrombus



Case 6: Atherosclerosis



Gross Pathology

The mounted specimen consists of the lower thoracic and abdominal aorta. *It shows multiple variable sized atheromatous plaques which become confluent in the abdominal aorta.* These plaques are well circumscribed, slightly raised and yellow/white in colour. Some of the larger plaques are complicated by superficial ulceration with adherent thrombus and focal dystrophic calcification.

1. history

2. macro

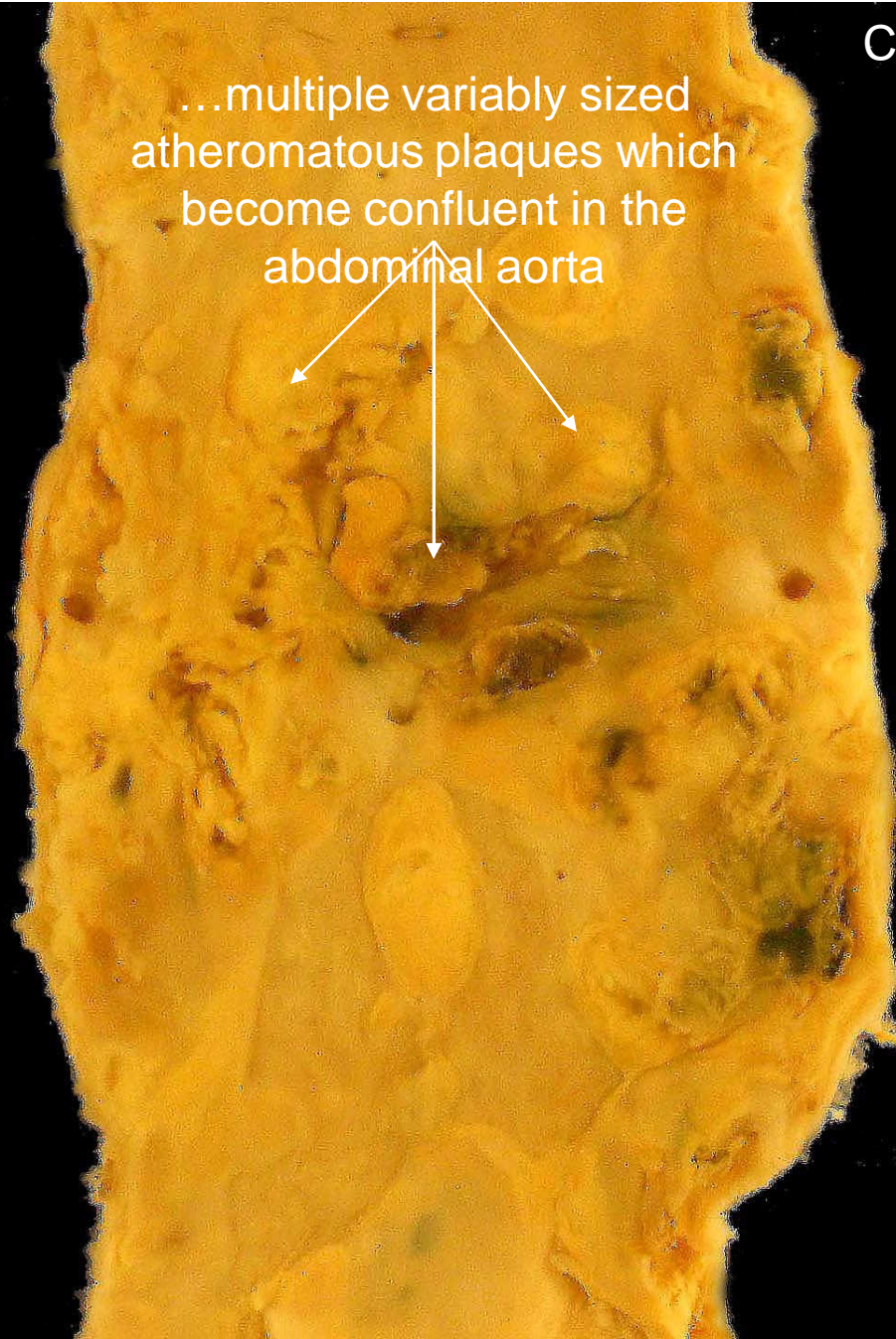
3. slide

4. micro

5. comment

Case 6: Atherosclerosis

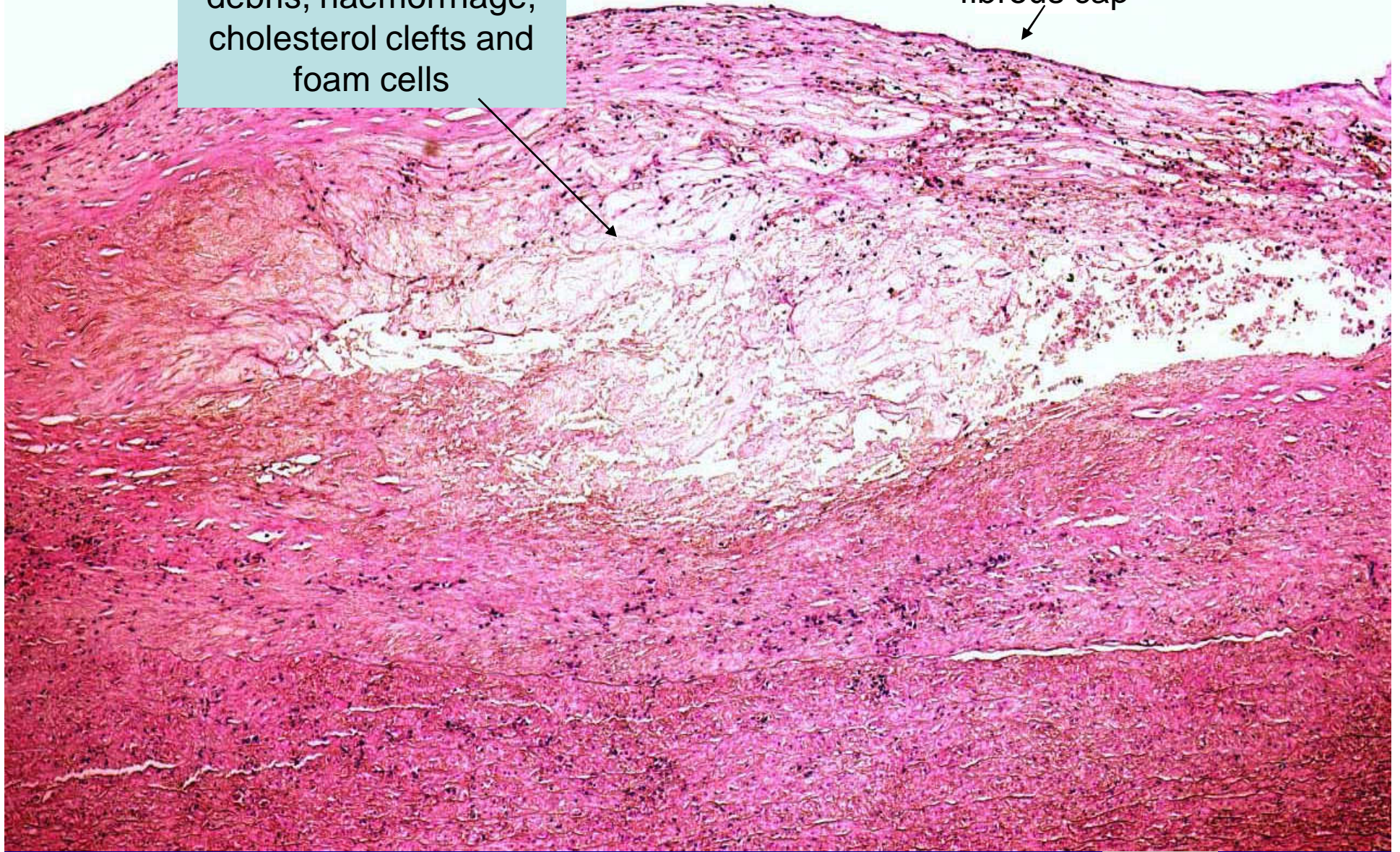
...multiple variably sized
atheromatous plaques which
become confluent in the
abdominal aorta



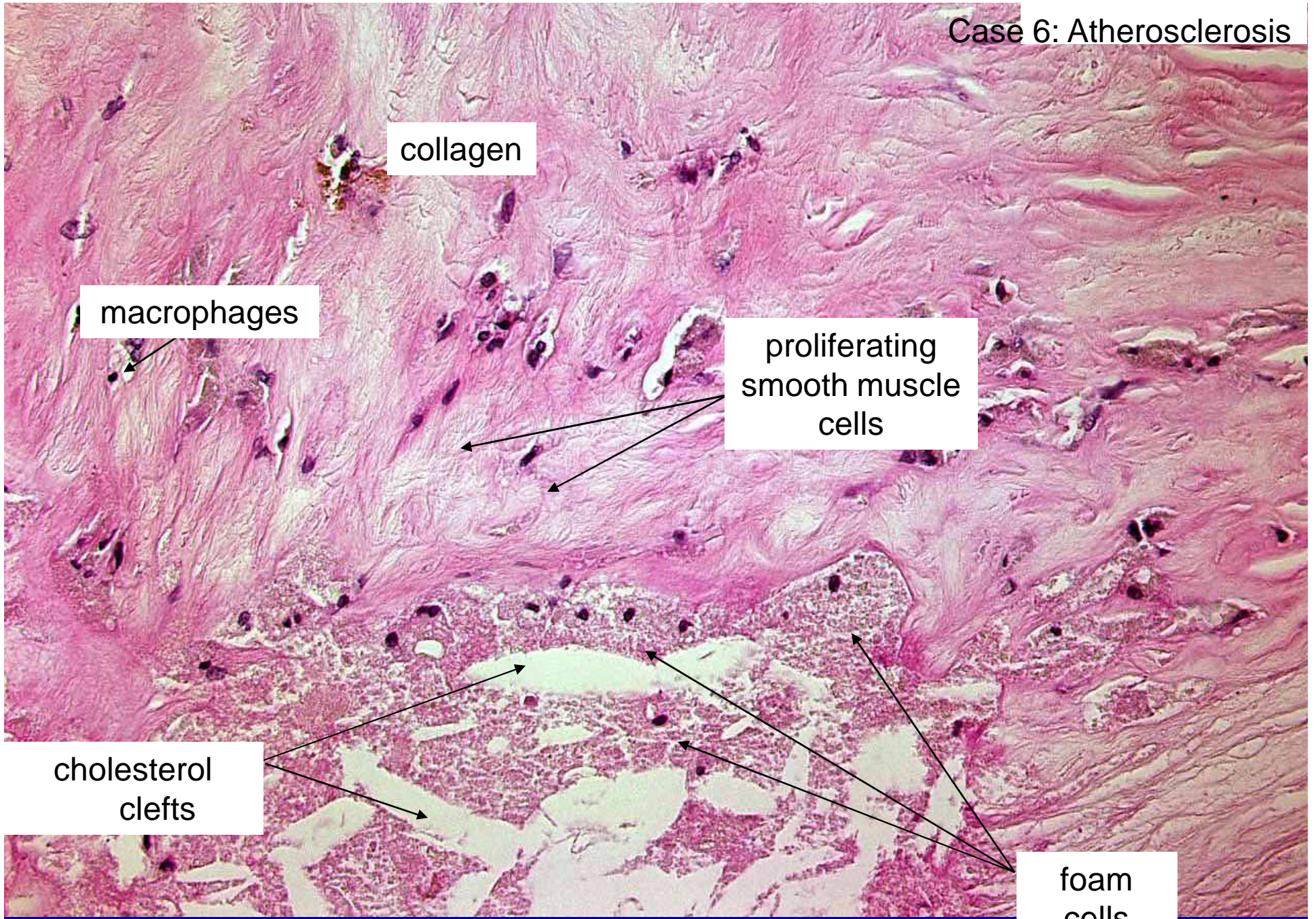
Case 6: Atherosclerosis

core of necrotic cell
debris, haemorrhage,
cholesterol clefts and
foam cells

fibrous cap



Case 6: Atherosclerosis



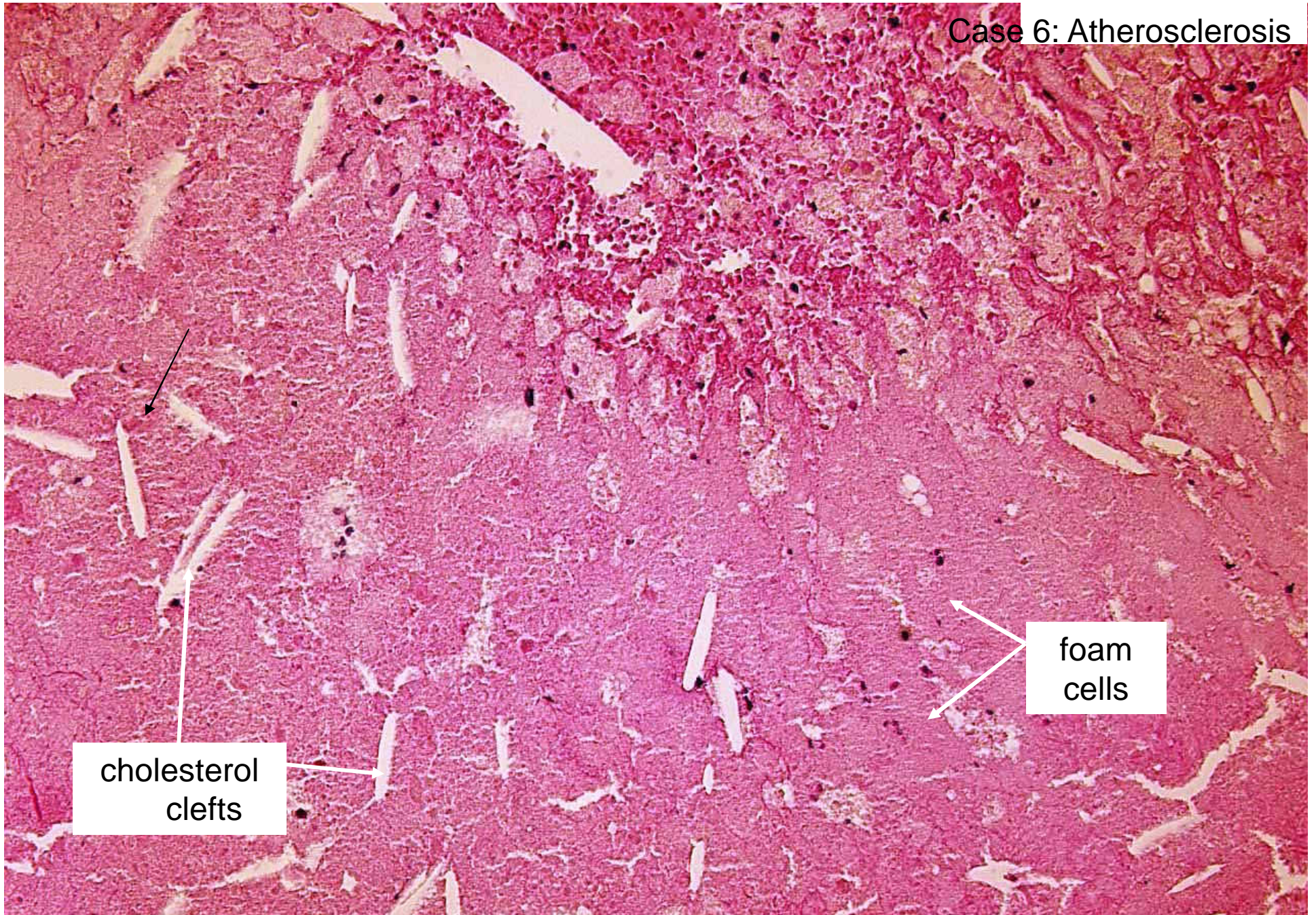
1. history

2. macro

3. slide

4. micro

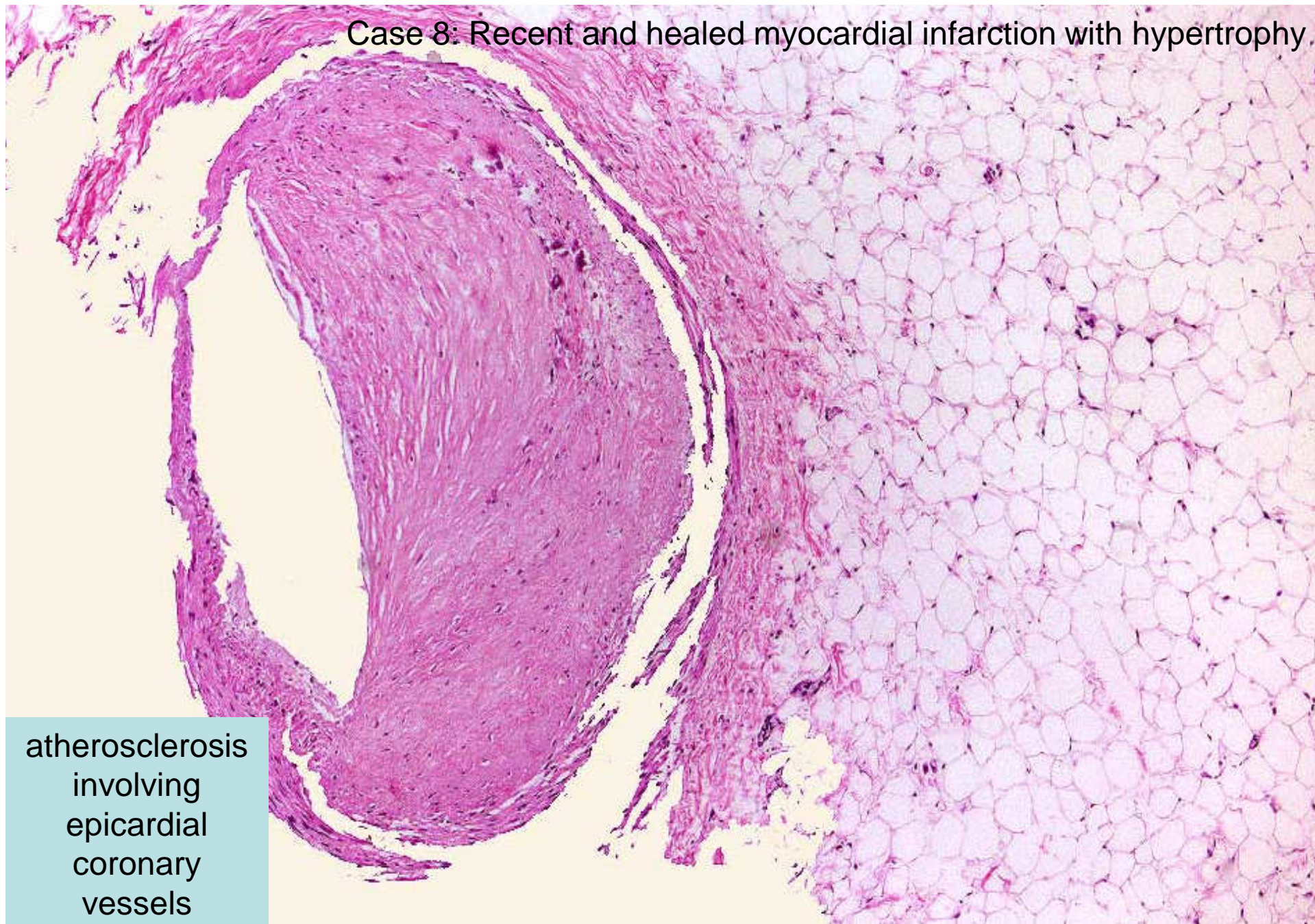
5. comment



cholesterol
clefts

foam
cells

Case 8: Recent and healed myocardial infarction with hypertrophy



atherosclerosis
involving
epicardial
coronary
vessels

1. history

2. macro

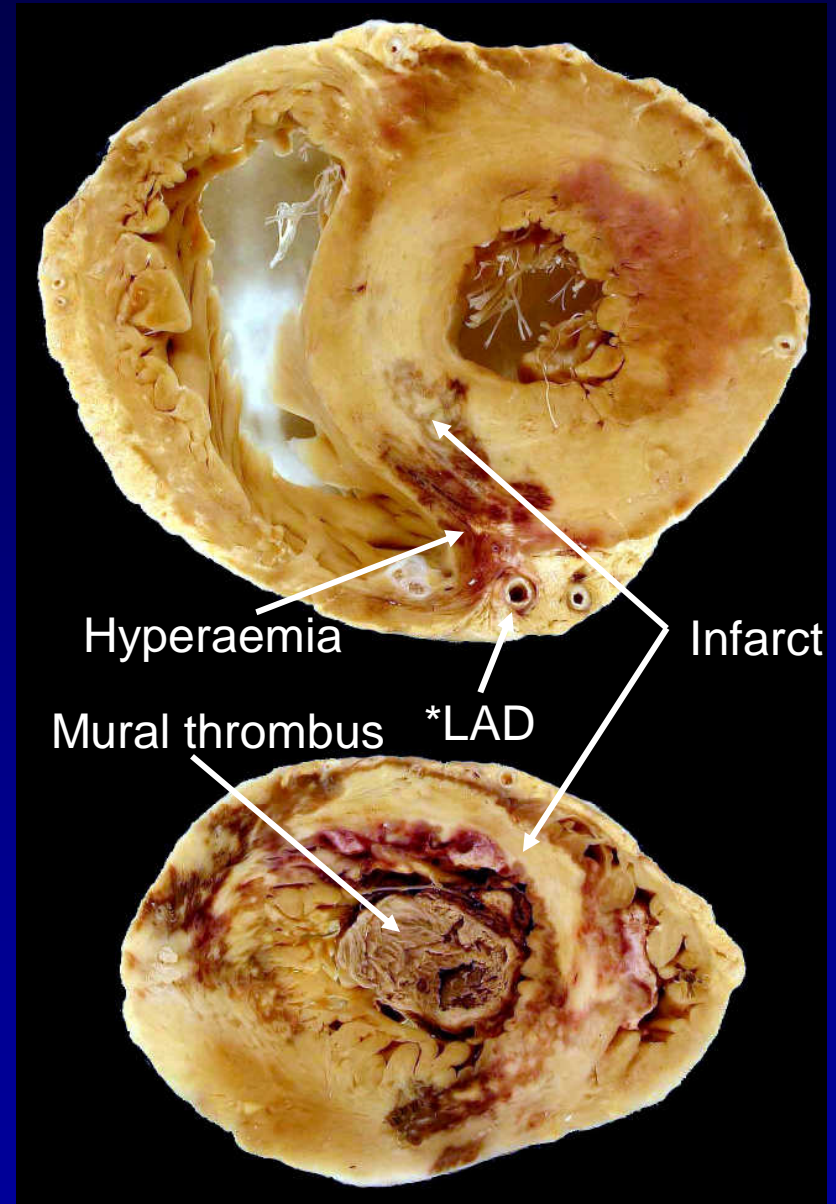
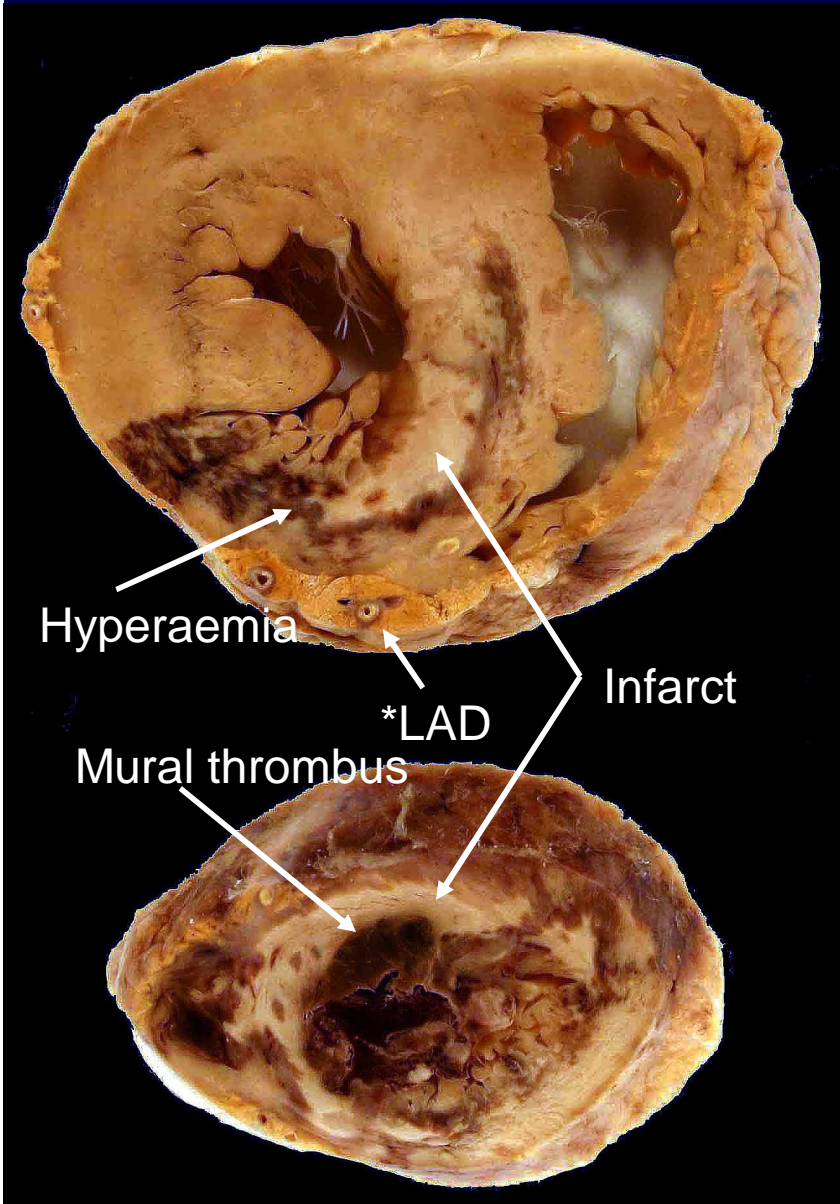
3. slide

4. micro

5. comment

7 of 8

Case 7: Recent myocardial infarction



1. history

2. macro

3. slide

4. micro

5. comment

Case 7: Recent myocardial infarction

the myocardial fibre outlines can be recognised and most fibres lack nuclei

Case 7: Recent myocardial infarction

viable and degenerate
neutrophils

the myocardial fibre
outlines can be
recognised and most
fibres lack nuclei

1. history

2. macro

3. slide

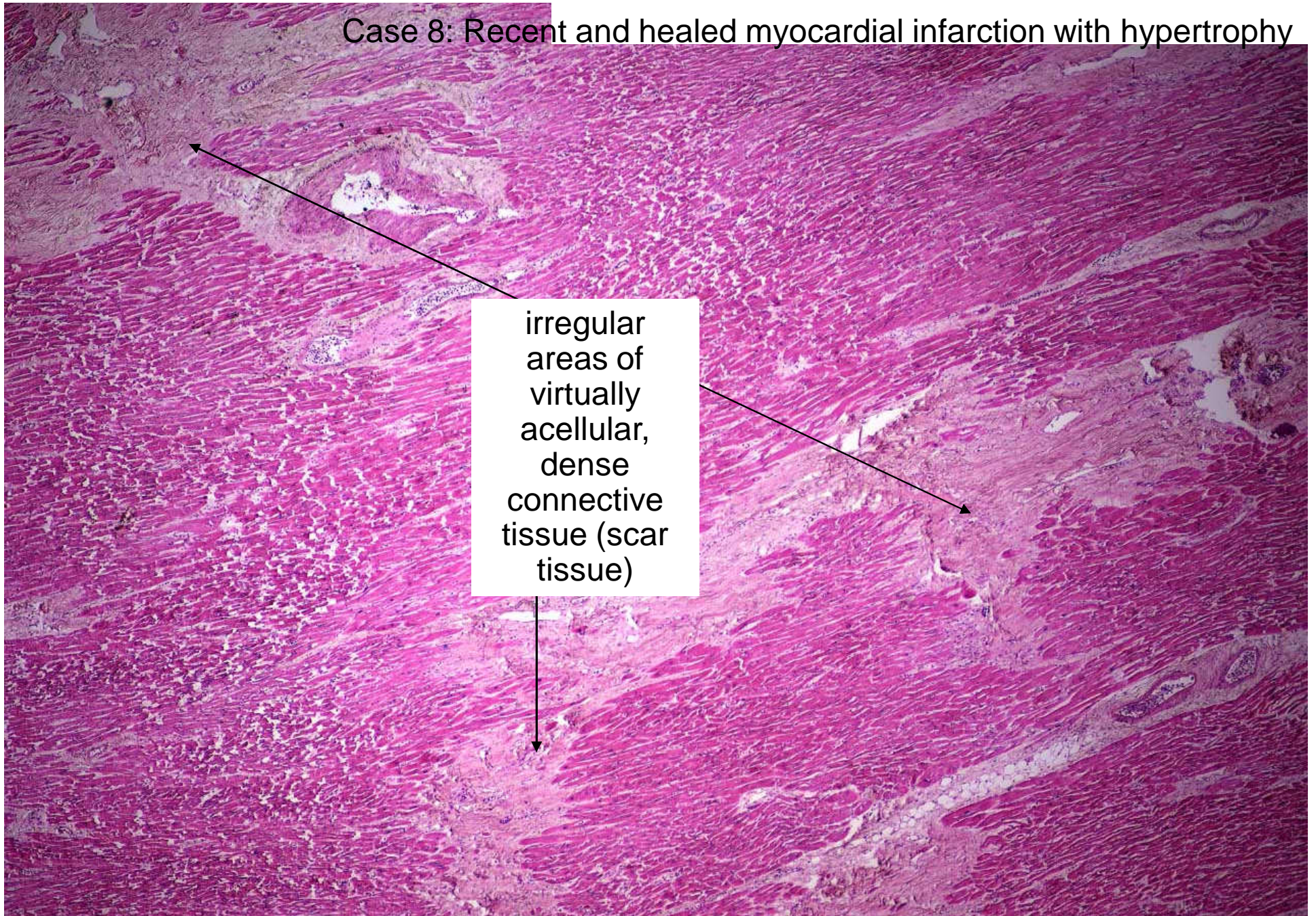
4. micro

5. comment

3

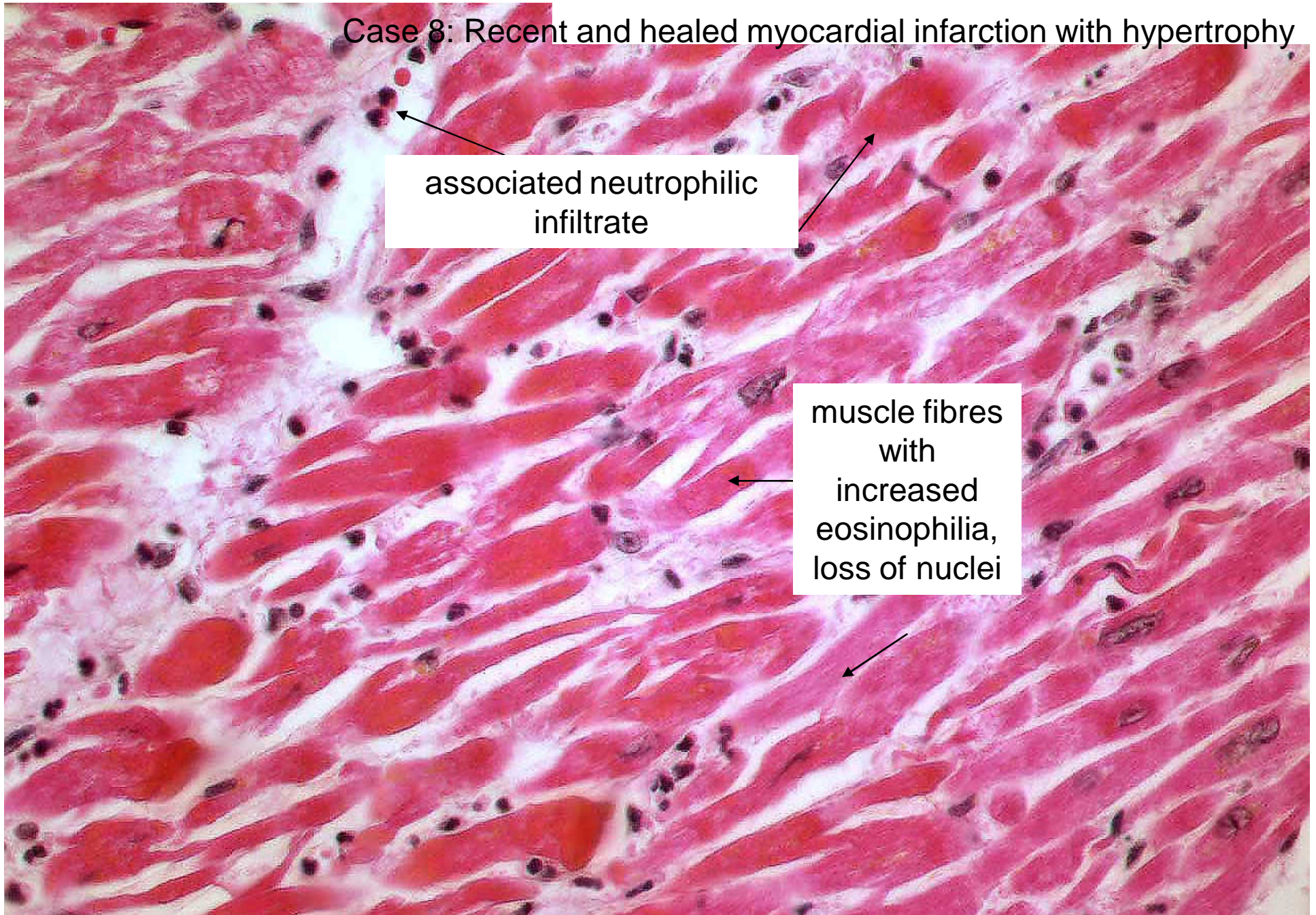
striations are still recognisable but appear fragmented and the cell cytoplasm of the necrotic cardiac fibres is more eosinophilic than the surviving fibres

Case 8: Recent and healed myocardial infarction with hypertrophy



irregular
areas of
virtually
acellular,
dense
connective
tissue (scar
tissue)

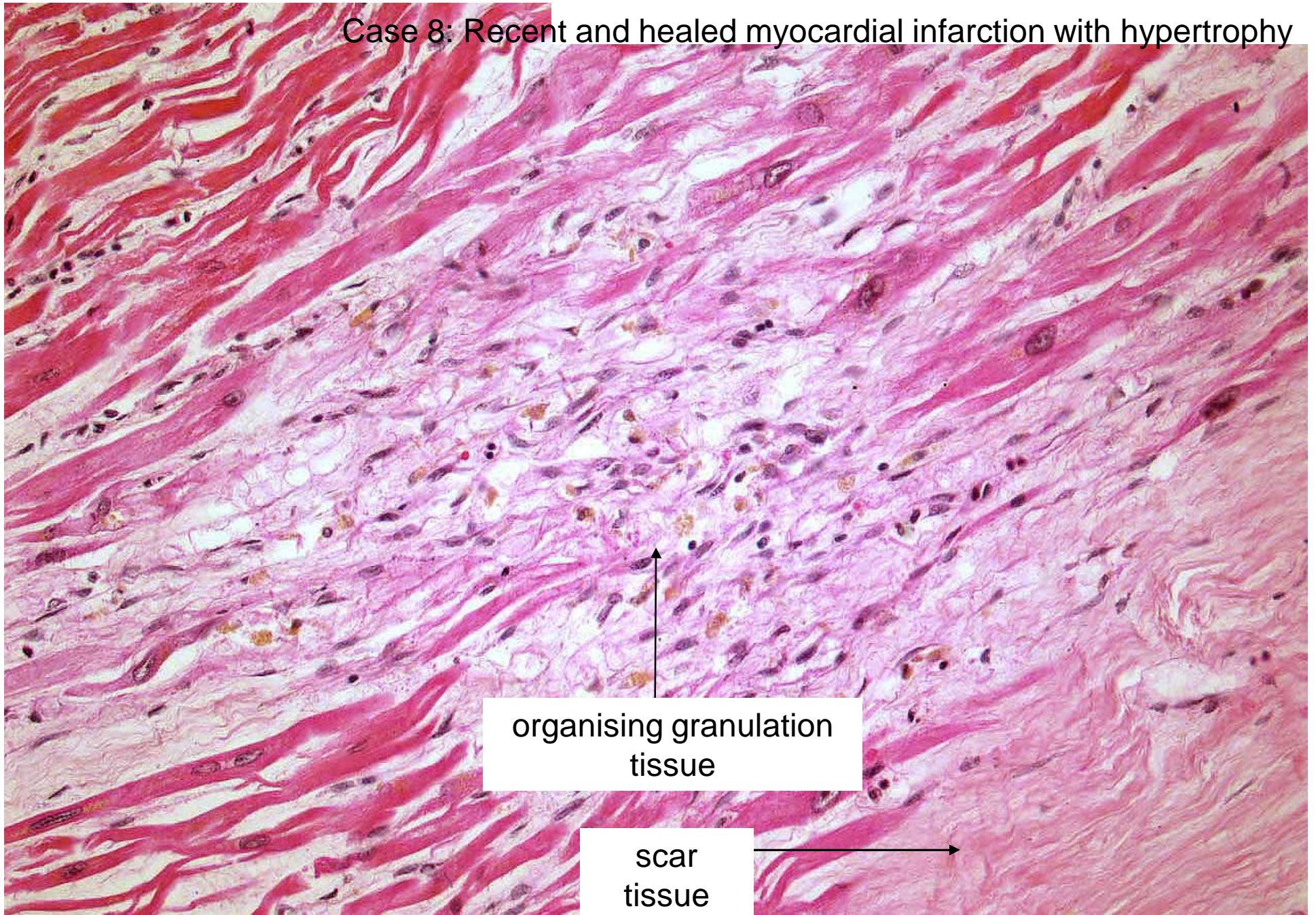
Case 8: Recent and healed myocardial infarction with hypertrophy



associated neutrophilic infiltrate

muscle fibres with increased eosinophilia, loss of nuclei

Case 8: Recent and healed myocardial infarction with hypertrophy



organising granulation
tissue

scar
tissue

1. history

2. macro

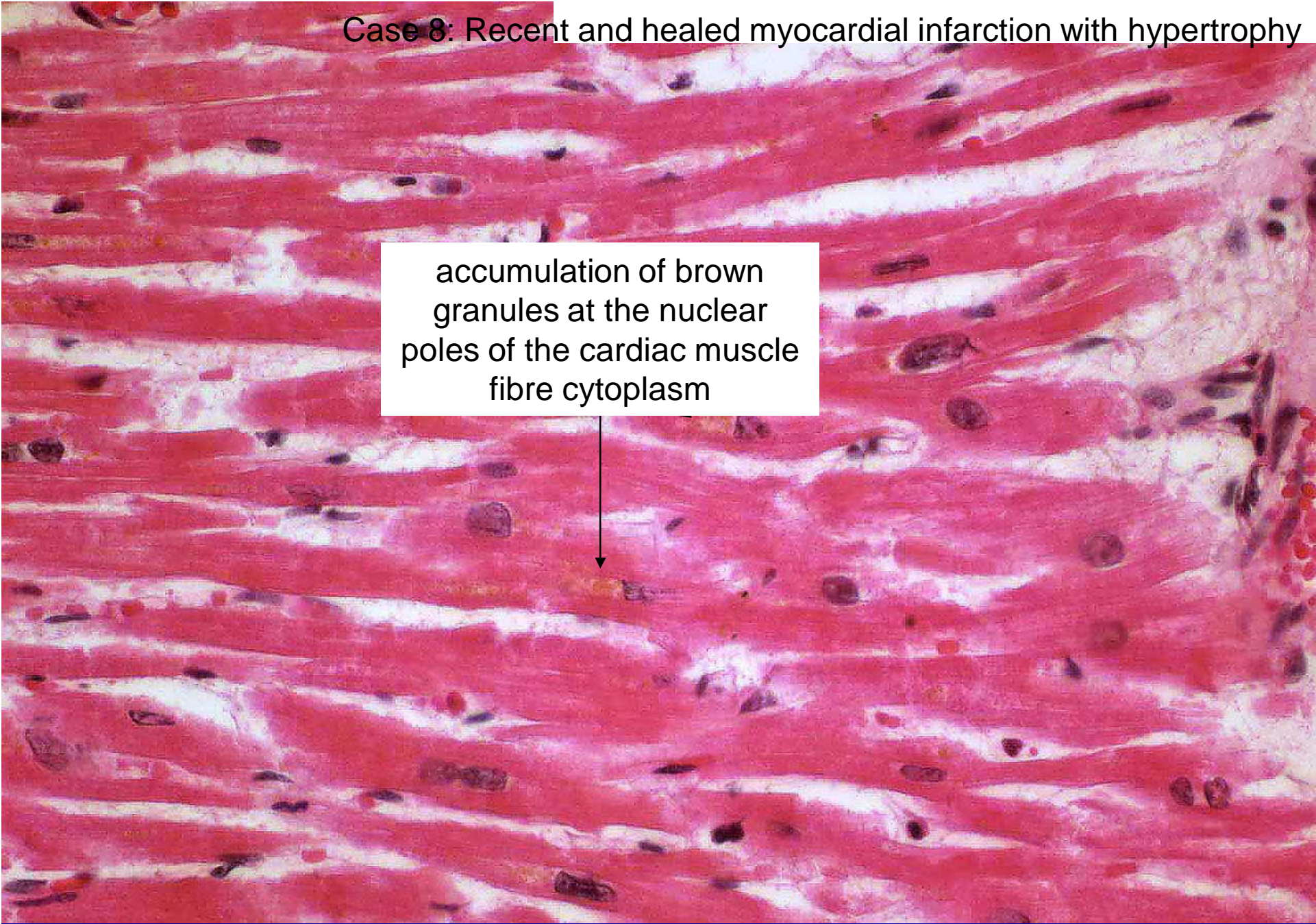
3. slide

4. micro

5. comment

Case 8: Recent and healed myocardial infarction with hypertrophy

accumulation of brown granules at the nuclear poles of the cardiac muscle fibre cytoplasm

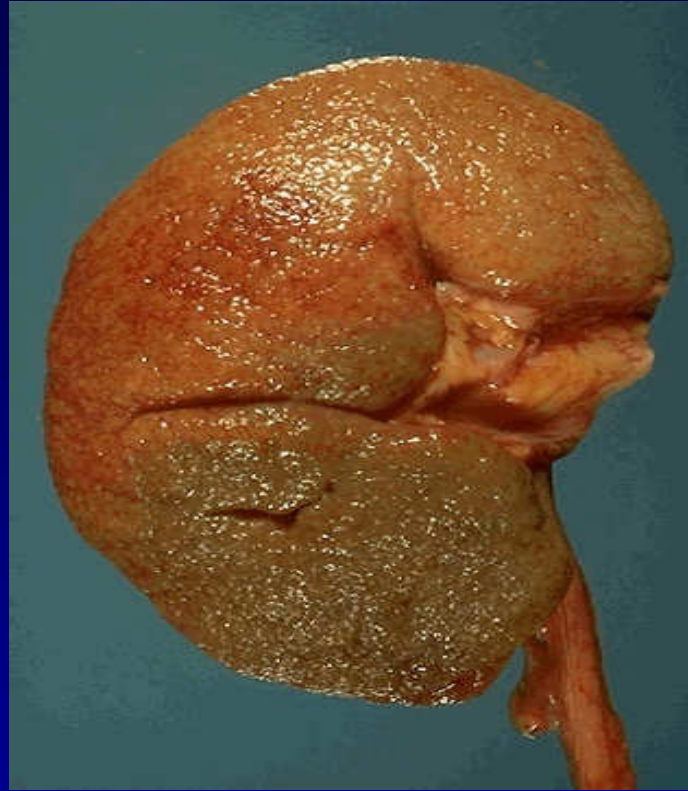


Case 8: Recent and healed myocardial infarction with hypertrophy



adipose cells are seen extending as finger-like projections between the muscle bundles

Hypertensive Nephropathy



Benign Nephrosclerosis. The smaller arteries in the kidney have become thickened and narrowed. There is patchy ischemic atrophy with focal loss of renal parenchyma that gives the surface of the kidney the **characteristic granular appearance** as seen here.

Vascular Changes in Kidneys

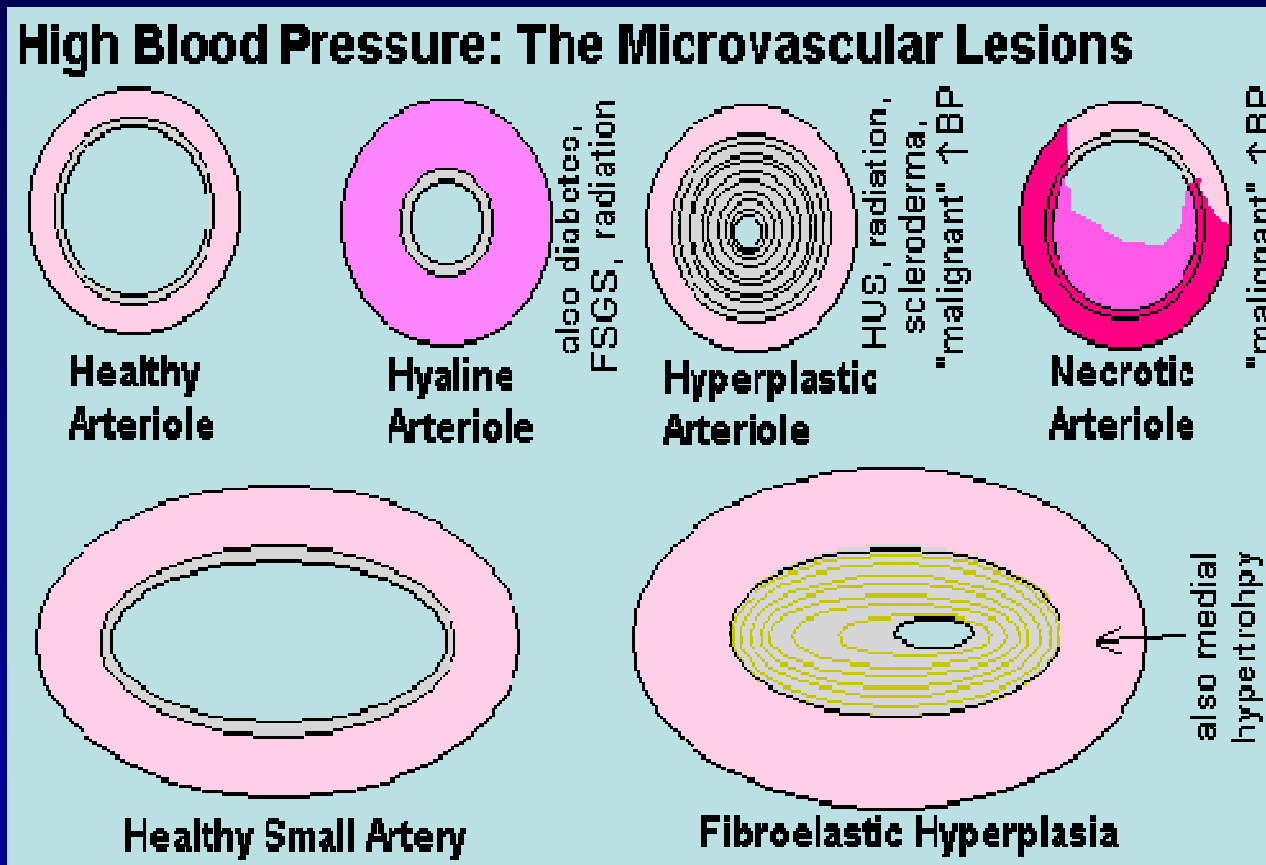
- **Hyaline arteriosclerosis:**

- Arterioles show homogenous, pink hyaline thickening with associated luminal narrowing.
- Due to increased smooth muscle cell matrix synthesis.
- Due to protein leakage across injured endothelial cells

- **Hyperplastic arteriosclerosis:**

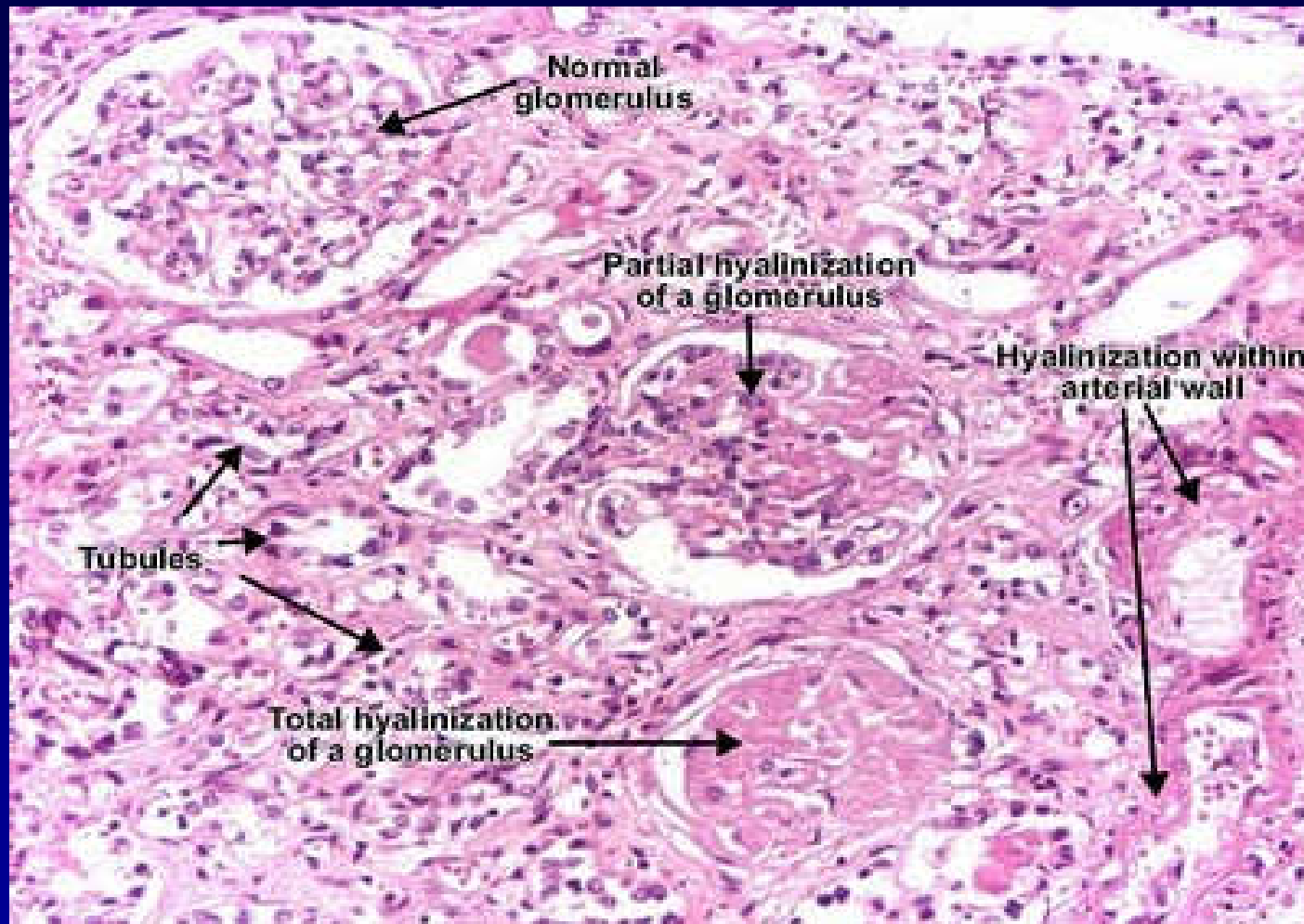
- Exhibit “onion skin lesion”.
- Concentric laminated thickening of walls & luminal narrowing.
- Laminations consists of smooth muscle cells with thickened BM

Vascular Changes in Kidney



Ref: www.pathguy.com

Benign Nephrosclerosis (Hyaline arteriosclerosis)



Recall: Hyaline refers to pink, acellular proteinaceous material. Glassy appearance.

Ref: medicalchoices.blogspot.com

Onion skin lesion

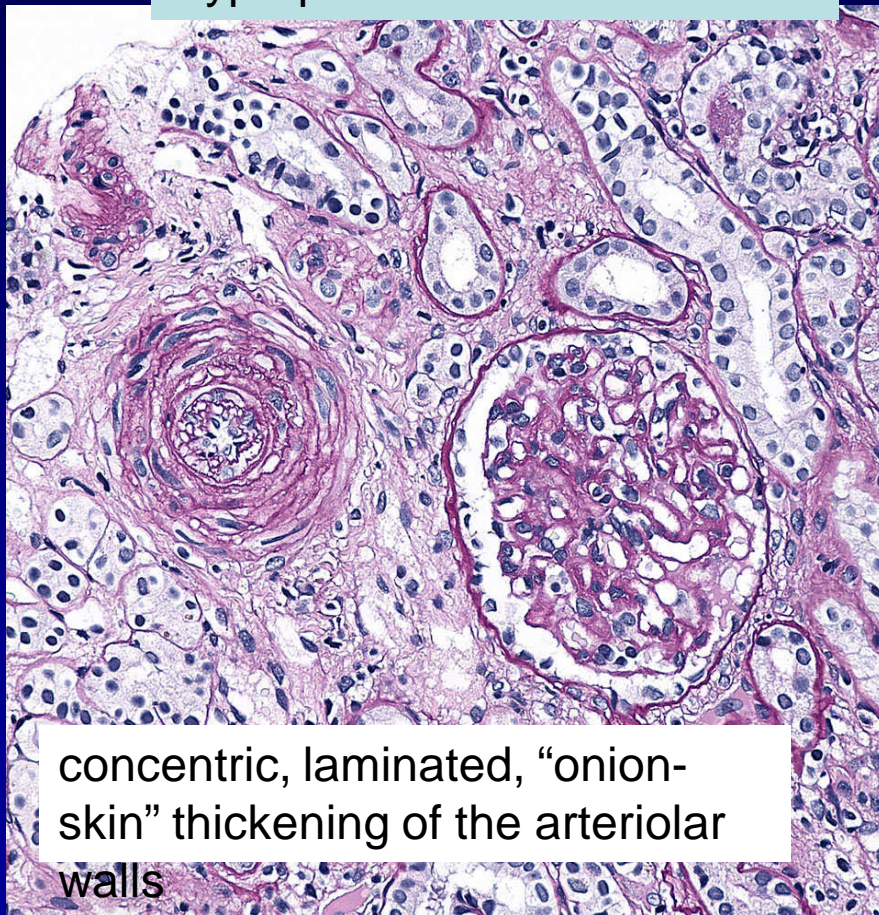


Laminated smooth muscle cells with thickened BM

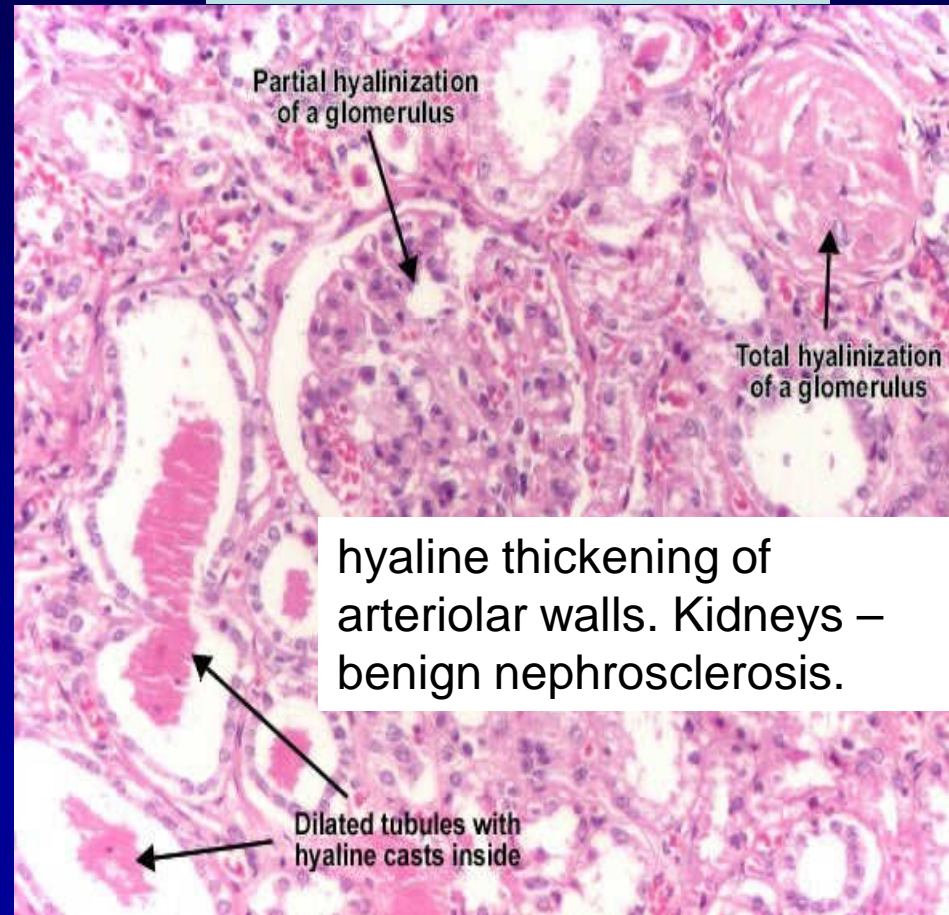
Ref: lookfordiagnosis.com

Pathological changes in hypertension: Hyperplastic Vs Hyaline Arteriosclerosis in arterioles

Hyperplastic arteriosclerosis



Hyaline arteriosclerosis



Ref: Wikipedia

Patient Evaluation Objectives

- To assess lifestyle and identify other cardiovascular risk factors or concomitant disorders that may affect prognosis and guide treatment
- To reveal identifiable causes of high BP
- To assess the presence or absence of target organ damage and CVD

(1) Cardiovascular Risk factors

- Hypertension
- Cigarette smoking
- Obesity (body mass index ≥ 30 kg/m²)
- Physical inactivity
- Dyslipidemia
- Diabetes mellitus
- Microalbuminuria or estimated GFR < 60 mL/min
- Age (older than 55 for men, 65 for women)
- Family history of premature cardiovascular disease (men under age 55 or women under age 65)

(2) Identifiable Causes of HTN

- Sleep apnea
- Drug-induced or related causes
- Chronic kidney disease
- Primary aldosteronism
- Renovascular disease
- Chronic steroid therapy and Cushing's syndrome, other endocrine disorders
- Pheochromocytoma
- Coarctation of the aorta
- Thyroid or parathyroid disease

Laboratory Work Up

- **ECG/Echo.**
- **Urinalysis.**
- **Blood glucose and hematocrit; serum potassium, creatinine (or estimated GFR), and calcium.**
- **HDL cholesterol, LDL cholesterol, and triglycerides.**
- **Optional tests**
 - urinary albumin excretion.
 - albumin/creatinine ratio.

End

References:

Robins Pathological Basis of Diseases
Interactive Pathology CD, UTAS.

Seminar notes available at:

www.pathologyatsmhs.com