BLOOD VESSEL DISEASES

Blood vessels have a primary function of nourishing various organs and tissues of the body by supplying them with blood.

Vascular diseases are manifested clinically through three mechanisms that are reflected through the diseases vessel (s) as:

- Progressive narrowing of the lumen associated with progressive ischemia of the relevant tissues.
- Thrombosis associated with partial or complete luminal obstruction and/or embolism.
- Aneurysmal dilatation that may eventuate in rupture with ischemic and destructive consequences.

ARTERIOSCLEROSIS

Definition

Variants

This generic term refers to a group of disorders having in common *thickening and loss of elasticity of arterial walls* and thus leading to sclerosis i.e. hardening of the wall.

There are three morphologic variants of arteriosclerosis that come under this generic term, what are they?

1. Atherosclerosis (the most frequent and important type)
2. Medial calcific sclerosis
3. Arteriolosclerosis

ATHEROSCLEROSIS

- This disease is responsible for more deaths and serious complications than any other disorder.
- This is because its prime targets are vital arteries, namely the coronaries, cerebral arteries, & the aorta.
- Accordingly the major consequences are

1. Myocardial infarction
2. Cerebral infarction
3. Aortic aneurysm

- **Myocardial infarction alone is responsible for about 25% of all deaths.**
- **By definition atherosclerosis is "*a disease primarily of large elastic arteries and medium sized muscular arteries.***
- ***Its basic lesion is the atheroma (fibro-fatty plaque), which is a raised patch within the intima having a core of lipid (mainly cholesterol and its esters) and a cap of fibrous tissue".***
- **Examples of large elastic arteries are aorta, carotid, and the iliac arteries, & examples of medium-sized muscular arteries are the coronaries and popliteal arteries.**

Epidemiology and risk factors

- Epidemiological data and that of the risk factors of atherosclerosis are expressed largely in terms of the incidence of deaths caused by ischemic heart disease (IHD).
- This is because atherosclerosis does not by itself produce signs and symptoms but its prevalence is detected by its effects on the most commonly involved arteries, namely the coronaries.

Epidemiological findings include

- A decline in the incidence of deaths from IHD that has started since 1975. This is due to the recognition and avoidance of the risk factors, better recognition and better management.
- Marked geographical variations in the incidence of atherosclerosis-related IHD. It is high in Europe and the USA but remarkably low in Asia (for e.g. the mortality rate from IHD is six times higher in the USA than that in Japan). This is probably related to differences in the life style and dietary customs.

The following increase the risk of atherosclerosis

a. Increasing age as death rates from IHD rise with each decade of life.

b. Male gender since myocardial infarction is particularly uncommon in premenopausal women.

c. Certain genetic defects: certain families suffer increased frequency of heart attacks at an early age. This familial predisposition appears to be related to hyperlipidemia (due for e.g. to genetic defects in lipoprotein metabolism), hypertension, and diabetes mellitus. It should be noted that both hypertension and diabetes also tend to be familial.

- **Risk factors that predispose to atherosclerosis and the resultant IHD can be divided into two main groups:**

Major Risk factors of atherosclerosis

A. Potentially modifiable (controllable)

1. Diet and hyperlipidemia
2. Hypertension
3. Cigarette smoking
4. Diabetes mellitus

B. Nonmodifiable

1. Increasing age
2. Male gender
3. Family history
4. Genetic abnormalities

Minor (uncertain risks)

1. Obesity
2. Physical inactivity
3. Stress (type A personality)
4. High carbohydrate intake
5. Lipoprotein (a)
6. Hardened unsaturated fat intake
7. Chlamydia pneumonia
8. Hyperhomocystinemia

Diet and Hyperlipidemia

Hyperlipidemia (particularly hypercholesterolemia) and other abnormalities in lipid metabolism are major risk factors in atherosclerosis.

The evidences linking hypercholestrolemia to atherosclerosis include the following:

1. **Atherosclerotic plaques are rich in cholesterol and its esters. These are largely derived from lipoproteins of the blood.**
2. **Atherosclerotic lesions can be induced in experimental animals by feeding them diets that raise their plasma cholesterol levels.**
3. **Genetic disorders that cause severe hypercholesterolemia lead to premature atherosclerosis, often fatal in childhood.**
4. **Acquired diseases associated with hypercholesterolemia (as part of their manifestations) for e.g. nephrotic syndrome and hypothyroidism, are associated with increasing risk of atherosclerosis**
5. **Populations having relatively high levels of serum cholesterol show higher mortality from IHD.**
6. **Treatment with diet and cholesterol-lowering drugs reduces cardiovascular mortality in patients with hypercholesterolemia.**

- High dietary intake of cholesterol and saturated fats, e.g. those present in egg yolk, animal fats, and butter, raises plasma cholesterol level.
- The higher the level of serum cholesterol the higher the risk particularly, with levels exceeding 200 mg/dl.
- The most striking association is with elevated levels of low-density lipoprotein (LDL). This is the lipoprotein moiety richest in cholesterol.
- In fact the major component of the total serum cholesterol associated with increased risk is LDL.
- Hypertriglyceridemia also appears to increase the risk.
- In contrast, serum levels of high-density lipoprotein (HDL) are inversely related to the risk. It is believed that HDL mobilizes cholesterol from developing atheromas and transports it to the liver to be eventually excreted into the bile.
- Exercise also raises the HDL level, whereas obesity and smoking lower it.

- Hypertension is a major risk factor at all ages.
- Elevated blood pressure accelerates the process of atherosclerosis and increases the incidence of IHD and cerebrovascular diseases.
- Men over the age of 45 years with a blood pressure exceeding 170/95 mm Hg have more than five-fold greater risk of IHD than normotensives.
- Antihypertensive therapy reduces the incidence of atherosclerosis-related diseases, particularly IHD and CVA (cerebrovascular accidents; strokes).

- Cigarette smoking is a well-established risk factor.
- It is the most important avoidable cause of IHD.
- Cigarette smoking is the main cause responsible for the relatively recent increase in the incidence and severity of atherosclerosis in women.
- In the context of IHD, two facts are related to smoking

a. It increases the incidence of sudden death among those with IHD

b. Cessation of smoking in high-risk individuals is followed within a few years by a reduction in the risk of dying from IHD.

Diabetes mellitus: diabetics are more susceptible, compared with nondiabetics to atherosclerosis-related diseases and in particular **IHD**, **cerebrovascular accidents (CVA) and gangrene of lower extremities.** This is probably related to

1. Hyperlipidemia, which is seen in up to 50% of diabetics
2. Increased platelets adhesiveness; predisposing to thrombotic episodes.
3. Some diabetics tend to be obese and hypertensive; thus have increased tendency to develop severe atherosclerosis.

All diabetics who have had the disease for at least ten years, irrespective of the age of onset, are likely to develop clinically significant atherosclerosis.

P23

Enumerate, in descending order of severity, the most heavily involved arteries in the body by atherosclerosis.

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P23

dr.hassan 24/11/2003

- The most heavily involved arteries by atherosclerosis and in descending order are

1. Abdominal aorta
2. Coronaries
3. Popliteal arteries
4. Descending thoracic aorta
5. Internal carotid arteries
6. Arteries forming the circle of Willis at the base of the brain.

- Atheromatous plaques are patchy in distribution and may involve the arterial wall in asymmetrical fashion i.e. involve one portion of the wall circumference more severely than elsewhere and as such produce eccentric lesions.

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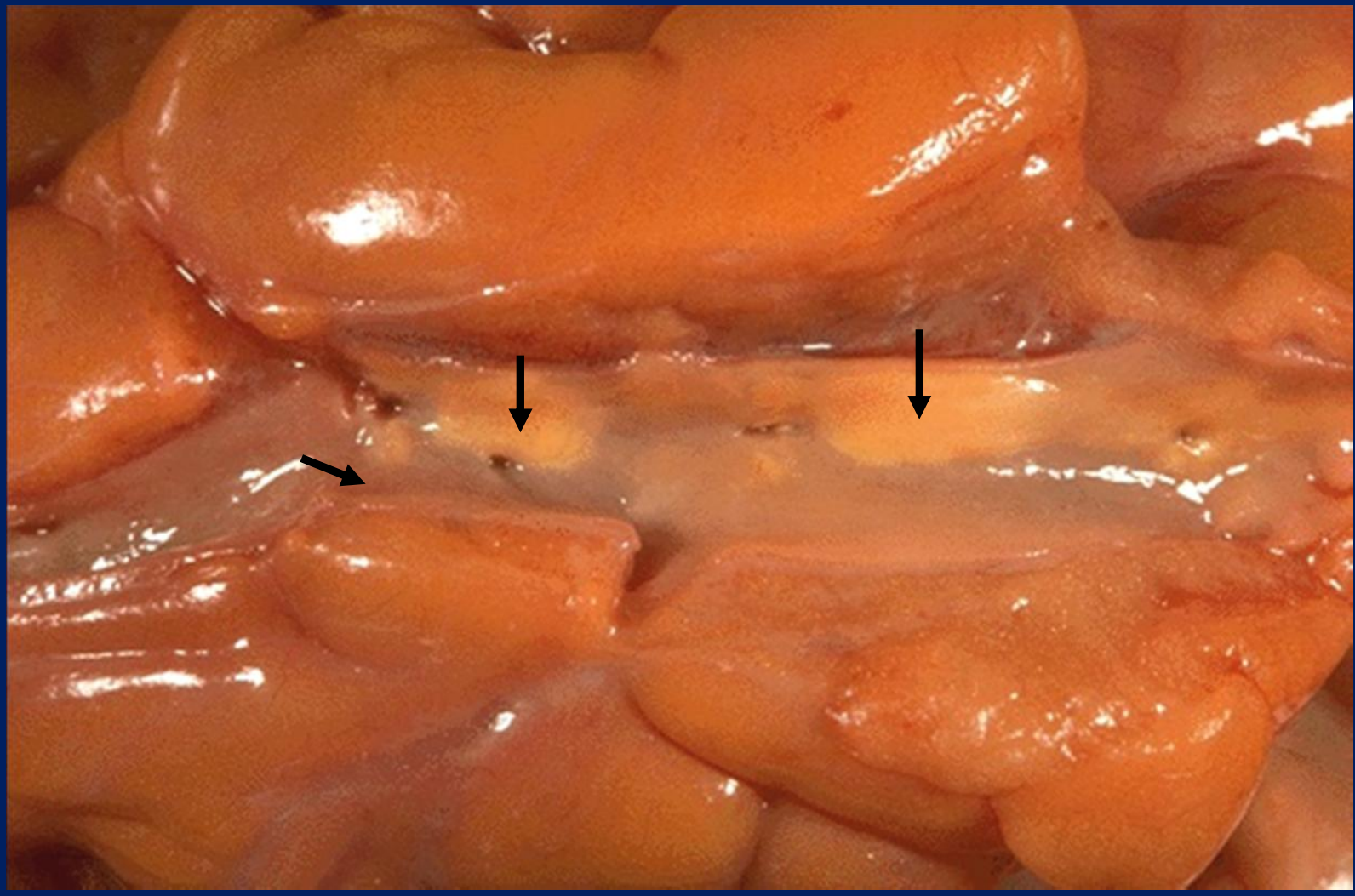
Gross features of atherosclerosis

The basic lesion in atherosclerosis is a focal intimal thickening termed *atheromatous plaque* or fibro-fatty plaque.

Each plaque is white to whitish yellow elevation up to 1.5 cm in diameter; adjacent plaques, however, may fuse to form larger plaques.

The superficial portion of these lesions (i.e. facing the lumen) tends to be firm and white; this is the fibrous cap, whereas the deep portion is yellow and soft and represents the lipid component. It is from this yellow soft debris, the term atheroma is derived (Greek word for gruel).

Mild degree of coronary athersclerosis



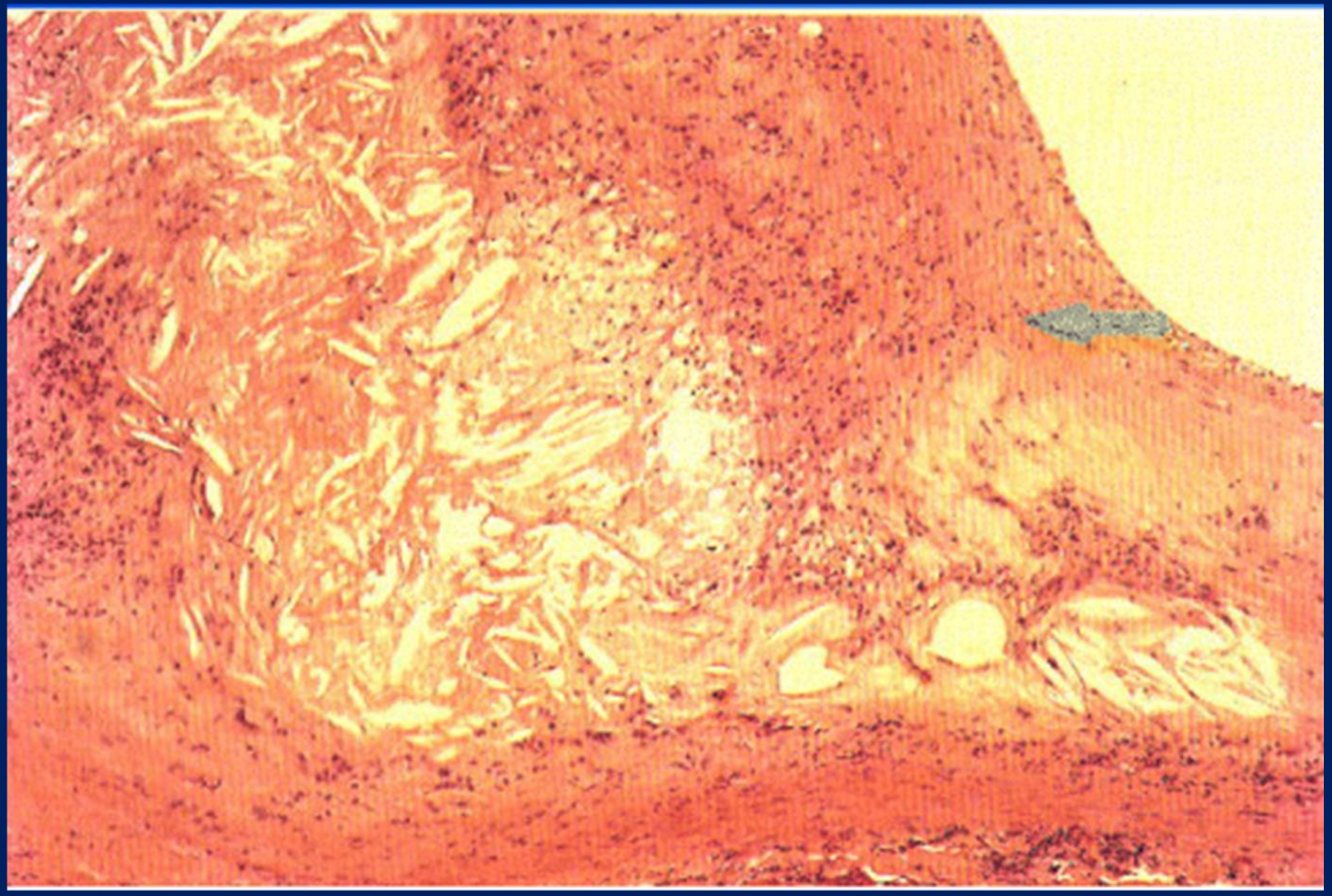
A coronary artery has been opened longitudinally. The coronary extends from left to right across the middle of the picture and is surrounded by epicardial fat. This coronary shows only mild atherosclerosis, with only an occasional yellow-tan lipid plaques (arrows) and no narrowing.

Microscopic features

- The superficial cap is composed of smooth muscle cells and relatively dense collagen fibers.
- Just beneath and to the sides of the cap there is a cellular area made up of variable mixture of macrophages, smooth muscle cells and T-lymphocytes.
- Deep to the cellular area is a necrotic core; consisting of lipid material, cholesterol clefts, cellular debris and lipid-laden cells Foam cells
- Finally, especially around the edges of the lesion there are proliferating small, thin-walled blood vessels.
- The above mentioned components may occur in varying proportions in different plaques, for e.g. some plaques may be composed mostly of smooth muscle cells and fibrous tissue (fibrous plaques).

P1

This is a microscopic section through an atheromatous plaque. Describe.



P1

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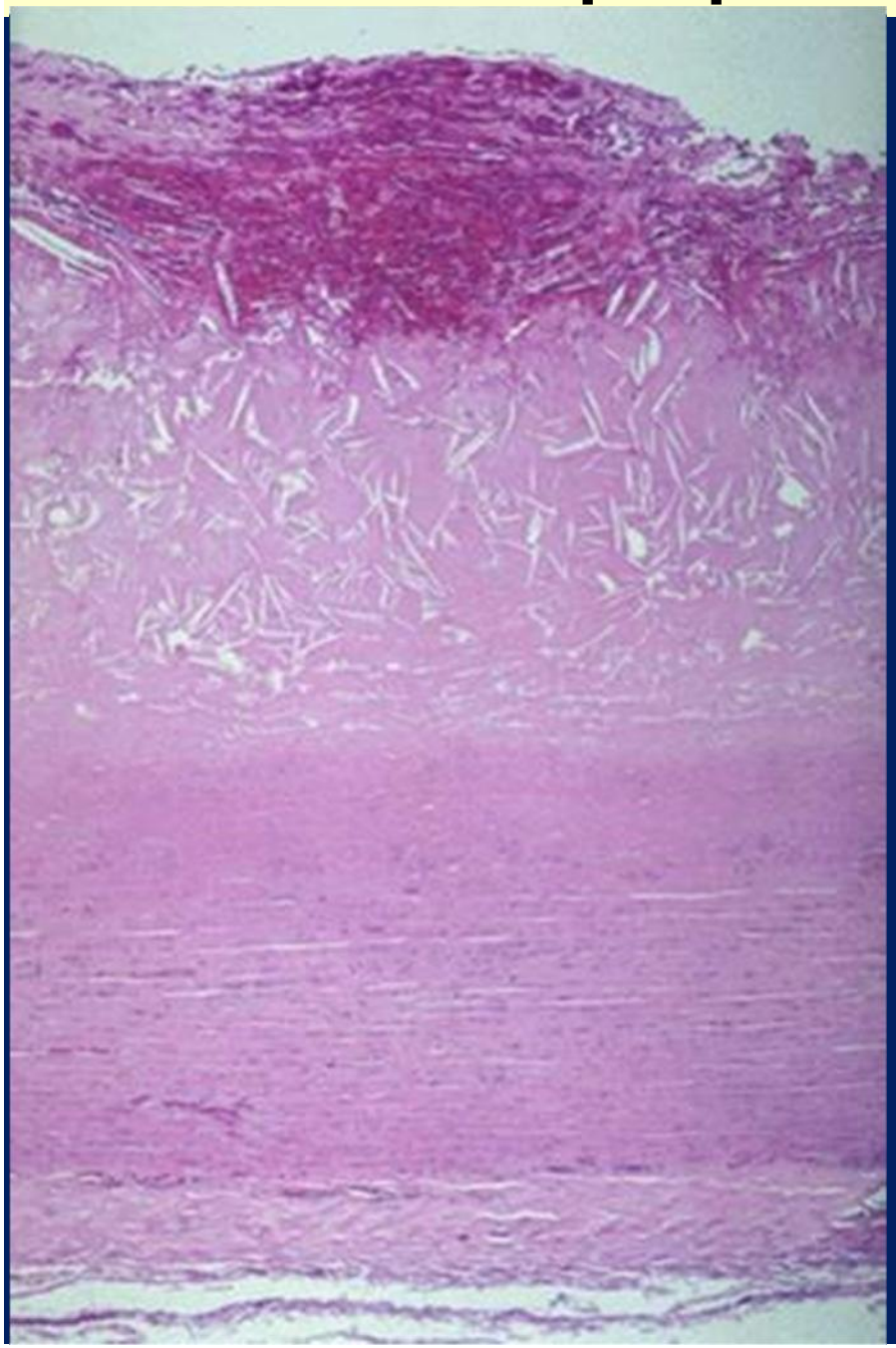
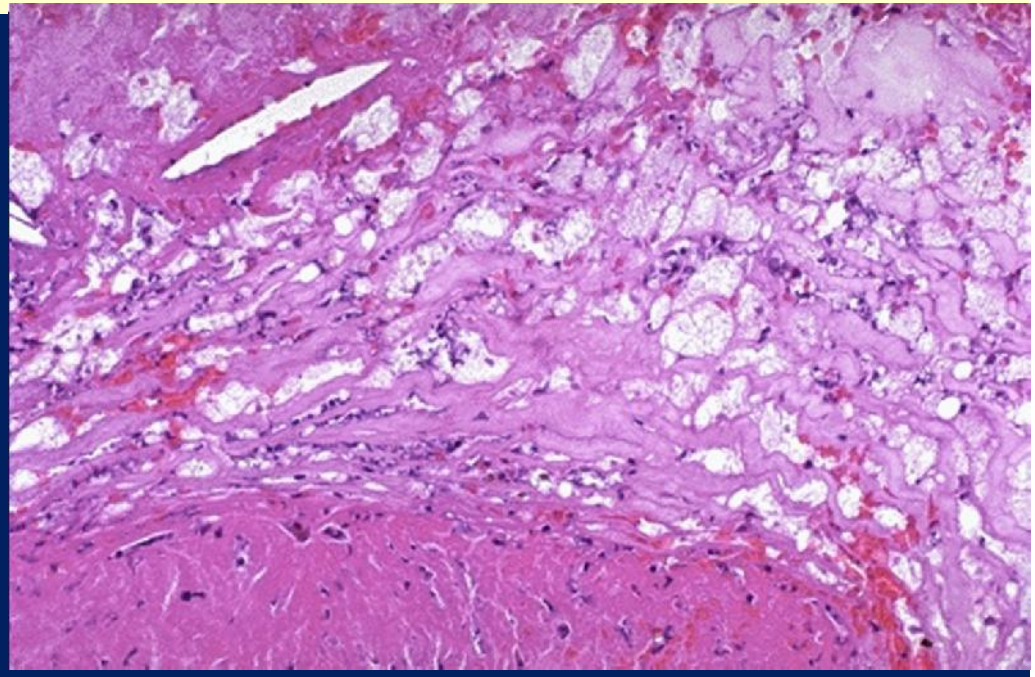
Microscopic features

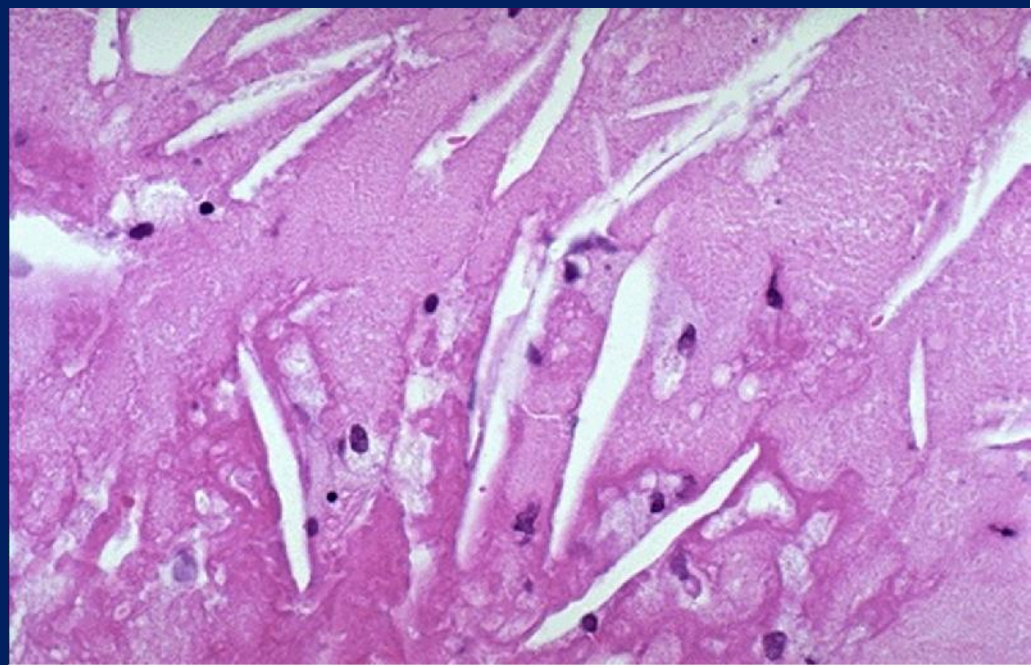
- The superficial cap is composed of smooth muscle cells and relatively dense collagen fibers.
- Just beneath and to the sides of the cap there is a cellular area, which consists of a mixture of macrophages, smooth muscle cells and T-lymphocytes.
- Deep to the cellular area is a necrotic core; consisting of lipid material, cholesterol clefts, cellular debris and lipid-laden foam cells. The lipid is primarily cholesterol and its esters.
- Finally, especially around the edges of the lesion there are proliferating small, thin-walled blood vessels.
- The above mentioned components may occur in varying proportions in different plaques, for e.g. some plaques may be composed mostly of smooth muscle cells and fibrous tissue (fibrous plaques).

P2

Three microscopic pictures LP, MP & HP. Describe







P2

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Lt.
This microscopic cross section of the aorta shows a large overlying atheroma on the left. Cholesterol clefts are numerous in this atheroma. The surface on the far left
shows ulceration and hemorrhage. Despite this ulceration, atheromatous emboli are rare (or at least, complications of them are rare).
Rt upper
dr.hassan 30/11/2003
NABEEL9/28/2004
This high magnification of the atheroma shows numerous foam cells and an occasional cholesterol cleft. A few dark blue inflammatory cells are scattered within the
atheroma.
Rt. lower
dr.hassan 30/11/2003
NABEEL9/28/2004
This is a high magnification of the aortic atheroma with foam cells and cholesterol clefts.
Pathology; 20/09/2009

The complication that may affect atheroma

***1-Focal rupture, ulceration, or erosion of the luminal surface*. This results in exposure of highly thrombogenic substances that induce thrombus formation. Alternatively, the fatty debris present within the core may be discharged into the blood stream producing microemboli (atheroemboli or cholesterol emboli).**

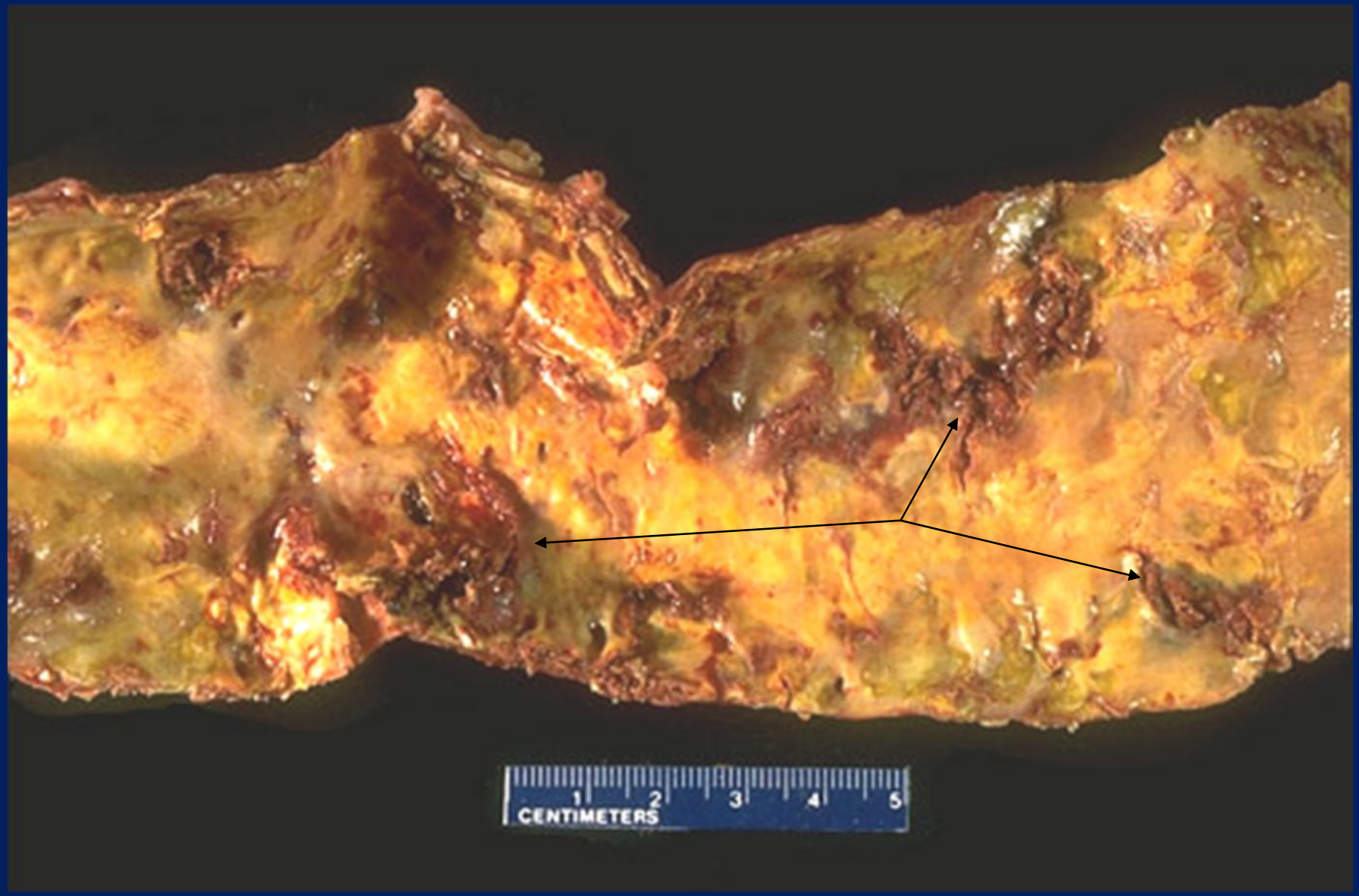
***2-Hemorrhage in to the plaque* which is especially seen in the coronaries, either from rupture of the fibrous cap or rupture of the thin-walled capillaries that vascularise the atheroma.**

3-Superimposed thrombosis **is the most serious complication that usually occurs on disrupted lesions (ruptured, ulcerated, eroded lesions or those with intralesional hemorrhage). Thrombi may partially or completely occlude the lumen. They may become incorporated into the athermatous plaque, enlarging it by subsequent organization.**

4-Aneurysmal dilatation**; in severe cases, particularly in large arteries such as the aorta, the underlying media undergoes pressure or ischemic atrophy with loss of elastic fibers. This may cause sufficient weakness that allows for aneurysaml dilatation.**

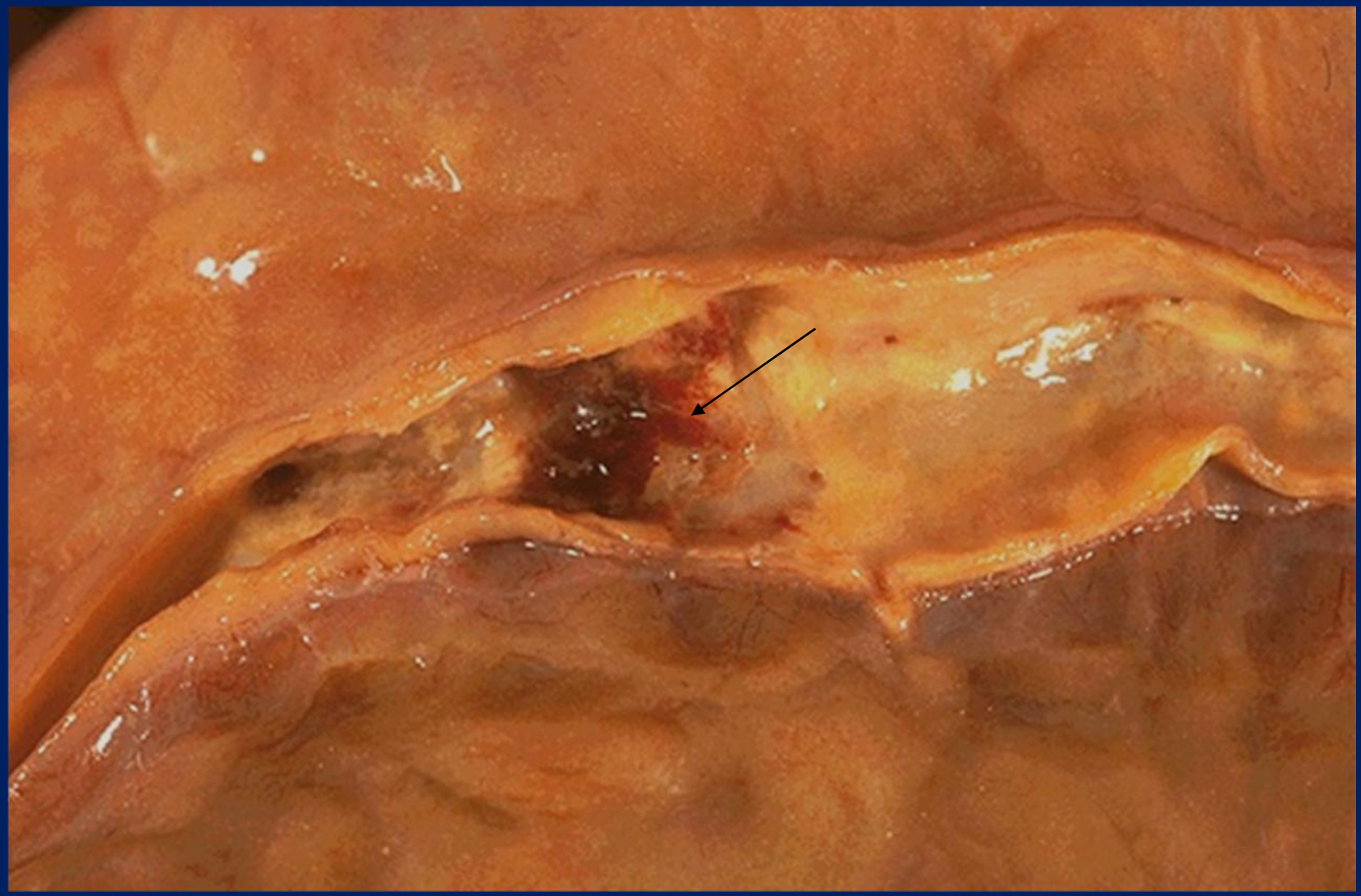
5-Calcification**, this may be patchy or massive.**

Atherosclerosis aorta: ulcerations with superadded thrombosis



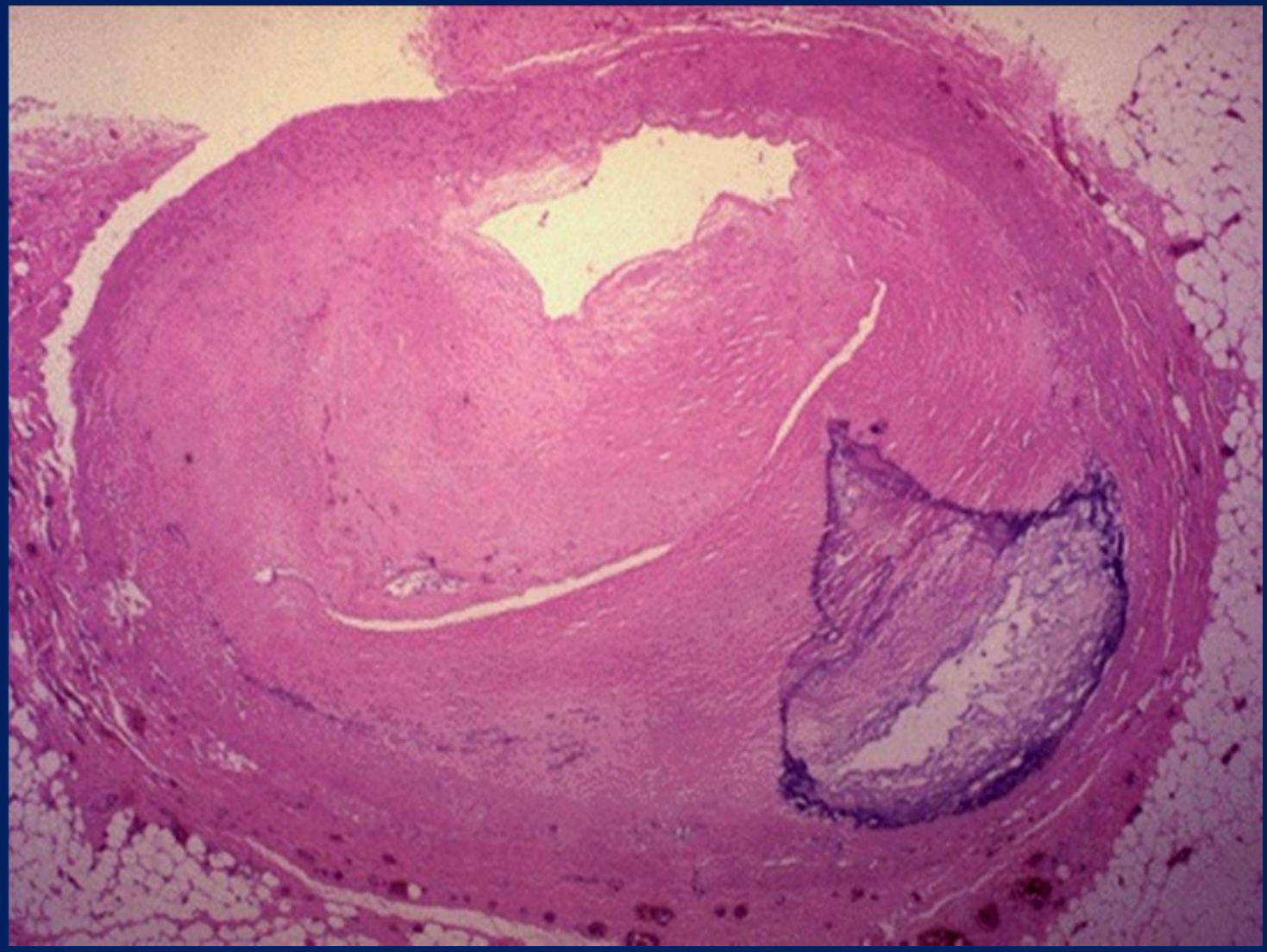
This is severe atherosclerosis of the aorta in which the atheromatous plaques have undergone ulceration along with formation of overlying mural thrombus (arrows).

Coronary atherosclerosis: plaque hemorrhage



This is coronary atherosclerosis with the complication of hemorrhage into atheromatous plaque (arrow). Such hemorrhage acutely may narrow the arterial lumen.

Stenosing coronary atheroma with calcification



There is a severe degree of narrowing in this coronary artery. It is "complex" in that there is a large area of calcification on the lower right, which appears bluish on this H&E stain. Complex atheroma have calcification, thrombosis, or hemorrhage. Such calcification would make coronary angioplasty difficult.

Pathogenesis of atherosclerosis

The most widely accepted theory of pathogenesis is called the ***response to injury hypothesis***. This theory states that:

Lesions of atherosclerosis are initiated as a response to some form of repeated chronic injury to arterial endothelium.

This injury increases endothelial permeability to plasma lipids as well as permitting blood monocytes and platelets to adhere to the endothelium.

Monocytes subsequently enter the intima, transform into macrophages and accumulate lipids to become foam cells.

Factors released from both platelets and macrophages cause migration of smooth muscle cells from the media into the intima with eventual synthesis and accumulation of collagen.

Hyperlipidemia, hypertension and smoking may be responsible for the endothelial injury.

Macrophages produce, among other substances, toxic free oxygen radicals (ROS) that oxidize (modify) LDL in the lesions. This oxidized LDL is considered atherogenic, as it is

- Chemotactic to blood monocytes
- Inhibits macrophage motility thus preventing them from leaving the atheroma.
- Cytotoxic to endothelial cells increasing their permeability

This suggests that antioxidants e.g. vitamin E and ß-carotene may be effective in preventing atherosclerosis by reducing LDL oxidation.

Clinical significance of atherosclerosis

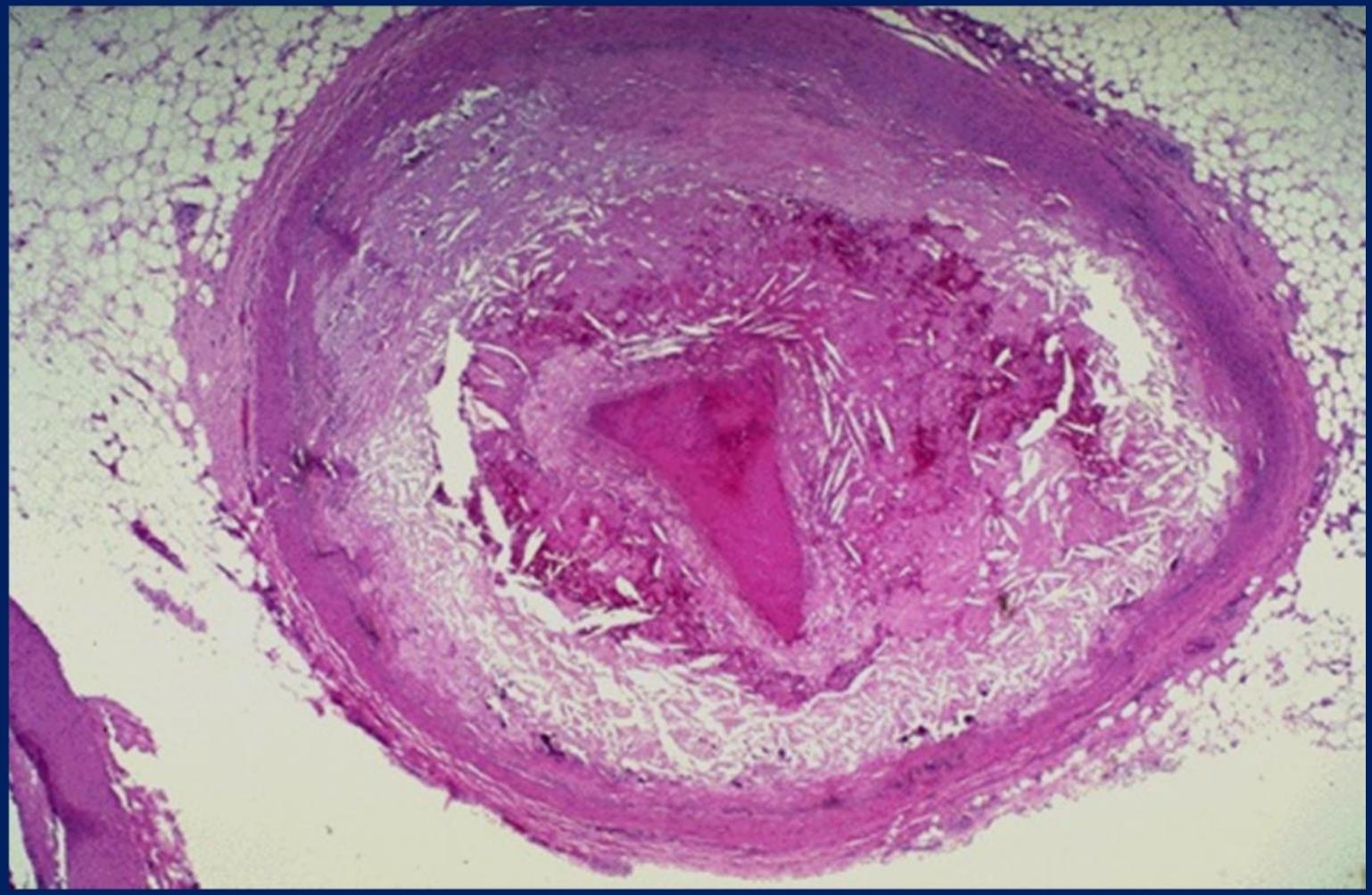
Atherosclerosis cause clinical disease through the following

1. Slow, progressive narrowing of the arterial lumen that result in chronic ischemia of the relevant tissues.
2. Sudden occlusion of the lumen by superimposed thrombosis or hemorrhage into the atheroma.

This may produce severe ischemia that if prolonged may terminate in infarction.

3. Providing a site for thrombosis and then embolism.
4. Weakening of the wall of an artery, causing aneurysmal dilatation with subsequent rupture.

Coronary atherosclerosis with superimposed occlusive thrombosis



There is a pink to red recent thrombosis in this narrowed coronary artery. The open, needle-like spaces in the atheromatous plaque are cholesterol clefts.

Atherosclerotic involvement of medium sized arteries produces a set of clinical features that differ from those arising from involvement of large arteries. Explain

In large arteries

- Large mural thrombi → peripheral emboli.
- Aneurysmal dilatation → rupture
- Rupture of the atheroma → cholesterol emboli.

In smaller arteries

- Narrowing of the lumen → chronic ischemia
- Superadded thrombosis ~~or plaque~~ hemorrhage

↓

occlusion of the vessel

What effects does hypertension have on blood vessels?

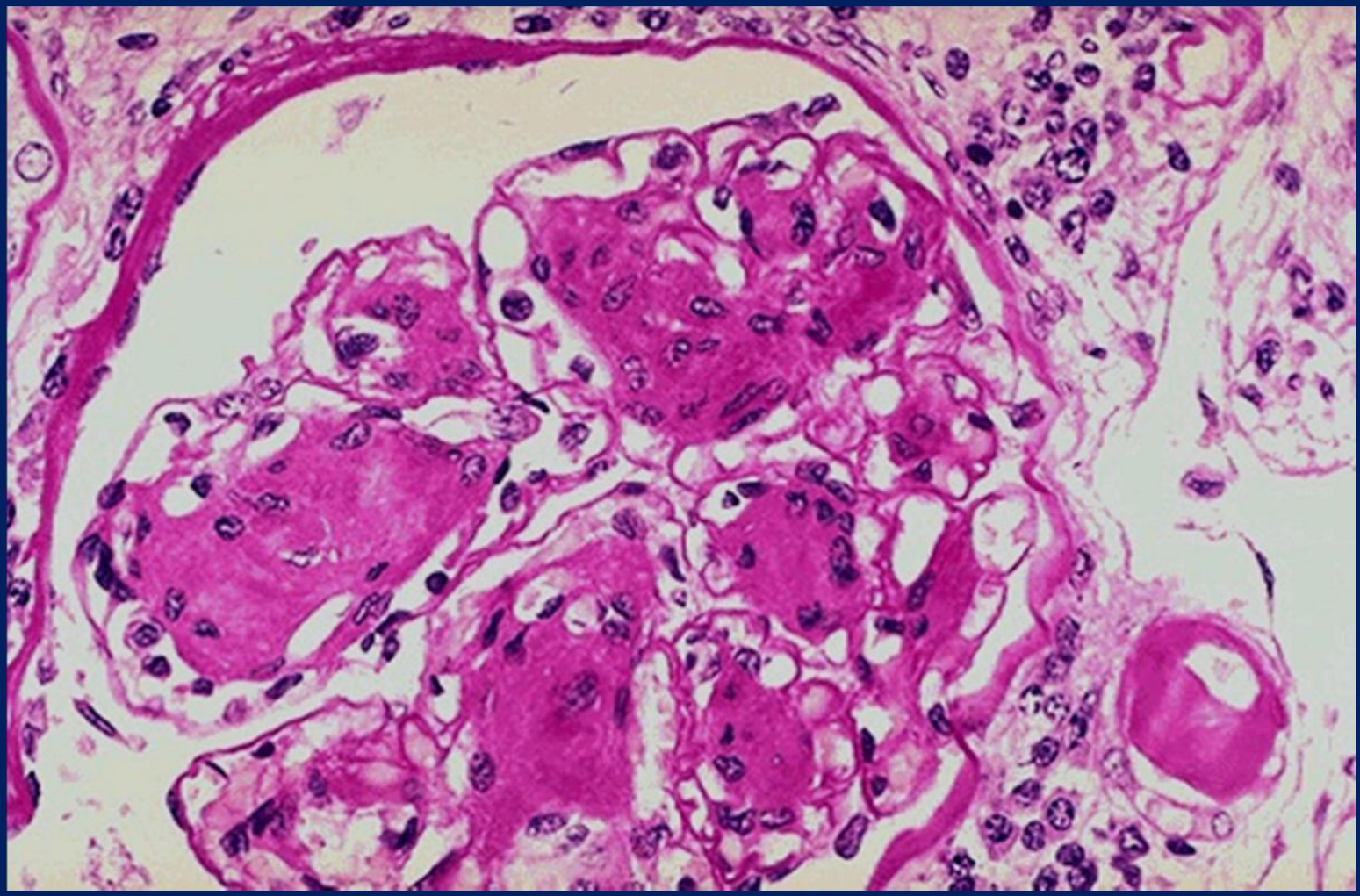
ARTERIOLOSCLEROSIS (Hypertensive vascular disease)

Hypertension is the most important cause of this group of vascular diseases. Hypertension has the following effects on blood vessels

1. It accelerates the process of atherosclerosis.
2. Causes structural changes in the blood vessel wall that predisposes to
   - Aortic dissection.
   - Cerebrovascular hemorrhage.
3. Induce changes in arterioles referred to as arteriolosclerosis.

There are two forms of arteriolosclerosis; **hyaline & hyperplastic**

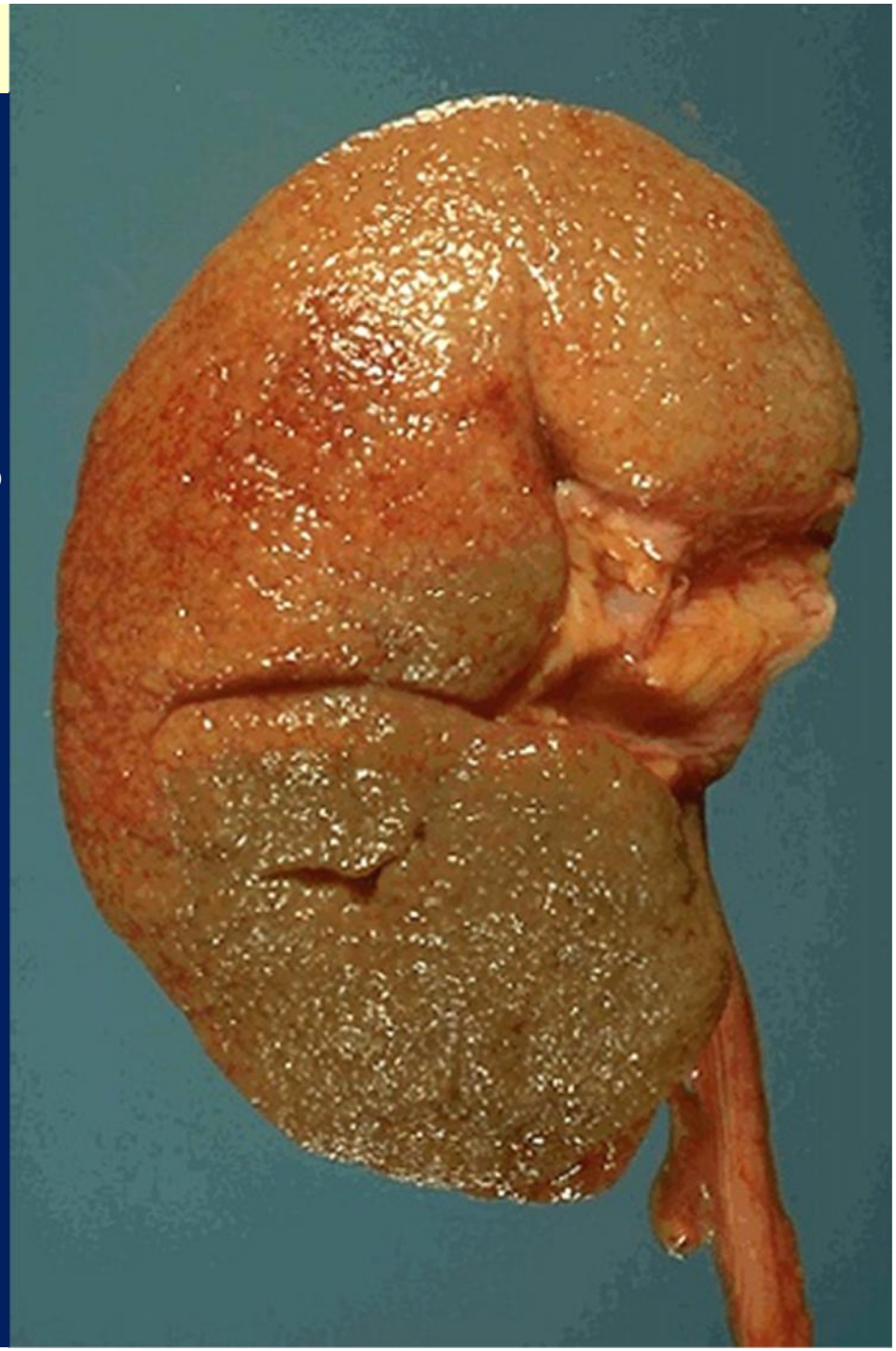
Hyaline arteriolosclerosis Kidney



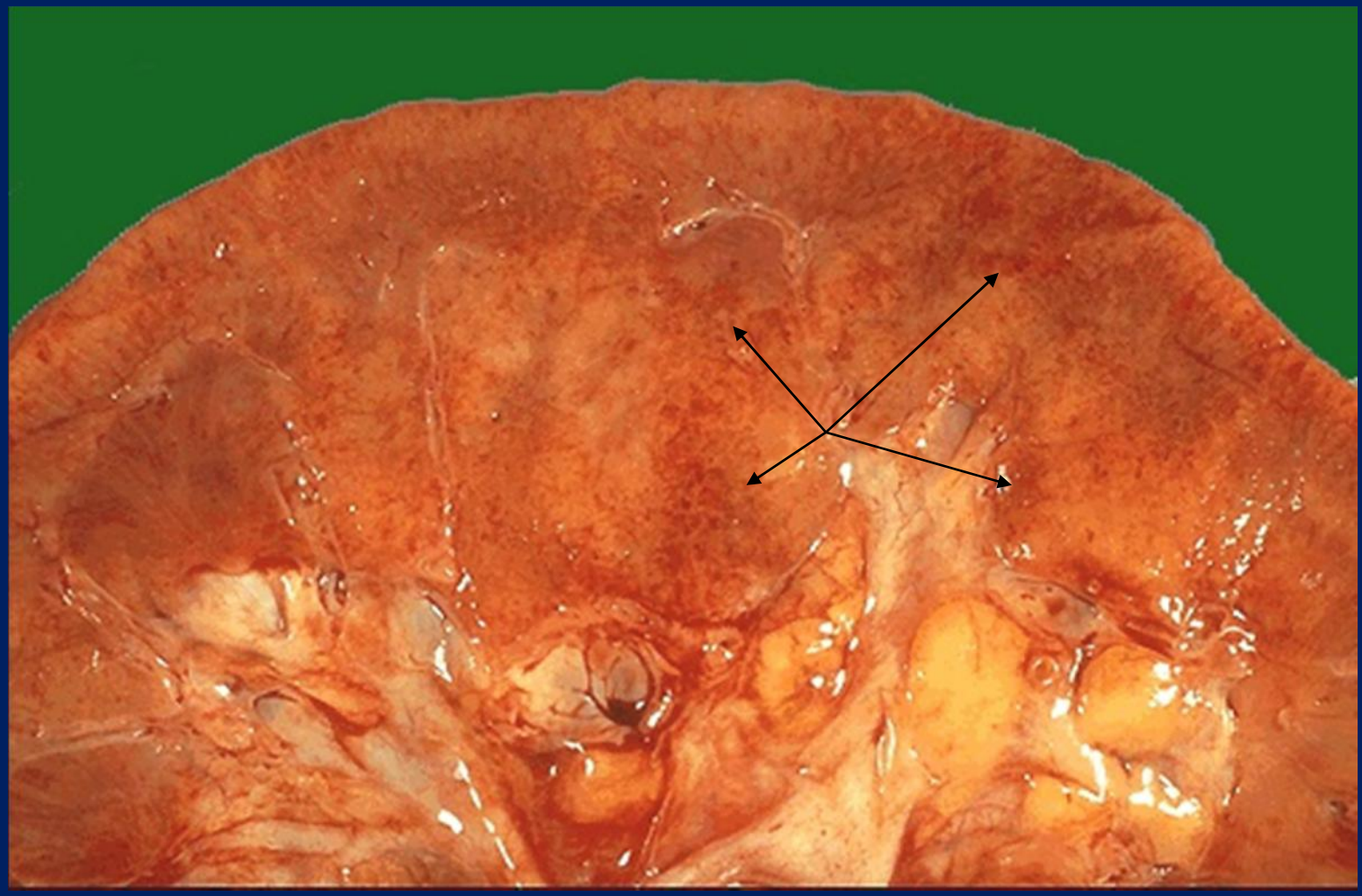
Arteriolosclerosis is typically seen in the kidneys. One form, called hyaline arteriolosclerosis, is demonstrated by the markedly thickened arteriole to the lower right of this glomerulus with PAS stain. Hyaline arteriolosclerosis is seen in the elderly, but more advanced lesions are seen in persons with diabetes mellitus and/or with hypertension.

Benign nephrosclerosis

The smaller arterioles in the kidney have become thickened and narrowed. This leads to patchy ischemic atrophy with focal loss of parenchyma that gives the surface of the kidney the characteristic granular appearance as seen here.

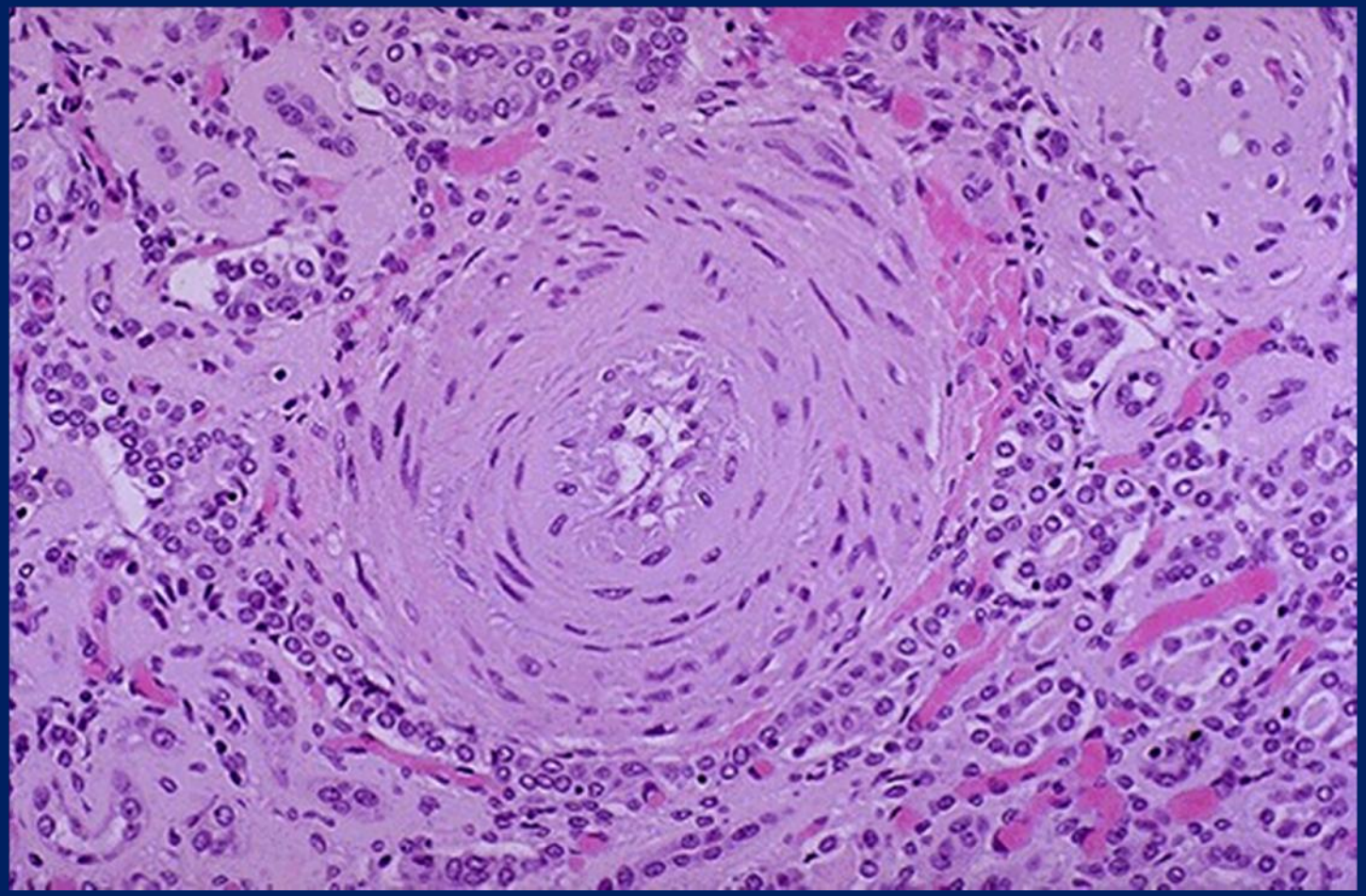


Malignant neophrosclerosis



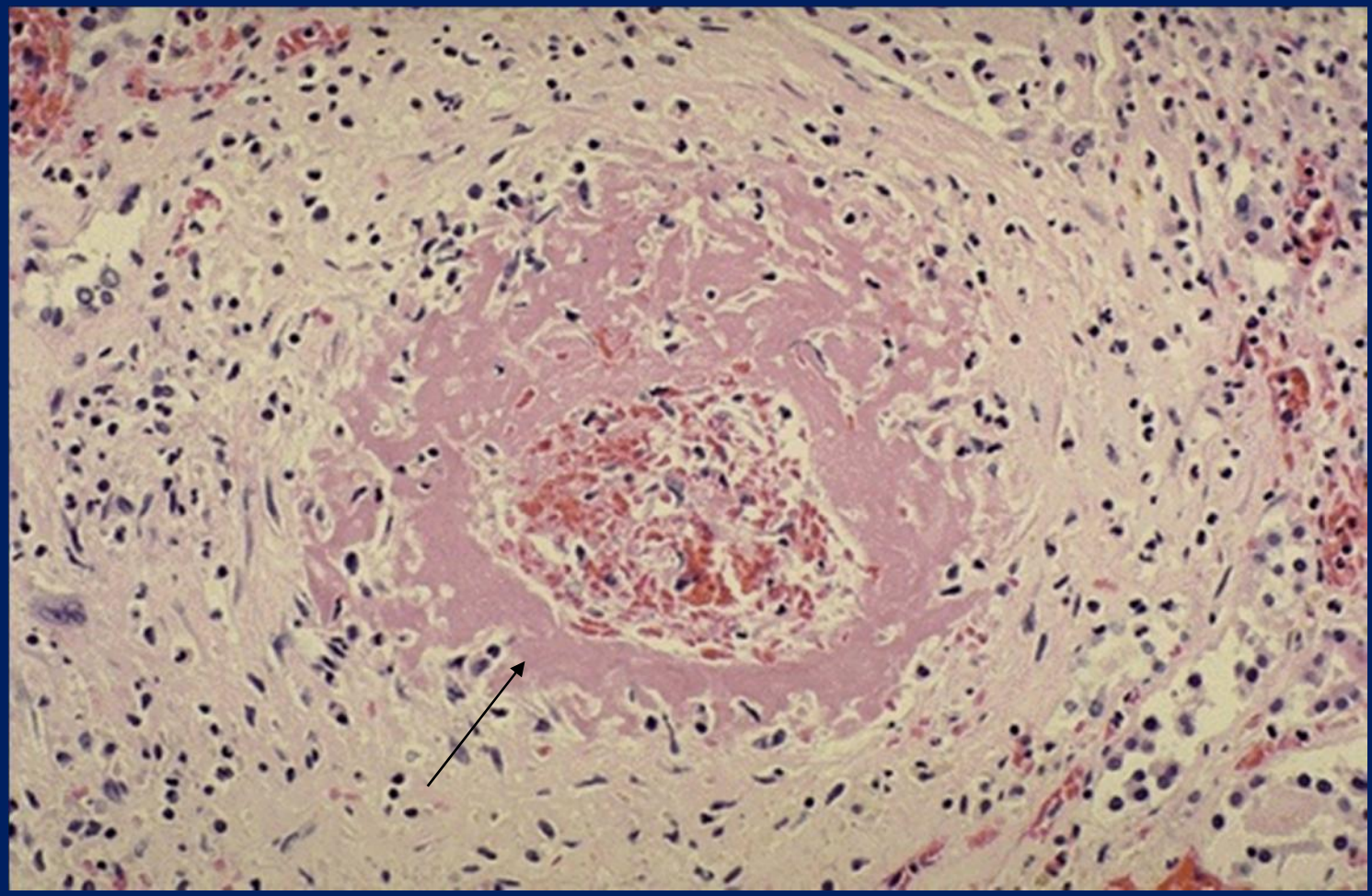
In malignant nephrosclerosis, the kidney demonstrates focal small hemorrhages. This is due to an accelerated phase of hypertension in which blood pressures are very high (such as 300/150 mm Hg).

Hyperplastic arterilosclerosis



Onion-skin concentric, laminated thickening of the arteriolar wall with progressive narrowing of the lumen.

Hyperplastic arterilosclerosis with fibrinoid necrosis



One complication of hyperplastic arteriolosclerosis with malignant hypertension is fibrinoid necrosis, as seen here in a renal arteriole. Rupture of the affected arterioles lead to grossly visible minute hemorrhages.

Aneurysms and dissection

Classifications

Morphological classification

- Berry
- Saccular
- Fusiform

Etiological classification

- Atherosclerosis
- Cystic medial degeneration
- Syphilis
- Vasculitides
- Trauma
- Congenital defects
- Infections

The two most important causes of aortic aneurysms are

1. atherosclerosis
2. cystic medial degeneration

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Classifications of aneurysms

- Aneurysms are classified either morphologically (according to their gross appearance or etiologically (the underlying mechanism responsible for their development).
- Morphological classification

This is based on the macroscopic shape and size

1. Berry aneurysm is a small, spherical dilatation usually up to 1.5 cm in diameter. It is most frequently seen within the circle of Willis at the base of the brain.

Berry: any small globular or ovate juicy fruit, not having a stone

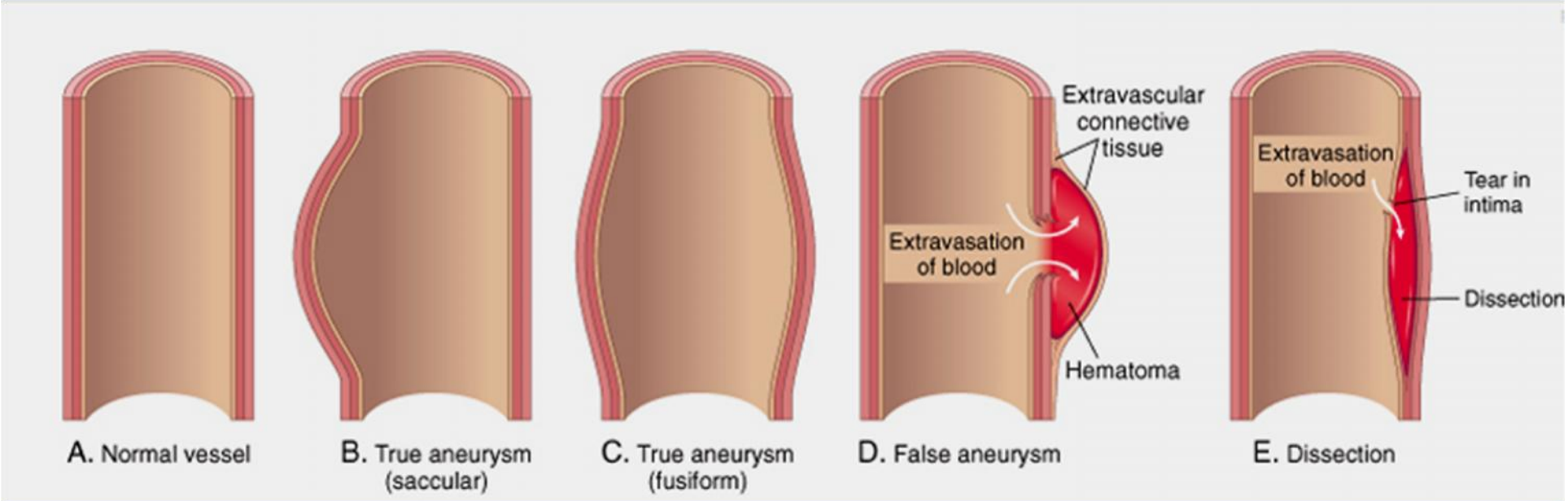
2. Saccular aneurysm is a spherical bulge from a portion of the vessel wall that varies in size from 5 to 20 cm in diameter. A saccular aneurysm could be considered from the morphological point of view a giant berry aneurysm.
3. Fusiform aneurysm results from gradual, progressive dilatation of the whole circumference of a segment of the vessel and may reach up to 20 cm in diameter.

- Etiological classification

1. Atherosclerosis
2. Cystic medial necrosis or degeneration
3. Syphilis
4. Vasculitides e.g. PAN and Kawasaki disease.
5. Trauma leading to arterio-venous aneurysm
6. Congenital defects such as that producing berry aneurysms in the brain
7. Mycotic aneurysm produced as a result of infection of the arterial wall.

The two most important causes of aortic aneurysms are atherosclerosis and cystic medial degeneration. However, any vessel may be affected by a wide variety of disorders that weaken the wall.

Morphological types of aneurysms



A. Normal vessel. B, True aneurysm, saccular type. The wall focally bulges outward and may be attenuated but is otherwise intact. C, True aneurysm, fusiform type. There is circumferential dilation of the vessel, without rupture. D, False aneurysm. The wall is ruptured, and there is a collection of blood (hematoma) that is bounded externally by adherent extravascular tissues. E, Dissection. Blood has entered (dissected) the wall of the vessel and separated the layers. Although this is shown as occurring through a tear in the lumen, dissections can also occur by rupture of the vessels of the vaso vasorum within the media.

Berry aneurysm
(saccular aneurysm-Congenital aneurysm)

- Most frequent of intracranial aneurysms
- Most frequent cause of subarachnoid hemorrhage
- 2% of general population have them
- Thin-walled bright red out-pouching
- Occur at arterial branch points along the circle of Willis
- Pathogenesis: congenital defect of the media
- Rupture most frequent in age group of 40-50 yr

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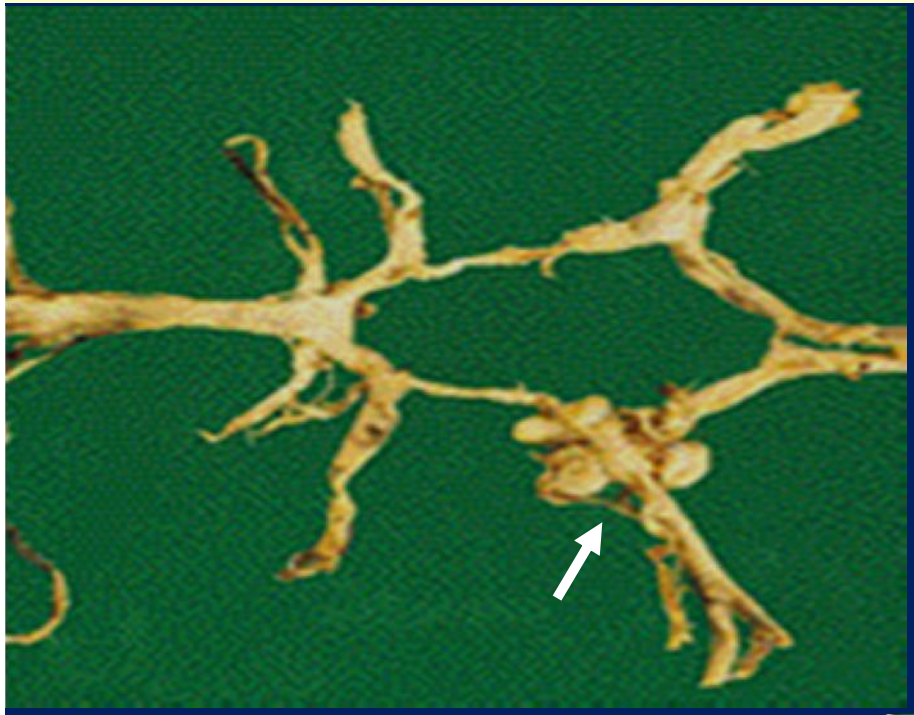
Berry aneurysm (saccular aneurysm-Congenital aneurysm)

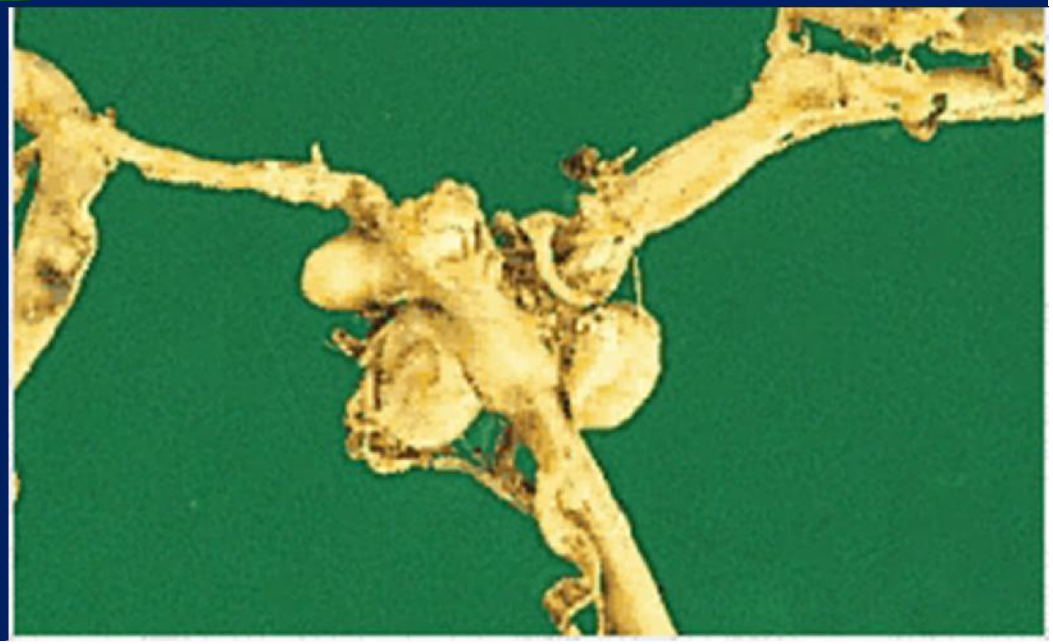
- This is the most frequent type of intracranial aneurysms and the one most frequently responsible for subarachnoid hemorrhage.
- It has an incidence of about 2% in the general population.
- An unruptured berry aneurysm is a thin-walled bright red out-pouching at arterial branch points along the circle of Willis or major vessels just beyond.
- The pathogenesis is thought to be due to congenital defect of the media especially at bifurcations.
- Ruptured berry aneurysm with clinically significant subarachnoid hemorrhage is most frequent in the age group of 40-50 years.

dr.hassan; 28/11/2003

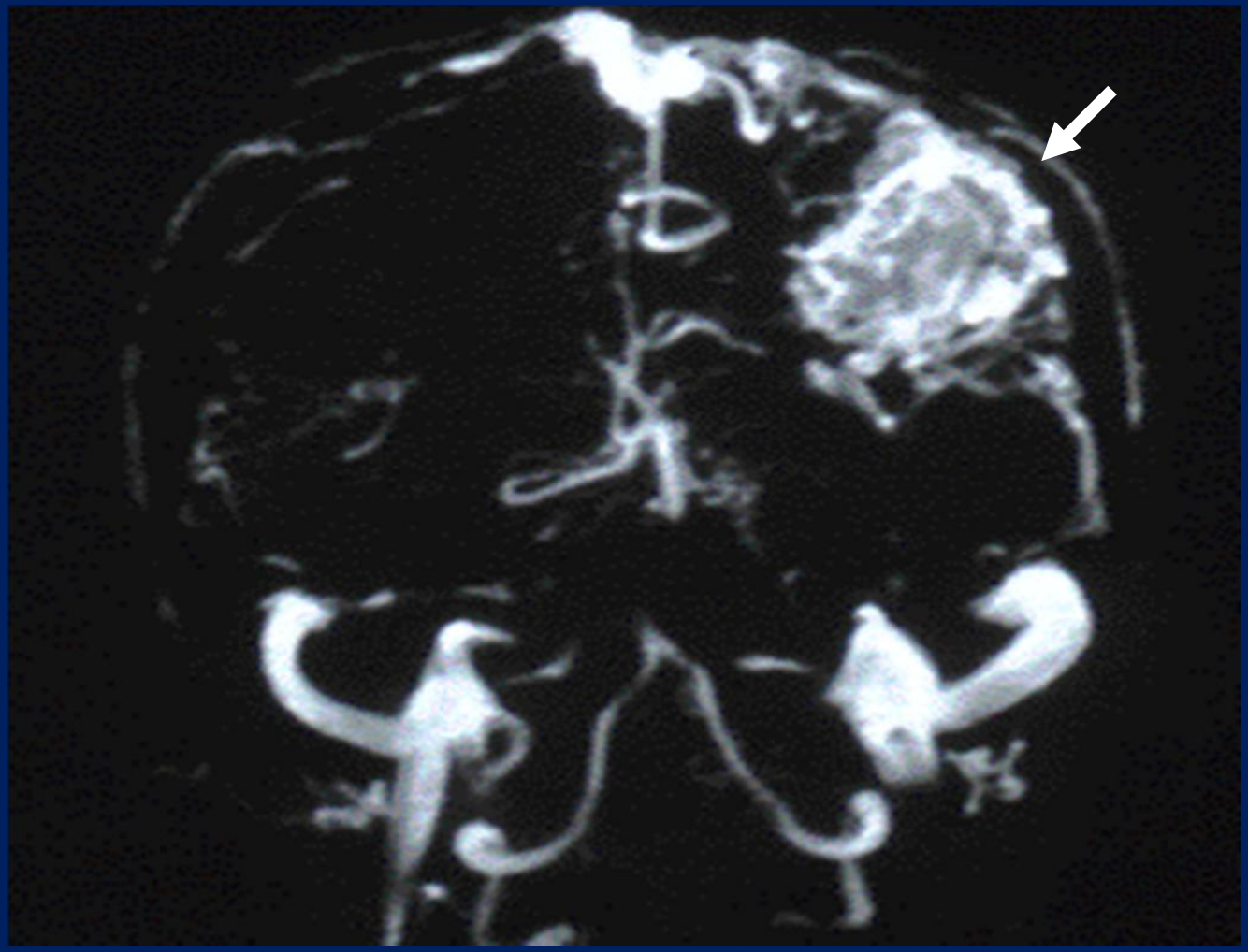
Berry aneurysms



Circle of Willis with anterior, middle and posterior cerebral arteries linked by communicating vessels. Berry aneurysms are seen arising where the internal carotid bifurcates into middle and anterior cerebral arteries (arrow).

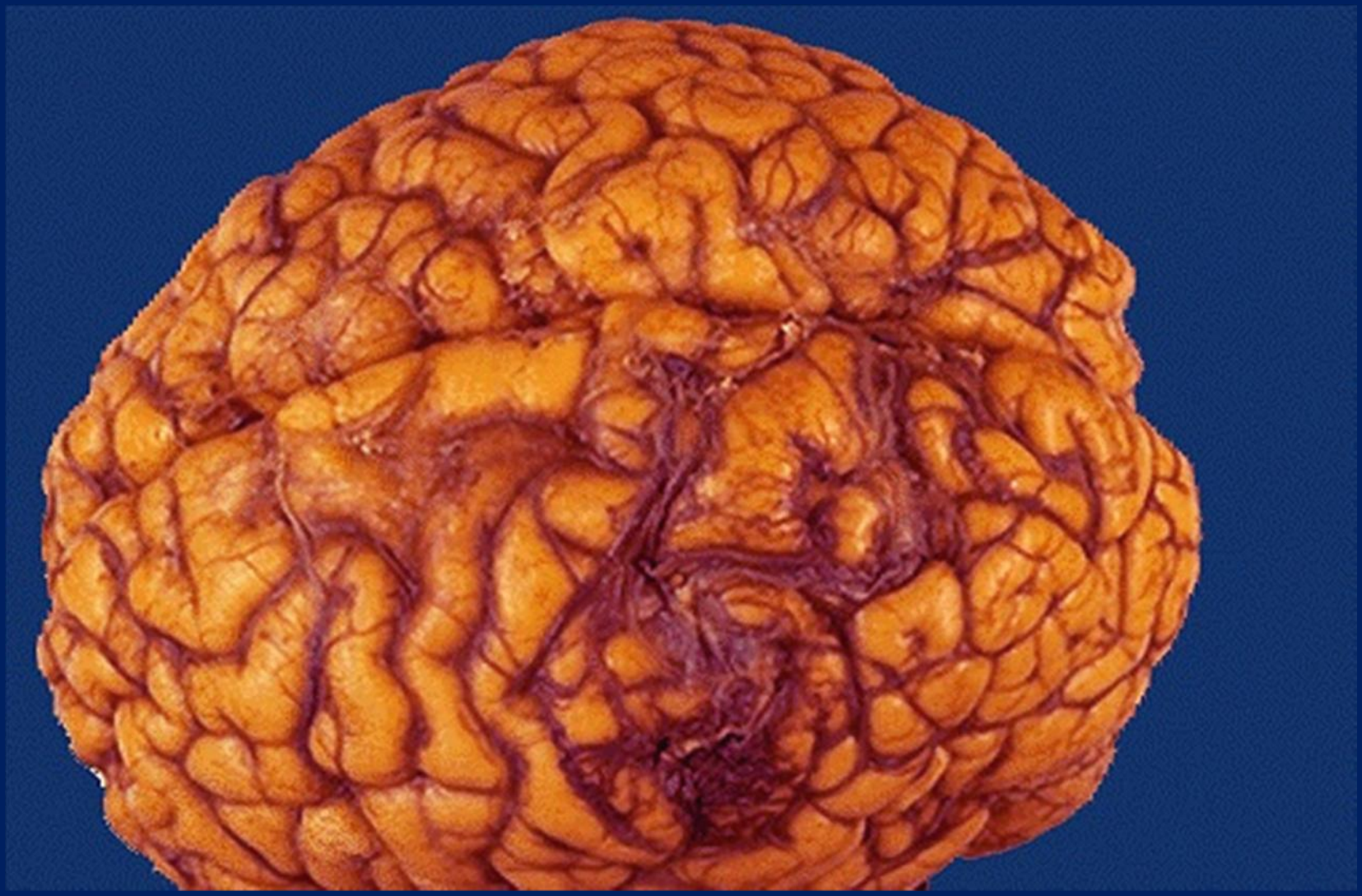


AVM (MRI of brain)



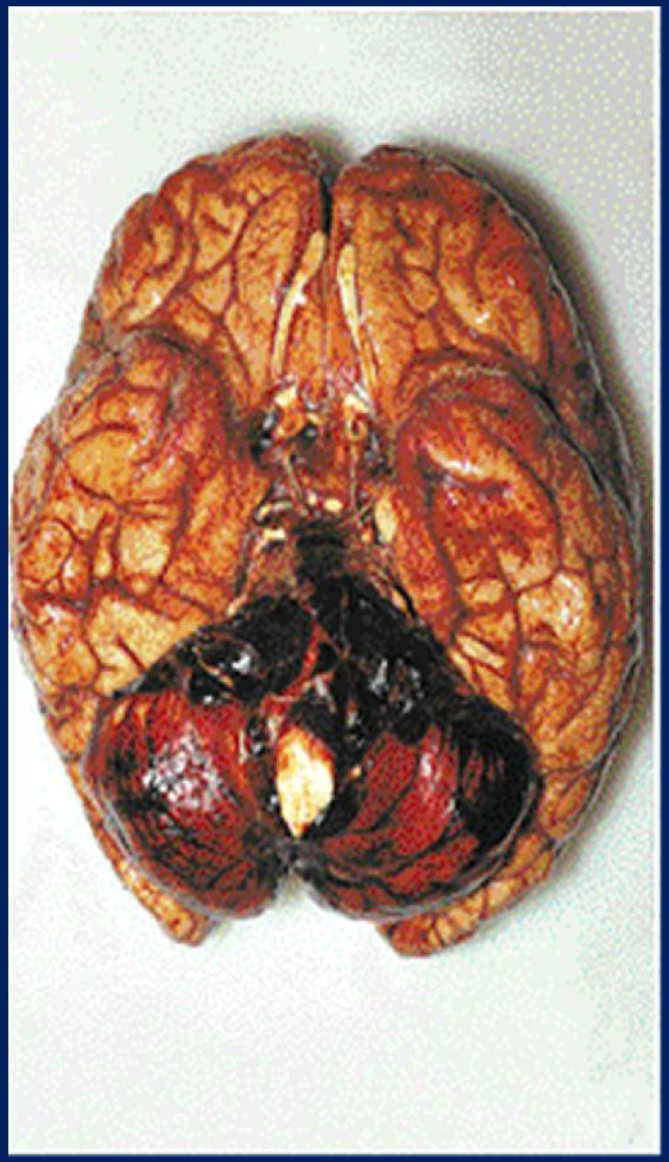
There is a large abnormal mass of vessels in the parietal lobe (arrow). Such abnormal vessels are prone to bleeding.

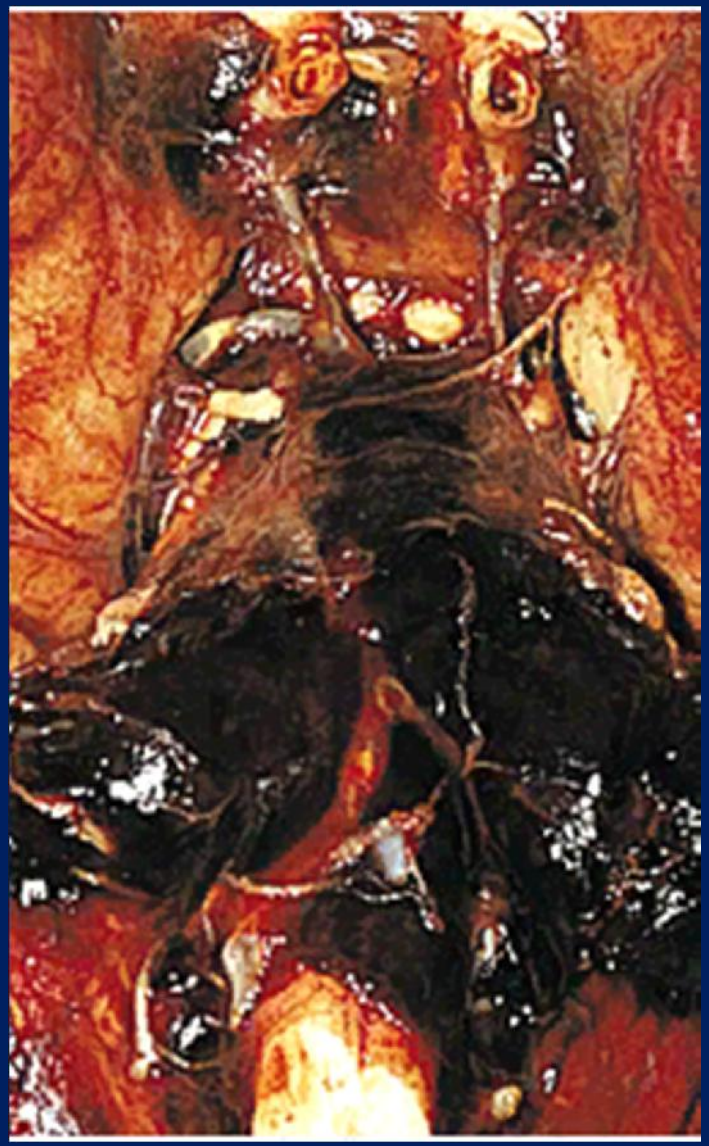
AVM Brain



There is a mass of irregular, tortuous vessels over the left posterior parietal region. This is one cause for hemorrhage, particularly in persons aged 10 to 30 years.

Ruptured Berry aneurysm with subarachnoid Hge

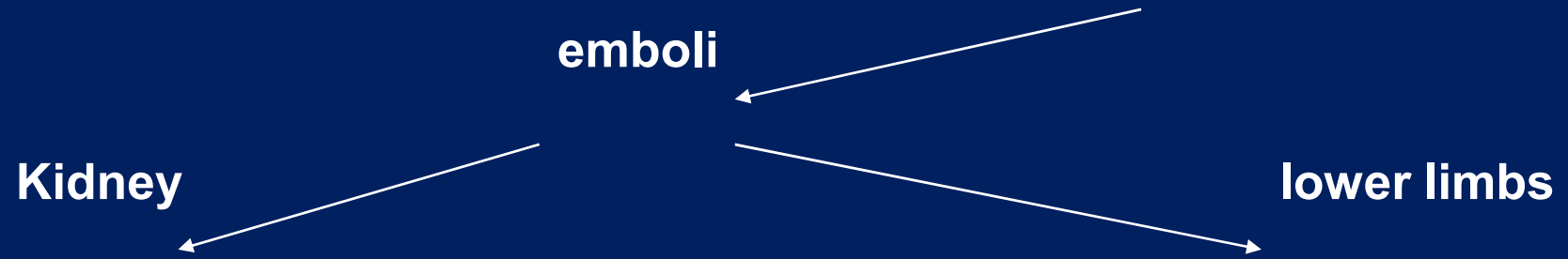




Blood is present in the sub-arachnoid space over the cerebellum. in this case the aneurysm was arising at the tip of the basilar artery.

Atherosclerotic aneurysm

- most frequent
- arterial wall thinning
- abdominal aorta
- usually fusiform
- contains atheromatous ulcers covered by mural thrombi



- Mycotic AAA
  - infected atherosclerotic aneurysm that has become infected
  - bacteremia complicating salmonella gastroenteritis
  - rapid course

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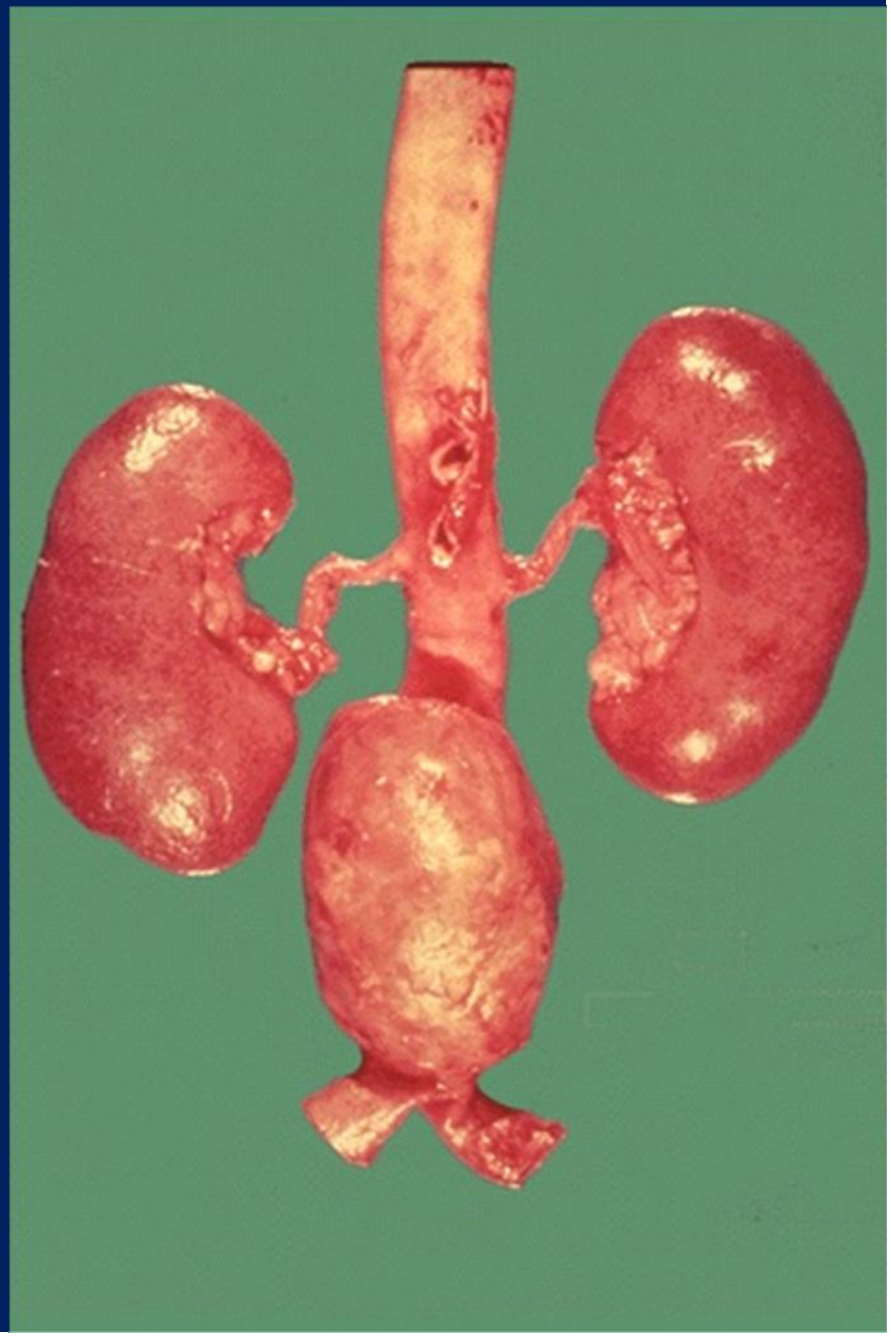
Atherosclerotic aneurysm

- Atherosclerosis is the most frequent etiology of aneurysms. It causes arterial wall thinning through medial destruction secondary to intimal plaques.
- Atherosclerotic aneurysms usually occur in the abdominal aorta, mostly between the renal arteries and the iliac bifurcation. They may also be seen in the common iliac arteries as well as in the arch and descending portions of thoracic aorta.
- The aneurysms are usually fusiform, contains atheromatous ulcers covered by mural thrombi. The latter may be a source of emboli that may lodge in renal vessels or those to the lower limbs.
- A variant of the abdominal aortic aneurysms (AAAs) is mycotic abdominal aortic aneurysm (Mycotic AAA). This is an atherosclerotic aneurysm that has become infected by circulating bacteria particularly from bacteremia complicating salmonella gastroenteritis (typhoid fever). In such cases the dilatation and eventual rupture of the aneurysm is more rapid because of the presence of a superadded destructive suppuration induced by the offending bacteria.
- Atherosclerosis is a major cause of abdominal aneurysms.

dr.hassan; 27/11/2003

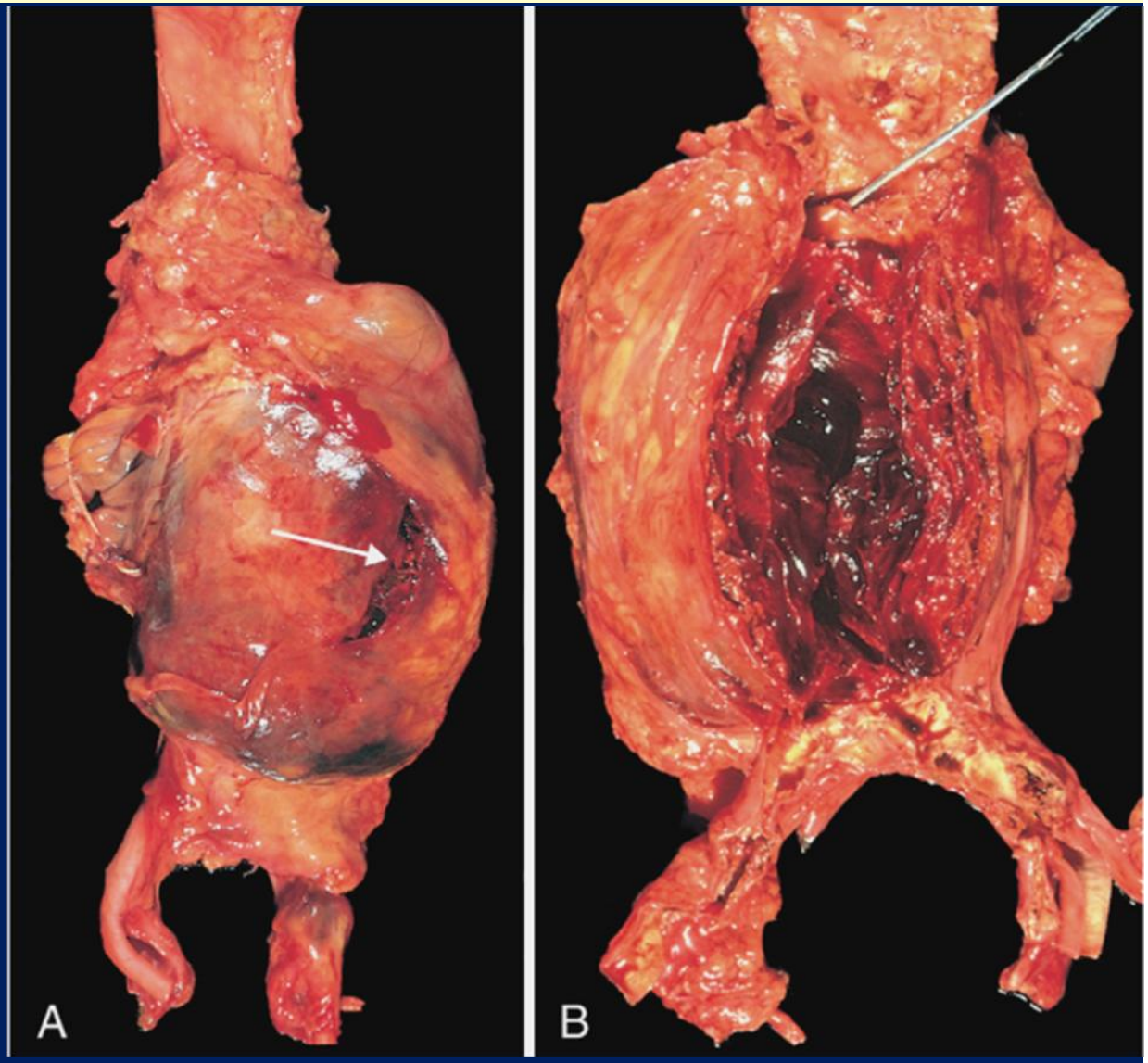
Atherosclerotic aneurysm of the abdominal aorta

A large "bulge" appears just above the aortic bifurcation. Such aneurysms are prone to rupture when they reach about 6 to 7 cm in size. They may be felt on physical examination as a pulsatile mass in the abdomen. Most such aneurysms are located below the renal arteries so that surgical resection can be performed with placement of a dacron graft.



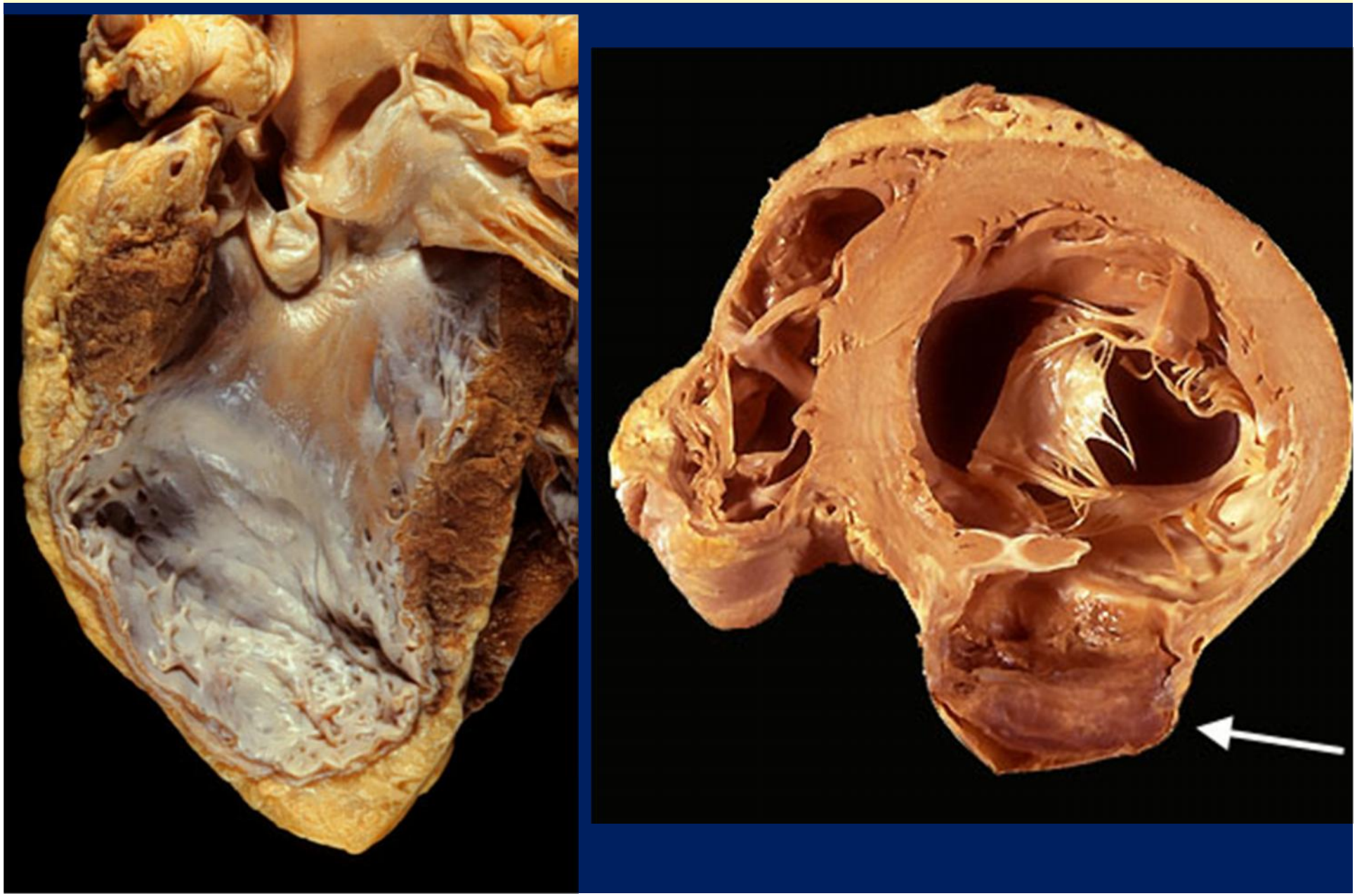
Abdominal aortic aneurysm

A, External view, gross photograph of a large aortic aneurysm that ruptured (arrow). B, Opened view, with the location of the rupture tract indicated by a probe. The wall of the aneurysm is exceedingly thin, and the lumen is filled by a large quantity of layered but largely unorganized thrombus.



P18

Below is a longitudinal & cross section of a heart. Describe the abnormalities. In what way this change affects the heart?



P18

Pathology 24/09/2009
NABEEL9/29/2004
There has been a previous extensive transmural myocardial infarction involving the free wall of the left ventricle. Note that the thickness of the myocardial wall is normal superiorly, but inferiorly is only a thin fibrous wall. The infarction was so extensive that, after healing, the ventricular wall was replaced by a thin band of collagen, forming an aneurysm. Such an aneurysm represents non-contractile tissue that reduces stroke volume and strains the remaining myocardium. The stasis of
blood in the aneurysm predisposes to mural thrombosis.

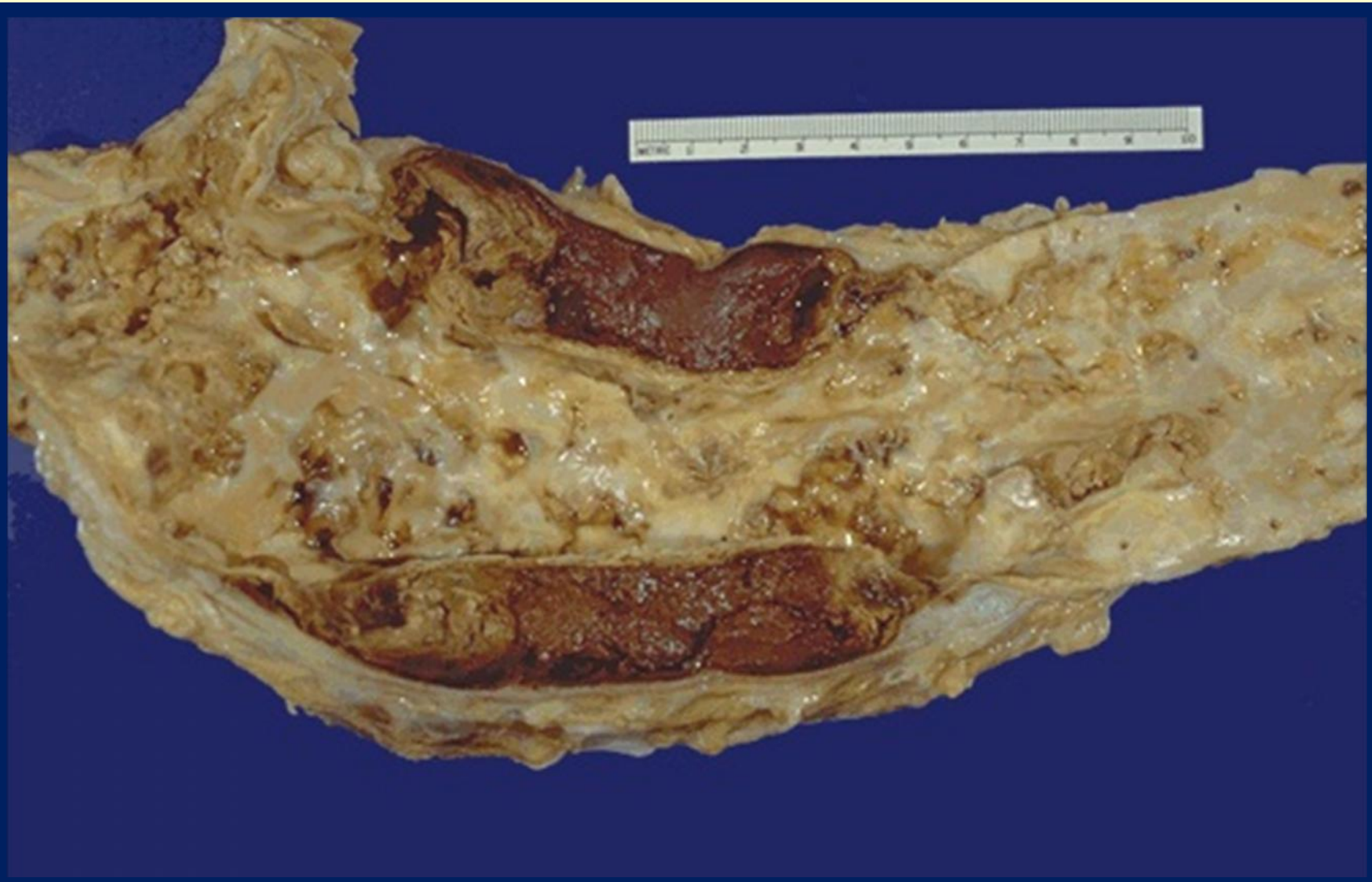
dr.hassan 30/11/2003
NABEEL9/29/2004
A cross section through the heart reveals a ventricular aneurysm with a very thin wall at the arrow. Note how the aneurysm bulges out. The stasis in this aneurysm
allows mural thrombus, which is present here, to form within the aneurysm.
Pathology; 24/09/2009

The clinical effects of aortic aneurysm include

- ***Rupture in to the peritoneal cavity*** *or retroperitoneum with massive or fatal hemorrhage.*
- ***Pressure on adjacent structures*** *leading for e.g. obstruction of a ureter or erosion of vertebrae.*
- ***Occlusion of a vessel*** *either by direct pressure or through intramural thrombus formation e.g. vertebral branches supplying spinal cord.*
- ***Embolism from the atheroma or mural thrombus.***
- ***Creation of abdominal mass*** *that may be confused with a tumor.*

Prosthetic grafts should replace large aneurysms (> 5 cm in diameter) to avoid the possibility of rupture.

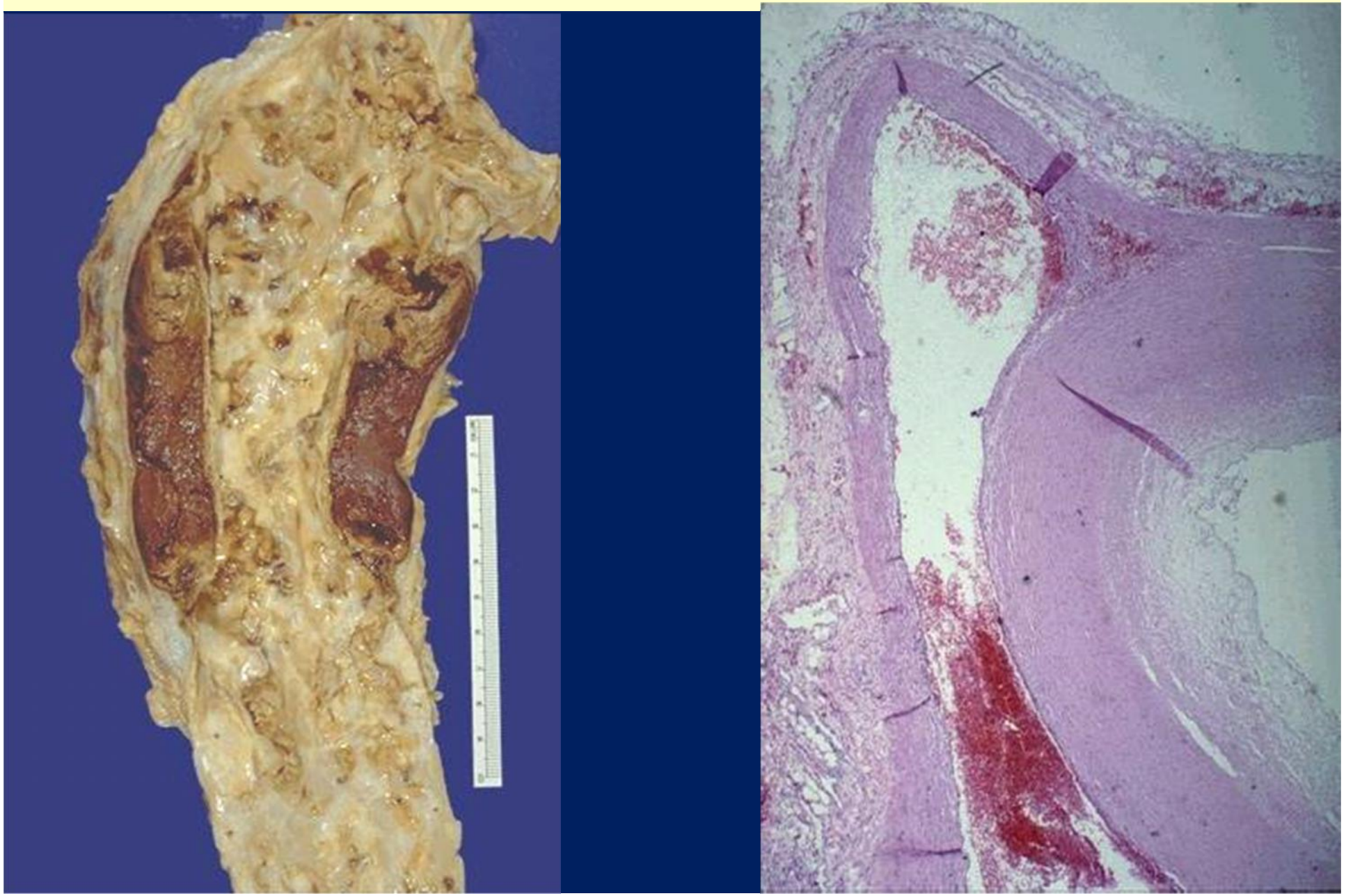
Aortic dissection



This aorta has been opened longitudinally to reveal an area of fairly limited dissection that is organizing. The red-brown hematoma can be seen in on both sides of the section as it extends around the aorta. The dissection creates a "double lumen" to the aorta. This aorta shows in addition severe atherosclerosis.

P20

The photo below shows an aorta opened longitudinally. The photo next to it is a microscopic section from the same aorta. Describe



P20

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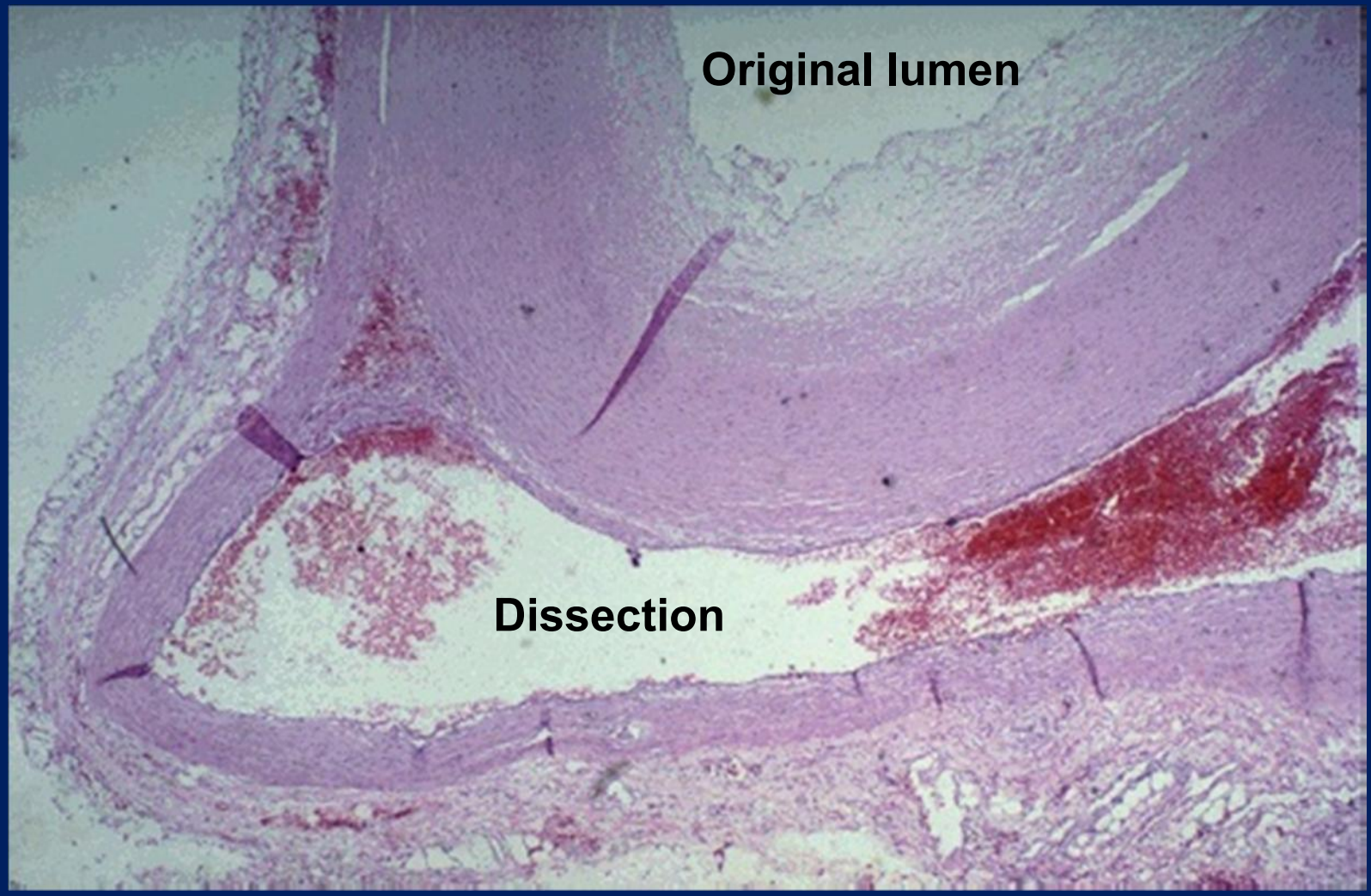
This aorta has been opened longitudinally to reveal an area of fairly limited dissection that is organizing. The red-brown thrombus can be seen in on both sides of the section as it extends around the aorta. The intimal tear would have been at the left. This creates a "double lumen" to the aorta. This aorta shows severe atherosclerosis which, along with cystic medial necrosis and hypertension, is a risk factor for dissection.

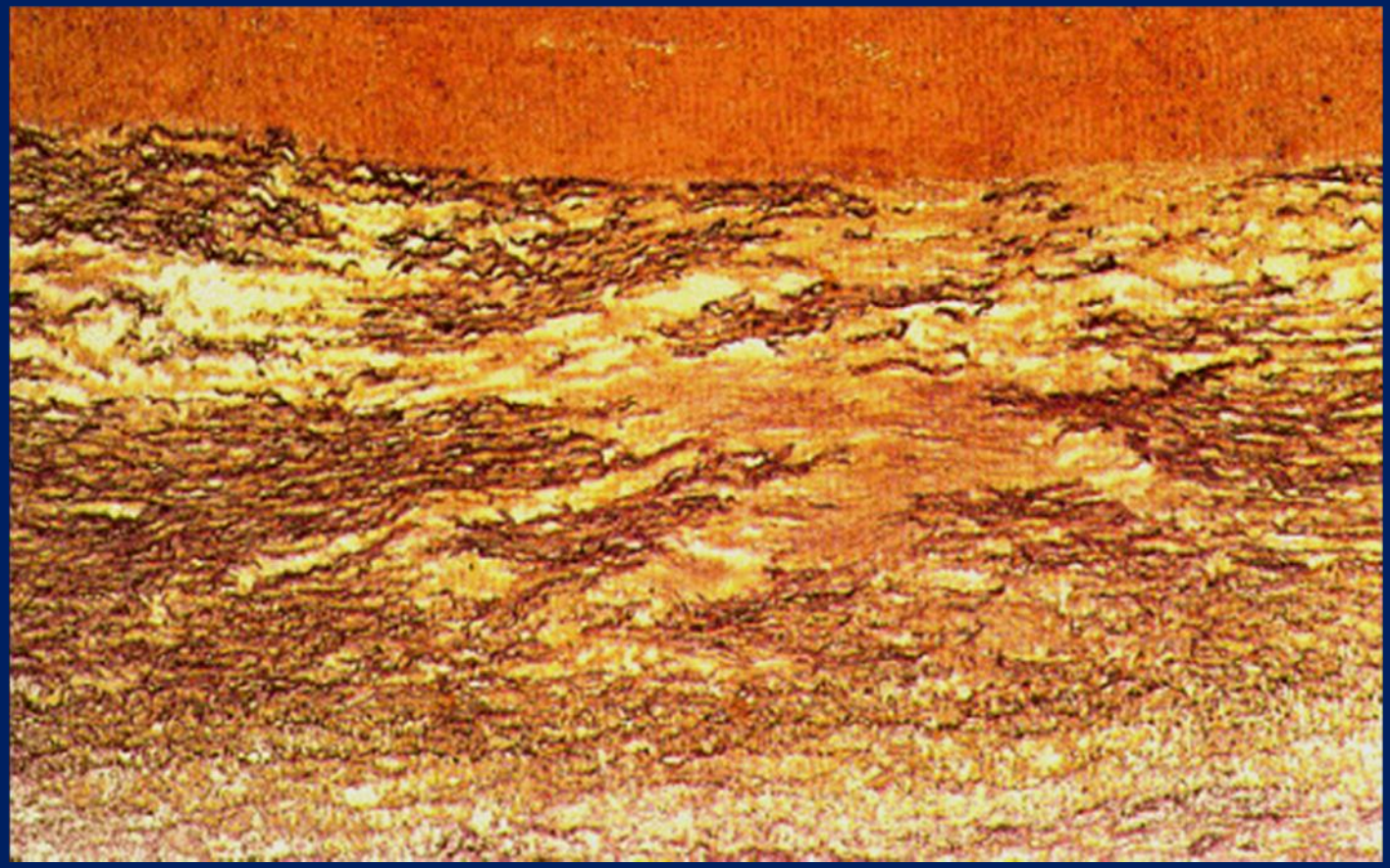
dr.hassan 30/11/2003
NABEEL9/29/2004
Here, the dissection went into the muscular wall. In any case, an aortic dissection is an extreme emergency and can lead to death in a matter of minutes. The blood
can dissect up or down the aorta. Blood dissecting up around the great vessels can close off the carotids. Blood can dissect down to the coronaries and shut them
off.

Aortic dissection



The dissection goes into the muscular wall creating an aorta with double lumina.

Aortic dissection: medial cystic degeneration



This special stain highlight the elastic fibers of the aortic wall. There is extensive fragmentation & destruction of the fibers associated with several coalescent cystic areas within the wall.

d11

Aortic dissection may have the following consequences

- Rupture into any of the three body cavities
- Extension of dissection
- Retrograde dissection
- Rupture in the lumen of the aorta through a second distal tear

d11

- Aortic dissection may have the following consequences

1. Rupture into any of the three body cavities i.e. pericardial, pleural or peritoneal. This is the most common cause of death.
2. Extension of the dissection into great arteries of the neck, coronaries, renal, mesenteric, or iliac arteries. This leads to their obstruction with subsequent ischemic damage to relevant organs or tissues e.g. myocardial infarction, renal infarction, and spinal cord ischemic injury (due to involvement of spinal arteries).
3. Retrograde dissection into the aortic root that leads to disruption of the valvular apparatus with consequent aortic valve insufficiency.
4. Rupture in the lumen of the aorta through a second distal tear. This is thought to avert a fatal extra-aortic hemorrhage.

dr.hassan; 27/11/2003

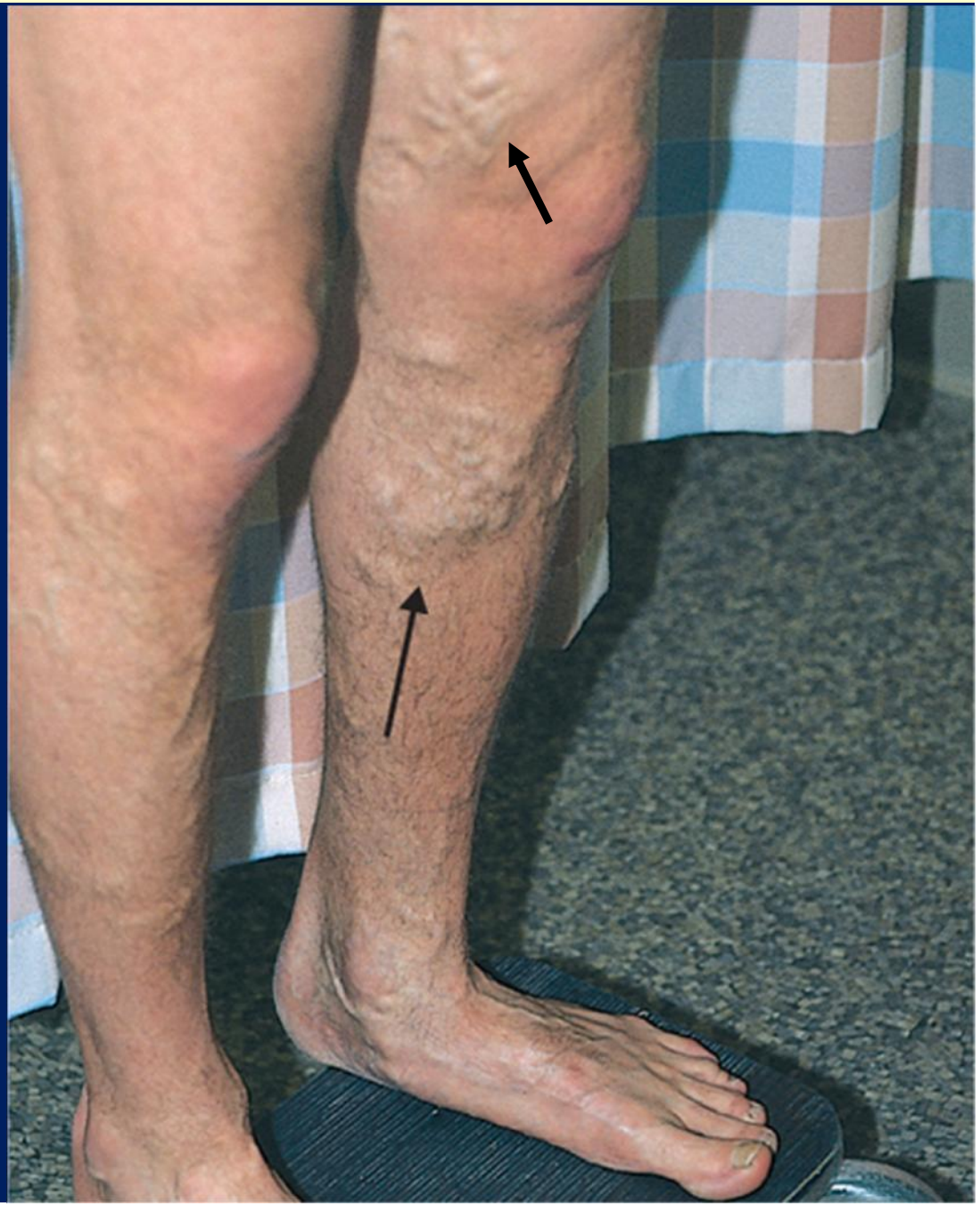
Diseases of veins

Varicose veins + phlebothrombosis =

90% of venous diseases.

Varicose veins of the leg

Note the prominently dilated & tortuous veins below & above the knee



d21

complications

Disabling complications include

- Persistent edema
- Stasis dermatitis
- Varicose ulcers.

d21

- Disabling complications include

1. Persistent edema
2. Stasis dermatitis (due to stasis of blood and liberation of hemosiderine)
3. Varicose ulcers.

dr.hassan; 28/11/2003

d27

Vascular tumors

Benign tumors and tumor-like conditions

Intermediate

Malignant

d27

Vascular tumors

- This is a heterogeneous group of neoplasms and tumor like conditions.
- It is best divided according to their biological behavior into the following three groups

1. Benign: e.g. hemangiomas, glomus tumor, and vascular ectasias.
2. Intermediate: locally aggressive tumors that rarely metastasize e.g. hemangioendothelioma.
3. Malignant: e.g. angiosarcoma, hemagiopericytoma, and Kaposis's sarcoma.

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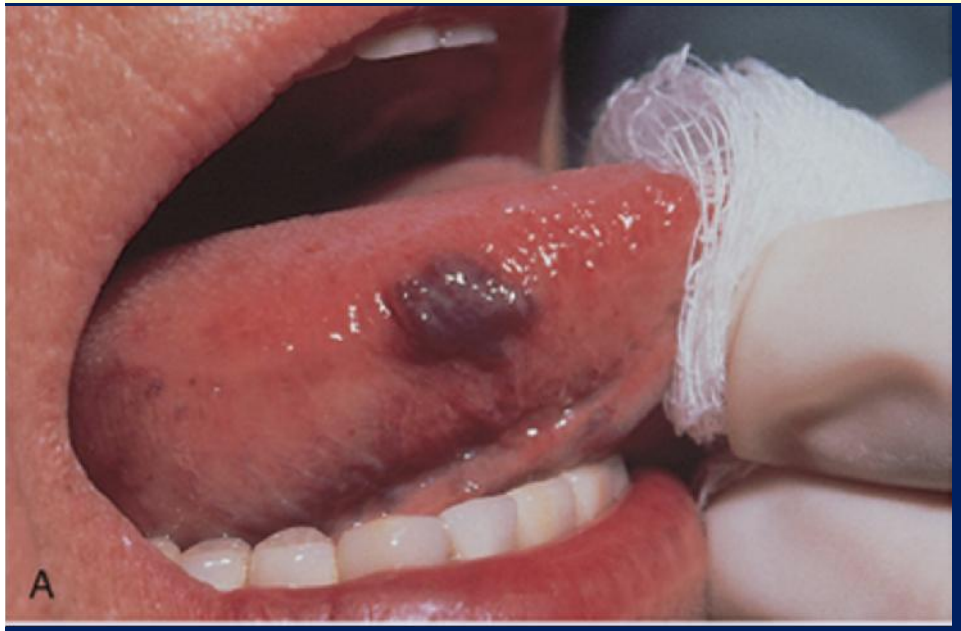
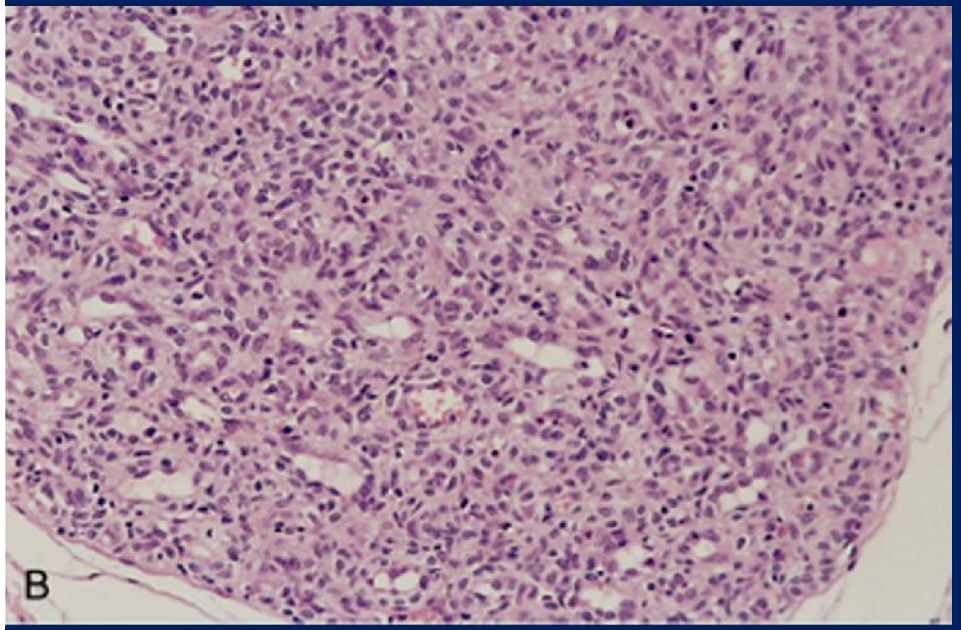
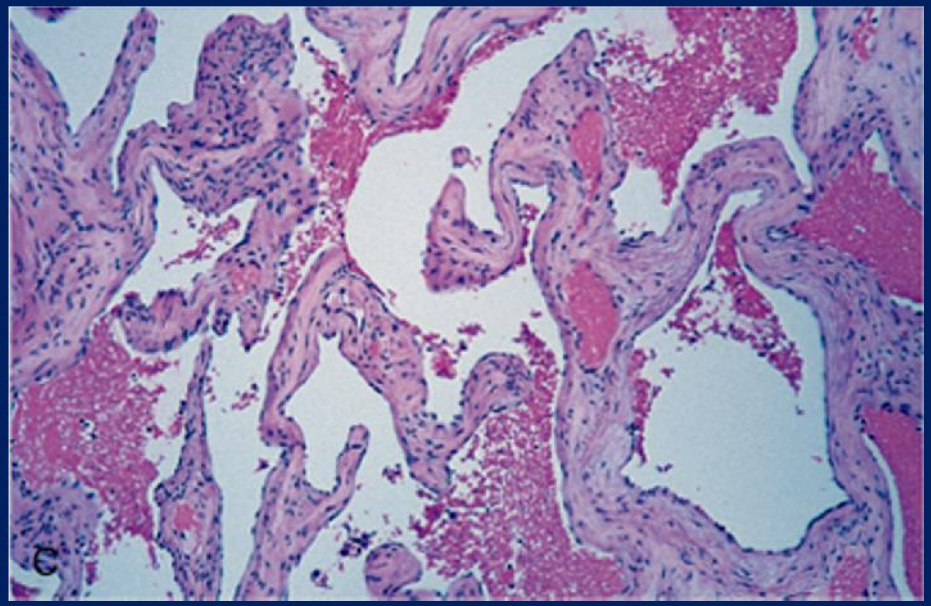
Hemangioma



A, Hemangioma of the tongue. B, Histology of juvenile capillary hemangioma. C, Histology of cavernous hemangioma





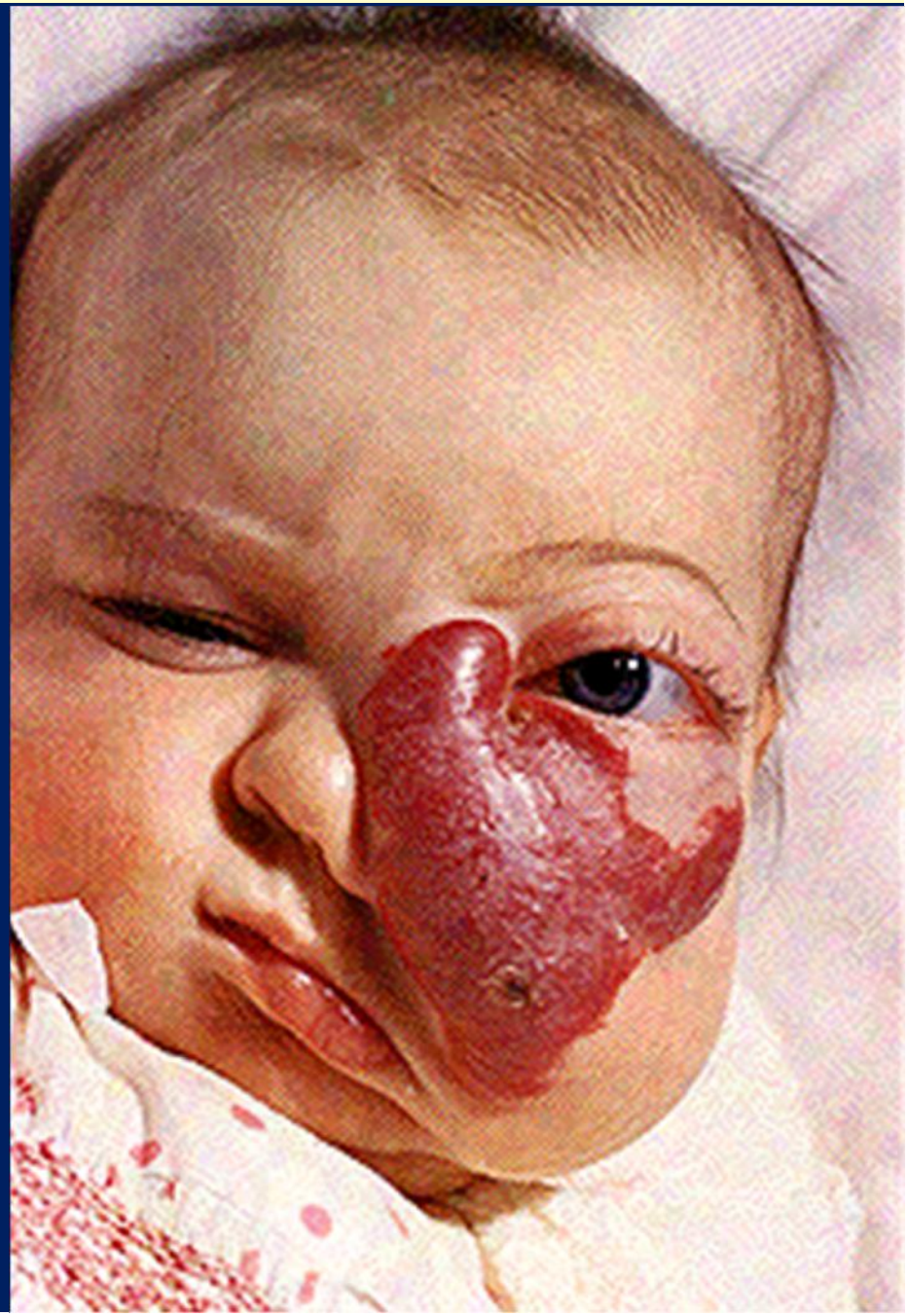
Mention the conditions in which cavernous hemangiomas are of clinical significance.

In most cases, the tumors are of little clinical significance; however,

1. There can be a cosmetic disturbance.
2. Visceral hemagiomas detected by imaging studies may need to be distinguished from more ominous malignant tumors.
3. Brain hemangiomas can cause pressure symptoms or rupture.
4 Cavernous hemangiomas are component of von Hippel-Lindau disease; they involve the cerebellum or brain stem and eye grounds, along with similar lesions in the pancreas and liver.

Disfiguring hemangioma of the face

Female infant shows a massive lesion distorting the nose and cheek.



II. Intermediate-Grade (Low-Grade Malignant) Tumors

A. Kaposi Sarcoma

- is used to be fairly common in patients with AIDS prior to the advent of effective antiretroviral therapy, and its presence is used as a criterion for diagnosing AIDS.
- Four forms of the disease are recognized, all of these share the same underlying viral pathogenesis:

1. Chronic KS (classic KS)

- ❖ characteristically occurs in older men.
- ❖ It is not associated with HIV.
- ❖ There multiple red to purple skin plaques or nodules, usually in the distal lower extremities.

2. Lymphadenopathic KS (African, endemic KS)

is particularly prevalent among South African Bantu children; it is also not associated with HIV. Skin lesions are sparse, and patients present instead with lymphadenopathy due to KS involvement; the tumor occasionally involves the viscera and is extremely aggressive.

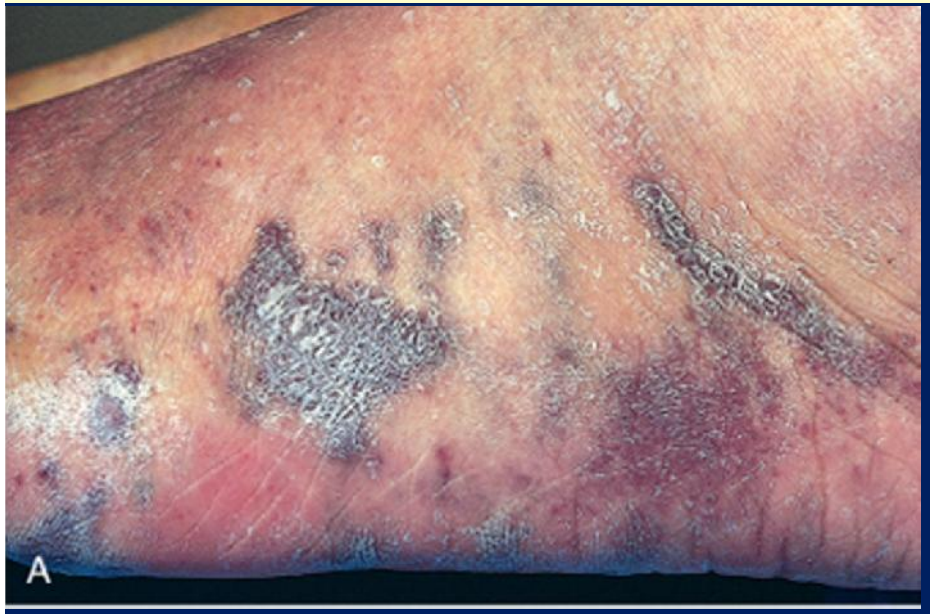
3. Transplant-associated KS :

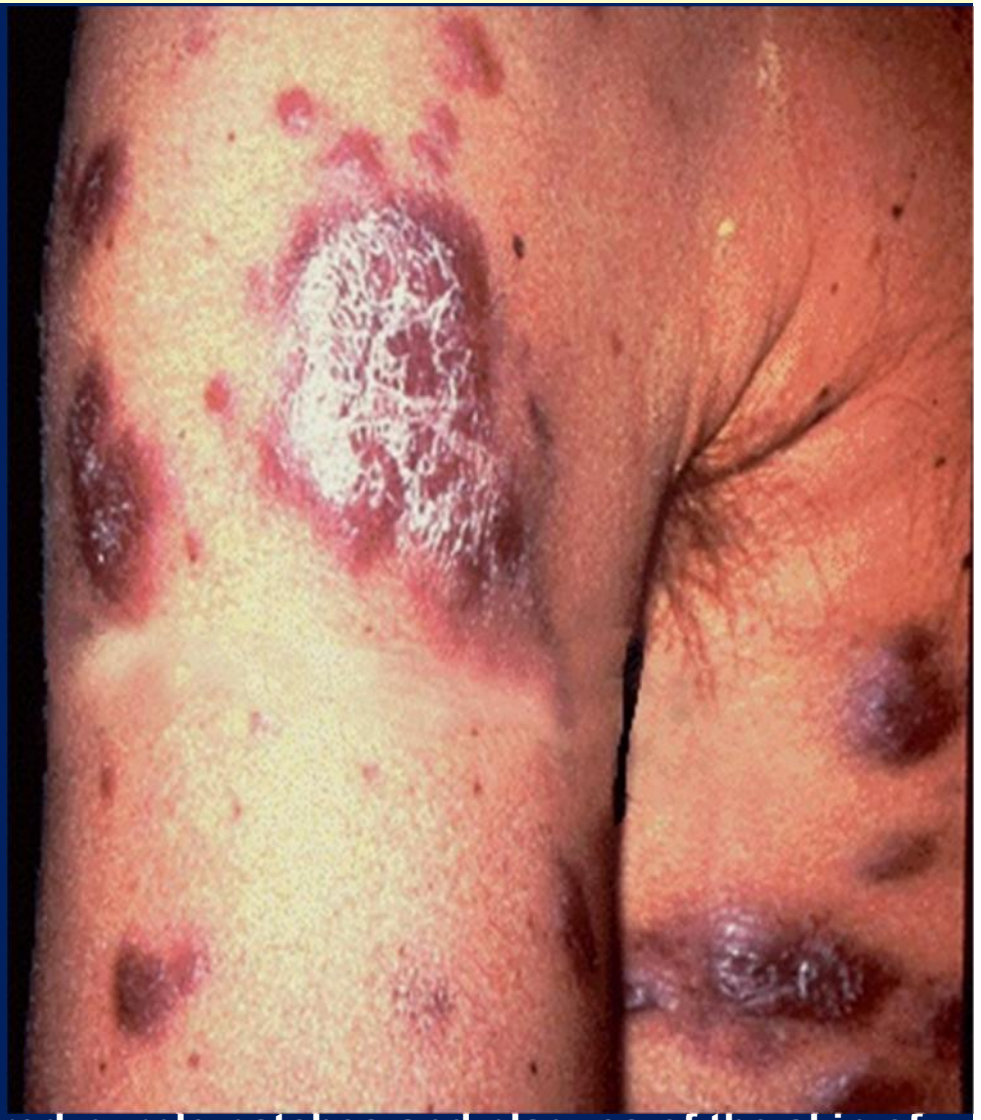
- Occurs in the setting of solid-organ transplantation with its attendant long-term immunosuppression.
- It tends to be aggressive (even fatal) with nodal, mucosal, and visceral involvement; cutaneous lesions may be absent.

4. AIDS-associated KS (epidemicKS) :

- was found in a third of AIDS patients, particularly male homosexuals.
- However, with current regimens of intensive antiretroviral therapy, KS incidence is now less than 1% (although it is still the most prevalent malignancy in AIDS patients in the United States).
- AIDS-associated KS can involve lymph nodes and viscera, with wide dissemination early in the course of disease.
- Most patients eventually die of opportunistic infectious rather than from KS.

Kaposi sarcoma

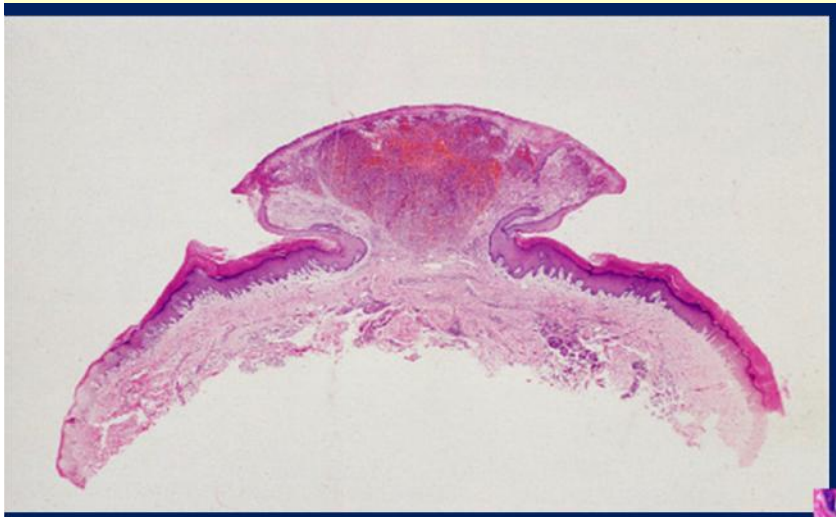




Rt. Gross photograph, illustrating coalescent red-purple patches and plaques of the skin of foot.

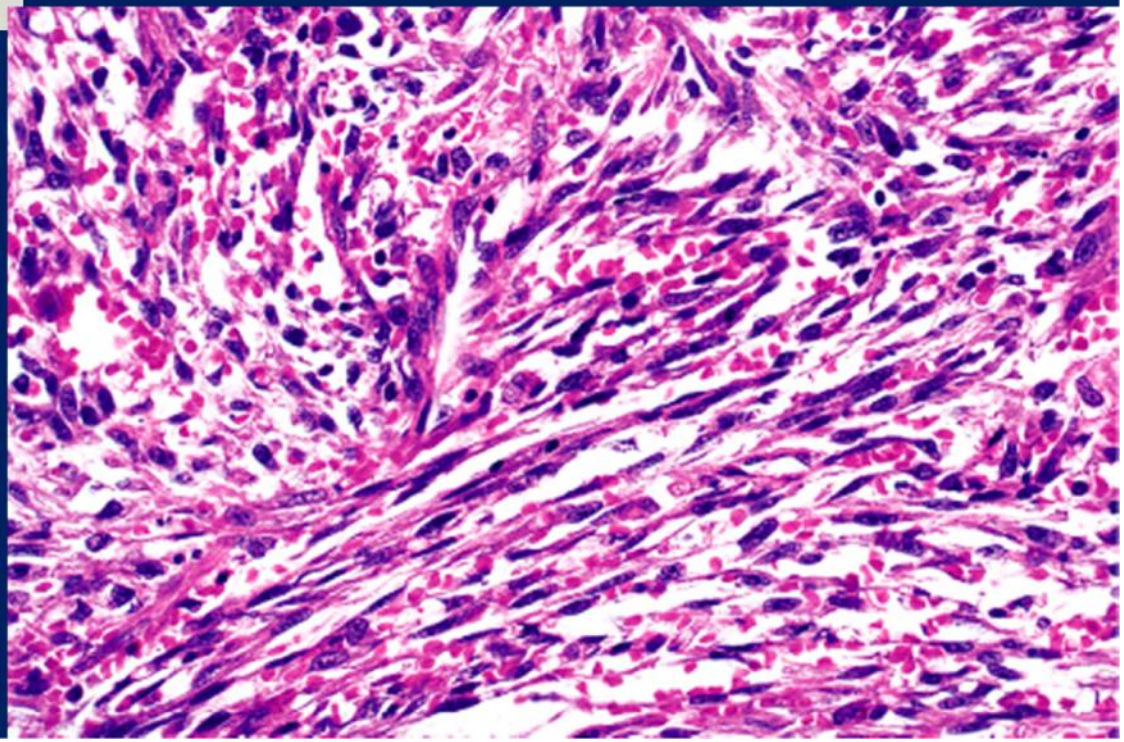
Lt. in the nodular stage, the lesions become nodular, larger, and more numerous.

Describe the salient microscopic features of Kaposi sarcoma in the nodular stage.



Low-power view of a lesion of Kaposi's sarcoma having a prominent nodular shape.

Microscopic appearance of Kaposi’s sarcoma. Elongated spindle cells showing minimal atypia are separated by slits containing red blood cells.



Pathogenesis

Regardless of the clinical subtype (described above), 95% of KS lesions have been shown to be due to *human herpesvirus 8 [HHV-8]* infection.

The virus is transmitted sexually and by poorly understood nonsexual routes.

III. Malignant Tumors

A. Angiosarcomas

- are malignant endothelial neoplasms with histology varying from highly differentiated tumors that resemble hemangiomas to anaplastic lesions. Older adults are commonly affected.
- They occur at any site but most often involve skin, soft tissue, breast, and liver.
- ***Hepatic angiosarcomas*** are associated with carcinogenic exposures, including arsenic (arsenical pesticides), Thorotrast (a radioactive contrast agent formerly used for radiologic imaging), and polyvinyl chloride (PVC; a widely used plastic).
- The increased frequency of angiosarcomas among PVC workers is one of the truly well-documented instances of human chemical carcinogenesis.
- Angiosarcomas can also arise in the setting of **lymphedema**, classically in the ipsilateral upper extremity several years after radical mastectomy for breast cancer; the tumor presumably arises from lymphatic vessels (*lymphangiosarcoma*). Angiosarcomas can also be induced by **radiation.** Clinically, angiosarcomas are locally invasive and can metastasize readily. The current 5-year survival rates approach 30%.

B. Hemangiopericytomas

- *Are* rare tumors derived from **pericytes-myofibroblast-like cells** that are normally arranged around capillaries and venules.
- They are most common on the **lower extremities (especially the thigh)** and in **the retroperitoneum.**
- They consist of numerous branching capillary channels and gaping sinusoidal spaces enclosed within nests of spindle-shaped to round cells.
- The tumors may **recur after excision**, and roughly **half metastasize**, usually **hematogenously to lungs, bone, or liver.**