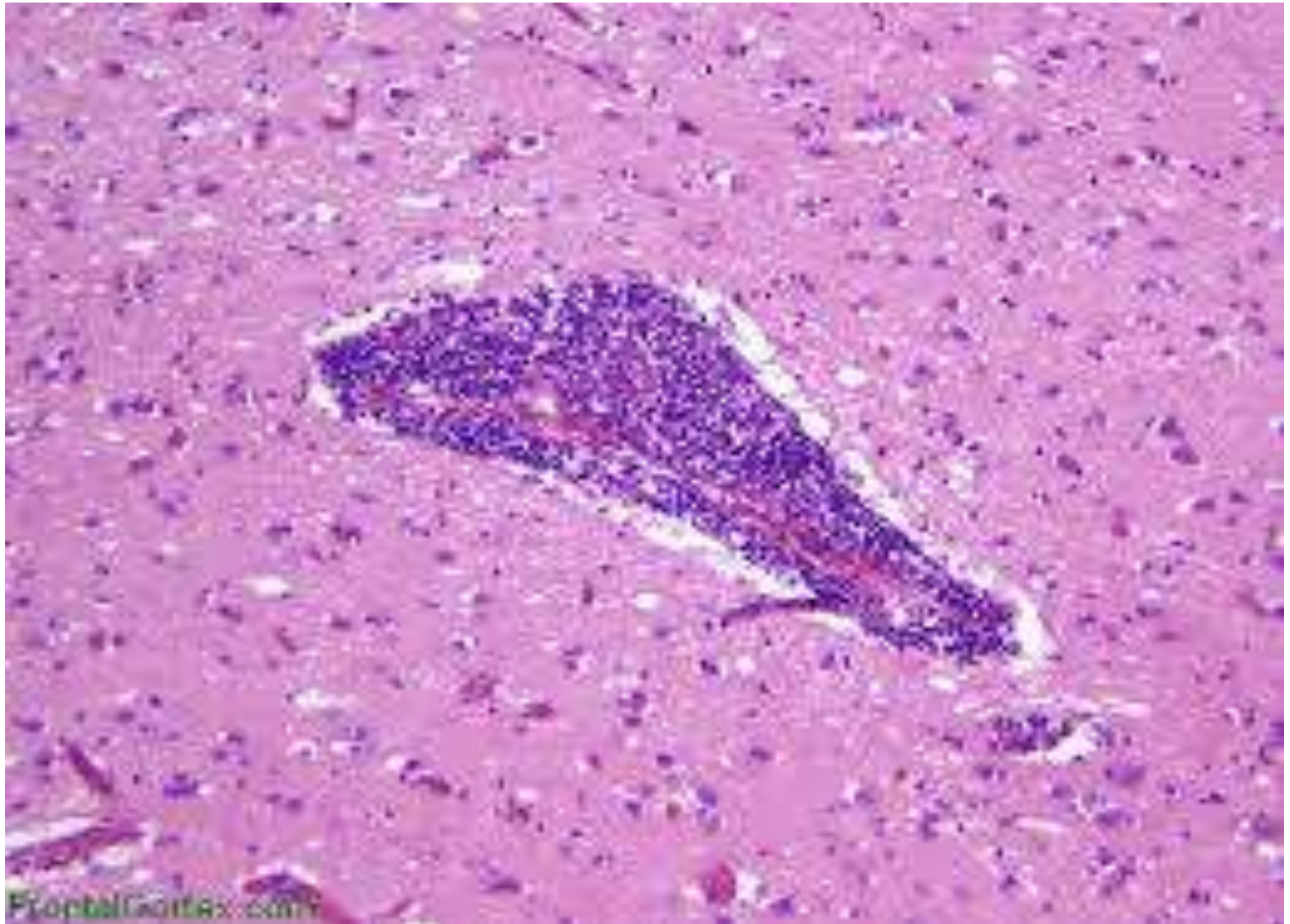


Cerebrovascular diseases-2

Primary angiitis of CNS



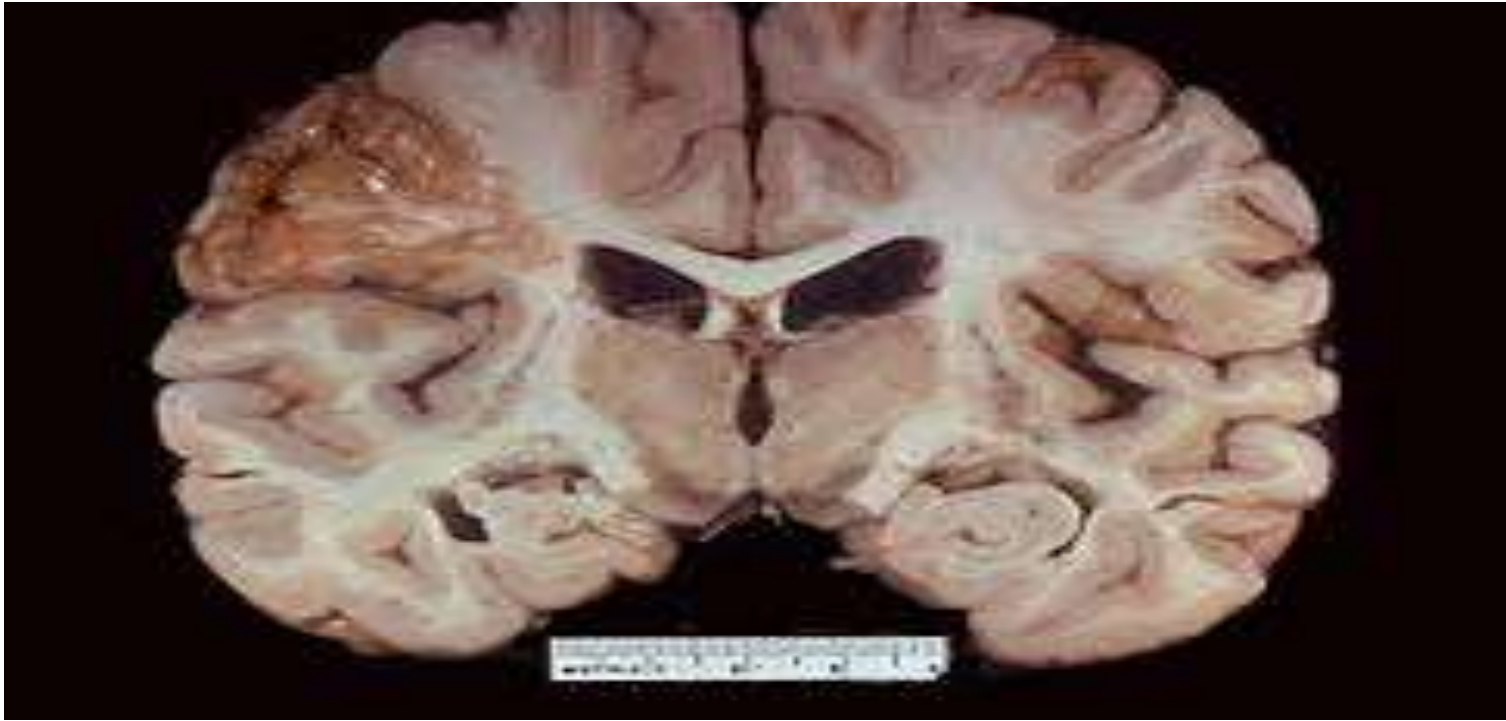
- Other causes of infarction
 - i. Hypercoagulable states
 - ii. Drug-abuse such as amphetamine, heroin and cocaine

Note

- The venous side of the circulation may also undergo thrombosis and cause significant cerebral ischemia
- The striking example is the thrombosis of the superior sagittal sinus which can occur with infections or hypercoagulability state

Morphology of focal cerebral ischemia

- *The macroscopic appearance changes in time.*
 1. During the first 6 hours of irreversible injury, little is observed
 2. By 48 hours
 - The tissue becomes pale, and swollen, as a result of edema



3. From 2 to 10 days

- The brain becomes gelatinous and friable,

4. From 10 days to 3 weeks

- The tissue liquefies, leaving a fluid-filled cavity which represents liquifactive necrosis

Microscopically,

1. After the first 12 hours:

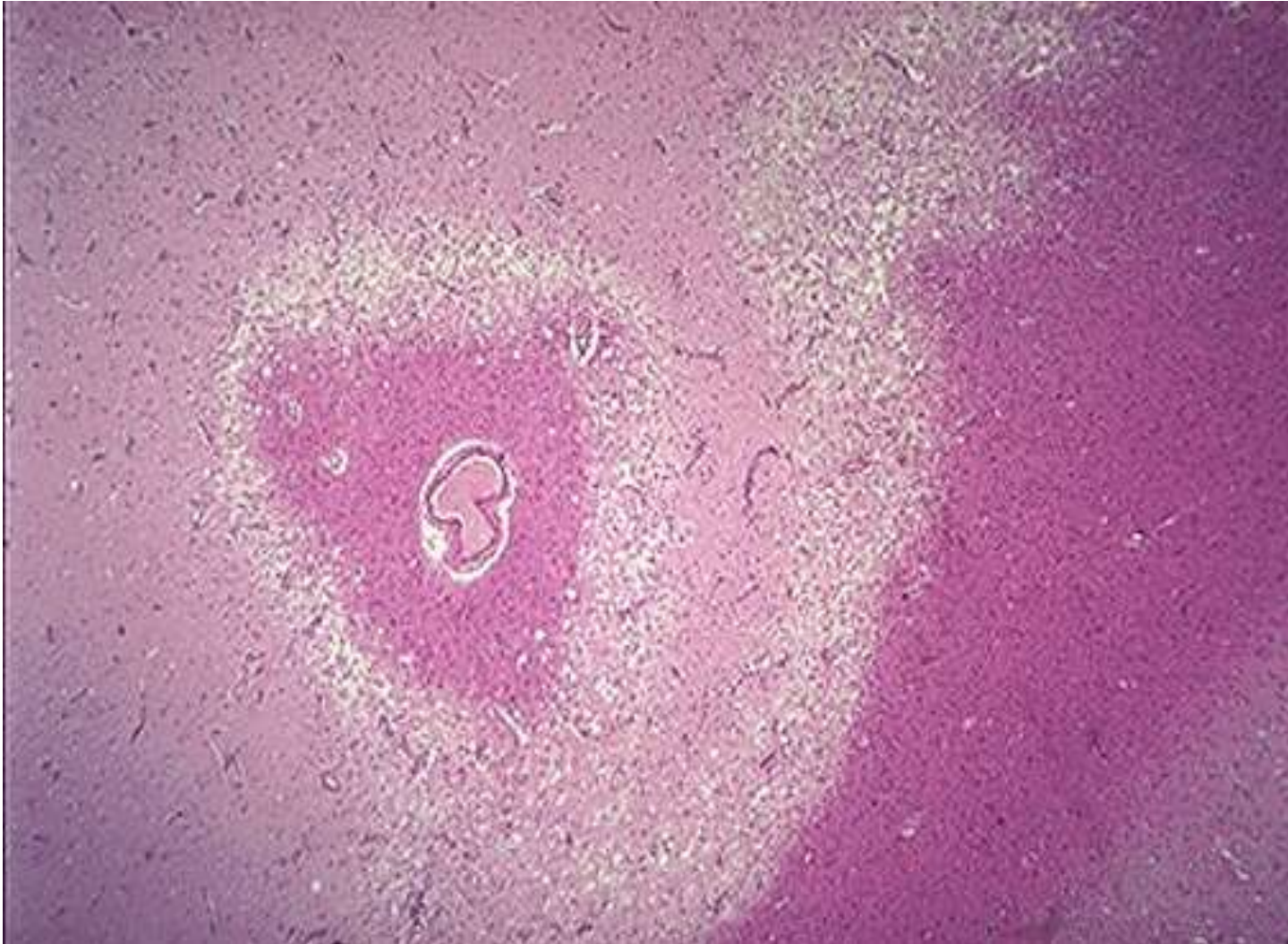
- a. Red neurons with edema
- b. Disintegration and myelinated fibers .

2. Up to 48 hours, there is some neutrophilic emigration

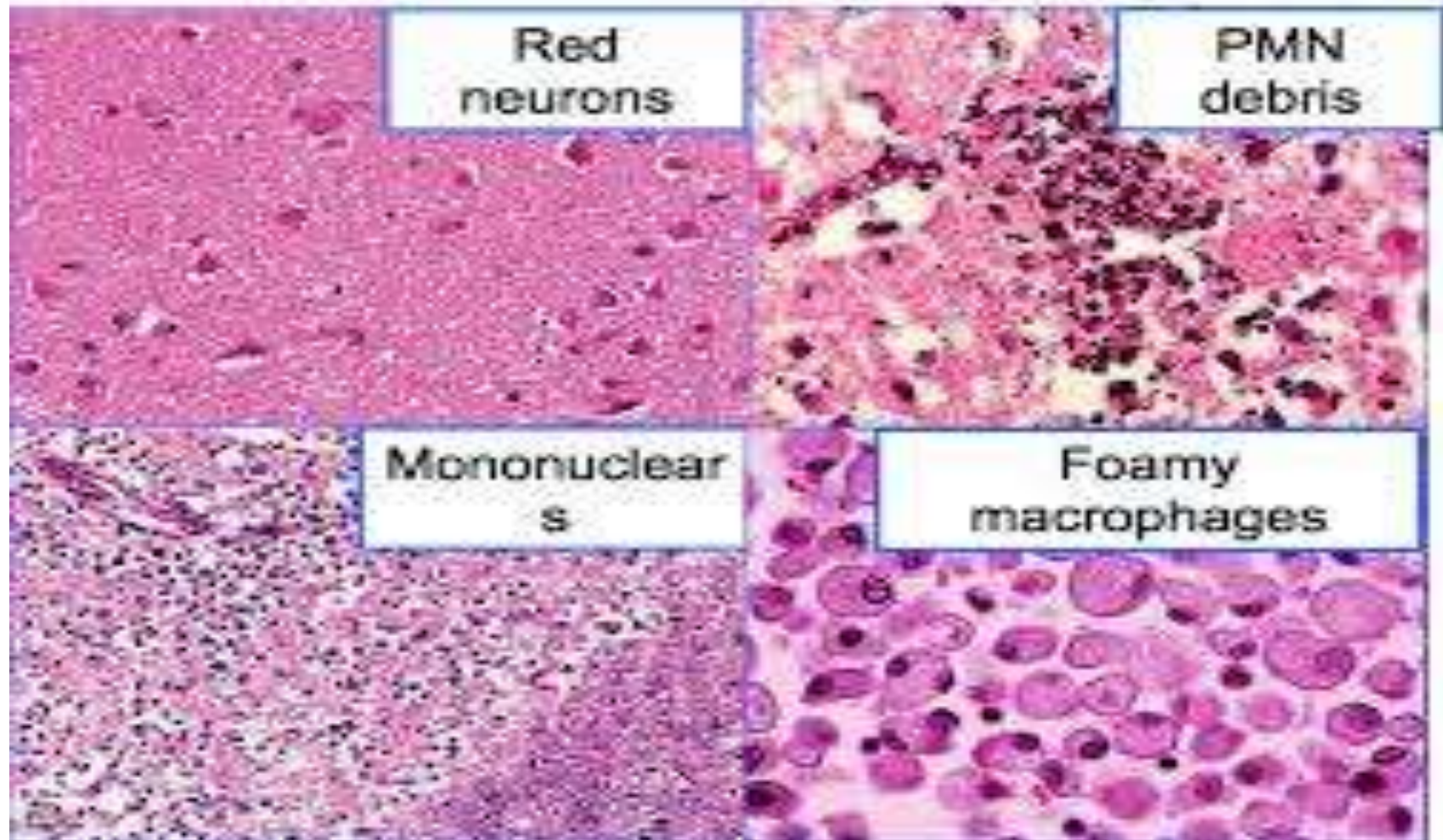
3. 2-3 weeks

- a. Mononuclear phagocytic cells predominate and macrophages containing myelin breakdown products or blood may persist in the lesion for months to years.
- b. Gemistocytic gliosis followed by fibrillary astrogcytosis after several months

Edema in infarction



Microscopic changes in infarction



Notes:

- a. In the cerebral cortex the cavity is delimited from the meninges and subarachnoid space by a gliotic layer of tissue, derived from the molecular layer of cortex.
- b- The pia and arachnoid are not affected and do not contribute to the healing process.

B. Hypertensive cerebrovascular diseases

Effect of hypertension on the brain include:

1. Lacunar infarcts
2. Slit hemorrhages
3. Hypertensive encephalopathy
4. Massive hypertensive intraparenchymal hemorrhage

1. *Lacunes or lacunar infarcts* :

Mechanism

- Hypertension causes arteriolosclerosis of the deep penetrating arteries and arterioles that supply the basal ganglia, hemispheric white matter and brain stem

- This arteriolosclerosis sclerosis leads to occlusion of these vessels
- The result is development of lake-like spaces defined as less than 15 mm wide
- Occur mainly in the
 - i. Lenticular nucleus

ii. Thalamus

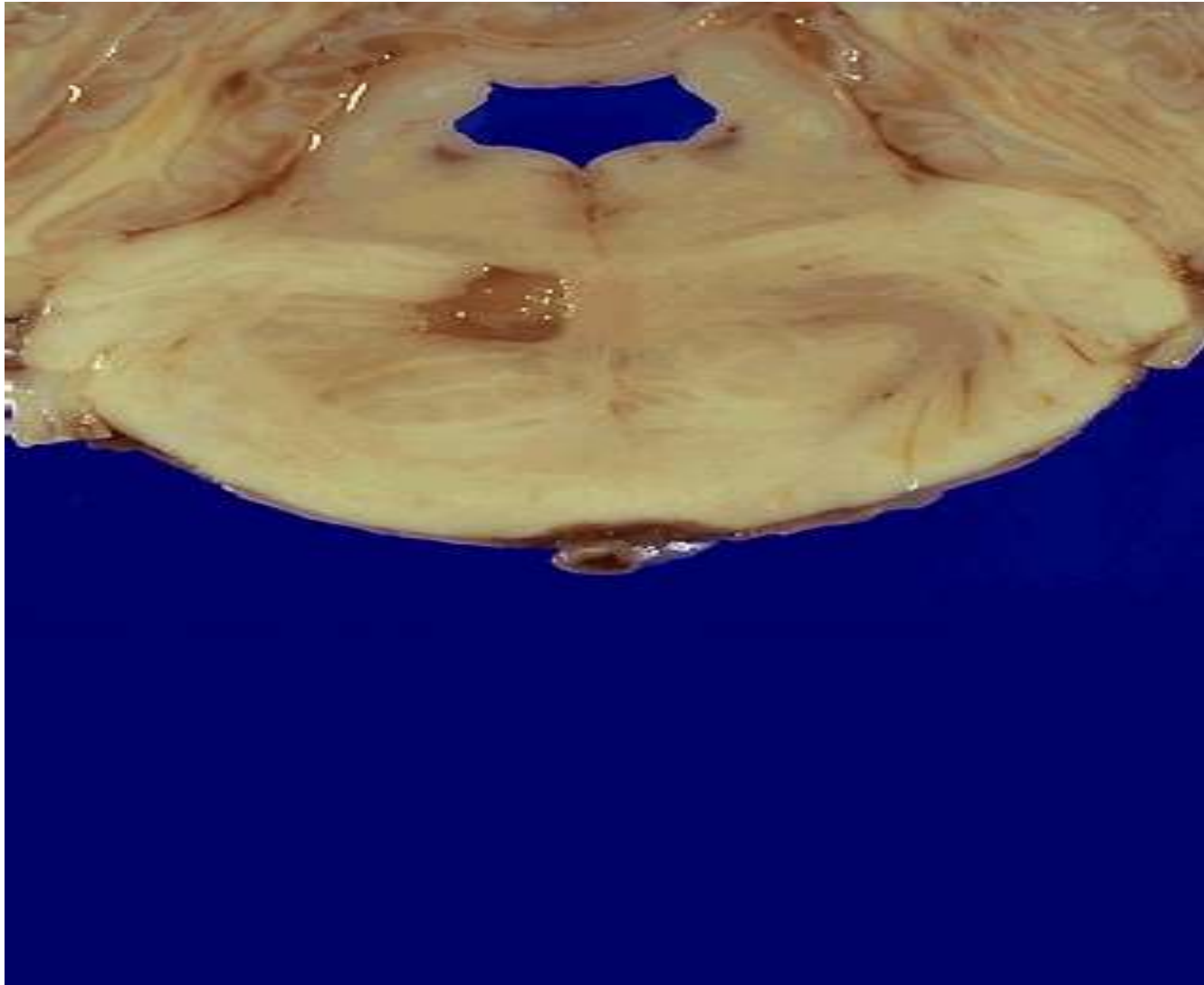
iii. Internal capsule

iv. Caudate nucleus

v. Pons

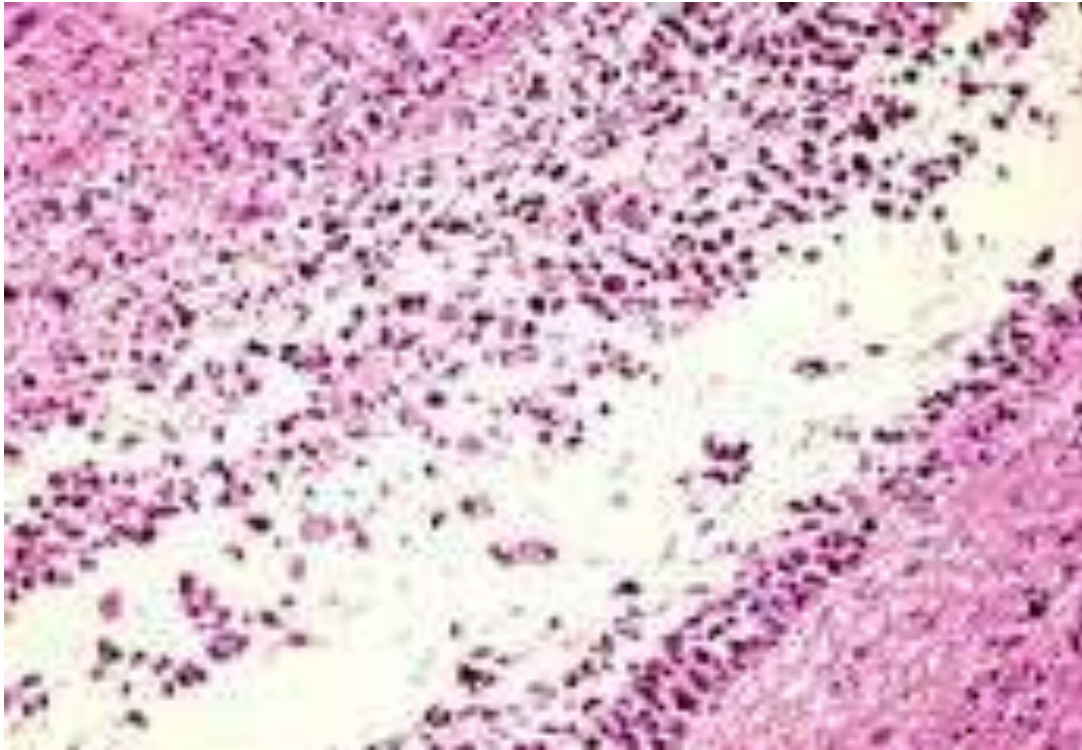
- In descending order of frequency

Lacunar infarct in the Pons



2. Slit hemorrhages;

- Hypertension can lead to rupture of the small caliber blood vessels and lead to development of small hemorrhages
- With time these hemorrhages resorb leaving behind a slit-like spaces called slit hemorrhages



- Microscopically characterized by
 - i. Focal tissue destruction
 - ii. Pigment-laden macrophages
 - iii. Gliosis

3. Hypertensive encephalopathy

- Is a clinico pathologic syndrome arising in the setting of malignant hypertension
- Most often is associated with sudden sustained rises in diastolic blood pressure to greater than 130 mm Hg and characterized

- a. By increased intracranial pressure due to loss of autoregulation and forceful overdistention of blood vessels, leading to fluid extravasation (hydrostatic edema)

- b Global cerebral dysfunction, manifesting as headaches, confusion, vomiting, convulsions, and sometimes coma.
- Rapid therapeutic intervention to reduce the intracranial pressure is essential because this syndrome does not remit spontaneously

- Postmortem examination shows edematous brain with or without transtentorial or tonsillar herniations
 - Microscopic examination shows
 - a. Fibrinoid necrosis and thrombosis of arterioles and capillaries
 - b. and microinfarcts and microhemorrhages
- Microscopic examination shows

C. Intracranial Hemorrhage

■

- May occur at any site within the CNS
either

1. Intraparenchymal hemorrhage

Causes

- a. Hypertension

- b. Amyloid angiopathy

2. Subarachnoid hemorrhages:

- Are most commonly seen with aneurysms but occur also with other vascular malformations.

3. Hemorrhages associated with the dura (in either subdural or epidural spaces) usually due to *trauma*.

1. Intraparenchymal hemorrhages

- Rupture of a small intraparenchymal vessel can lead to intraparenchymal hemorrhage and this condition is called hemorrhagic stroke

- Spontaneous non traumatic brain hemorrhage occur most commonly in the middle to late adult life
- The peak incidence is at about 60 years

- Is divided into

- i. Ganglionic hemorrhages

- Occur in the basal ganglia and thalamus
- Mainly caused by hypertension

- ii. Lobar hemorrhages

- In the cerebral hemispheres
- Main cause by cerebral amyloid angiopathy

Lobar intraparenchymal hemorrhages



- Other causes of intraparenchymal hemorrhages
 - i. Systemic coagulation disorder
 - ii. Neoplasms
 - a. Primary brain tumors mainly glioblastoma and oligodendroglioma
 - b. Metastatic tumors to brain mainly melanoma and renal cell carcinomaiv.
 - iii. Vascular malformations

Hypertensive hemorrhages

- Hypertension is the risk factor for deep brain parenchymal hemorrhages
- Accounts for more than 50% of clinically significant brain hemorrhages
- It accounts for about 15% of deaths among individuals with hypertension

- Sites

- i. Putamen in 50-60% of the case

- ii. Thalamus

- iii. Pons

- iv. cerebellum

Basal ganglia hemorrhage



Mechanisms of massive hemorrhage in Hypertension:

1. Hyaline arteriolar sclerosis

- Affects the deep penetrating arteries and arterioles that supply the basal ganglia and the brain stem
- Affected arteriolar walls are weakened and are more vulnerable to rupture.

2. Chronic hypertension results in formation of minute aneurysms (*Charcot-Bouchard microaneurysms*)
- *Mainly occurs in the basal ganglia*
 - Form in vessels less than 300 μm in diameter

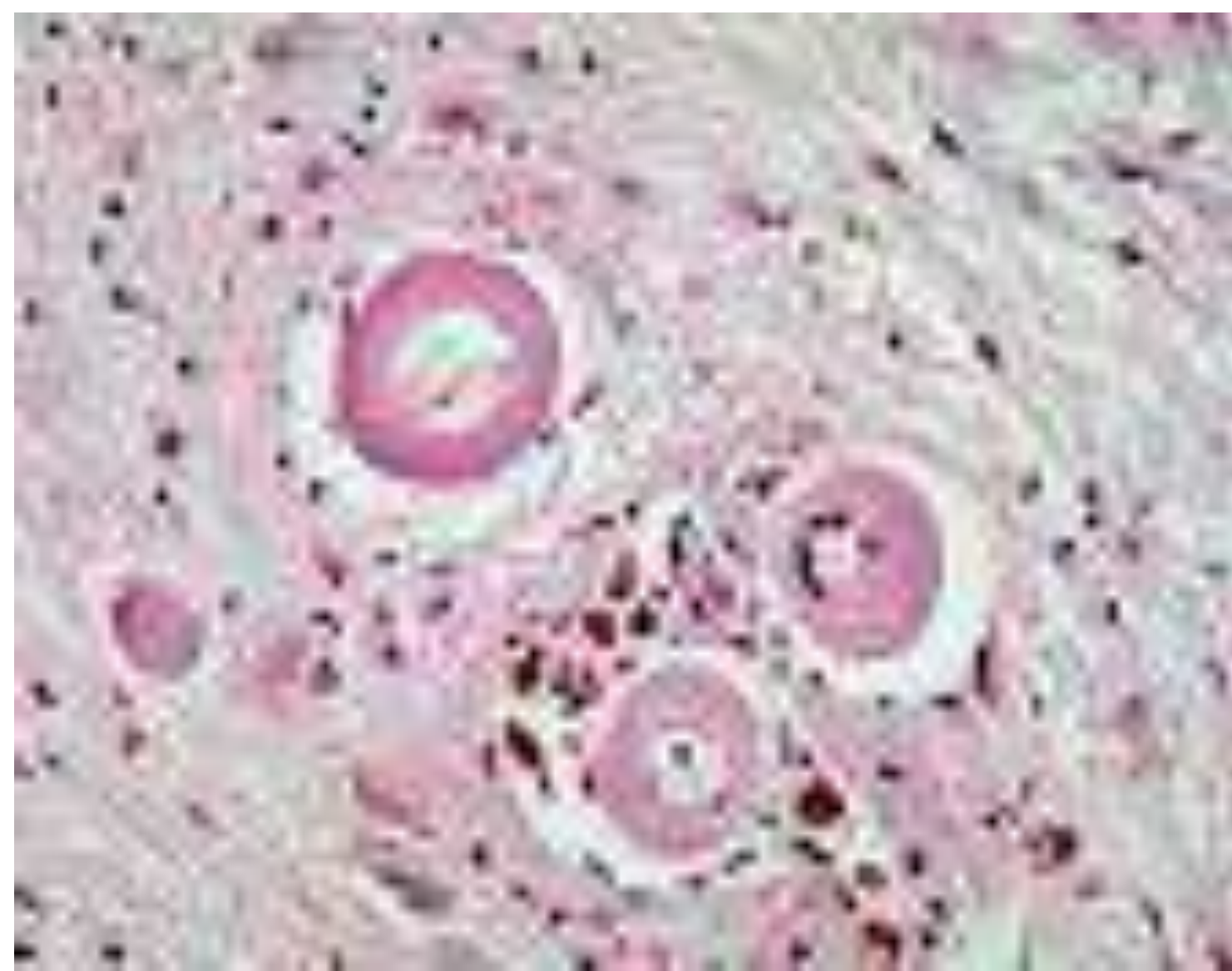
Cerebral Amyloid Angiopathy (CAA) :

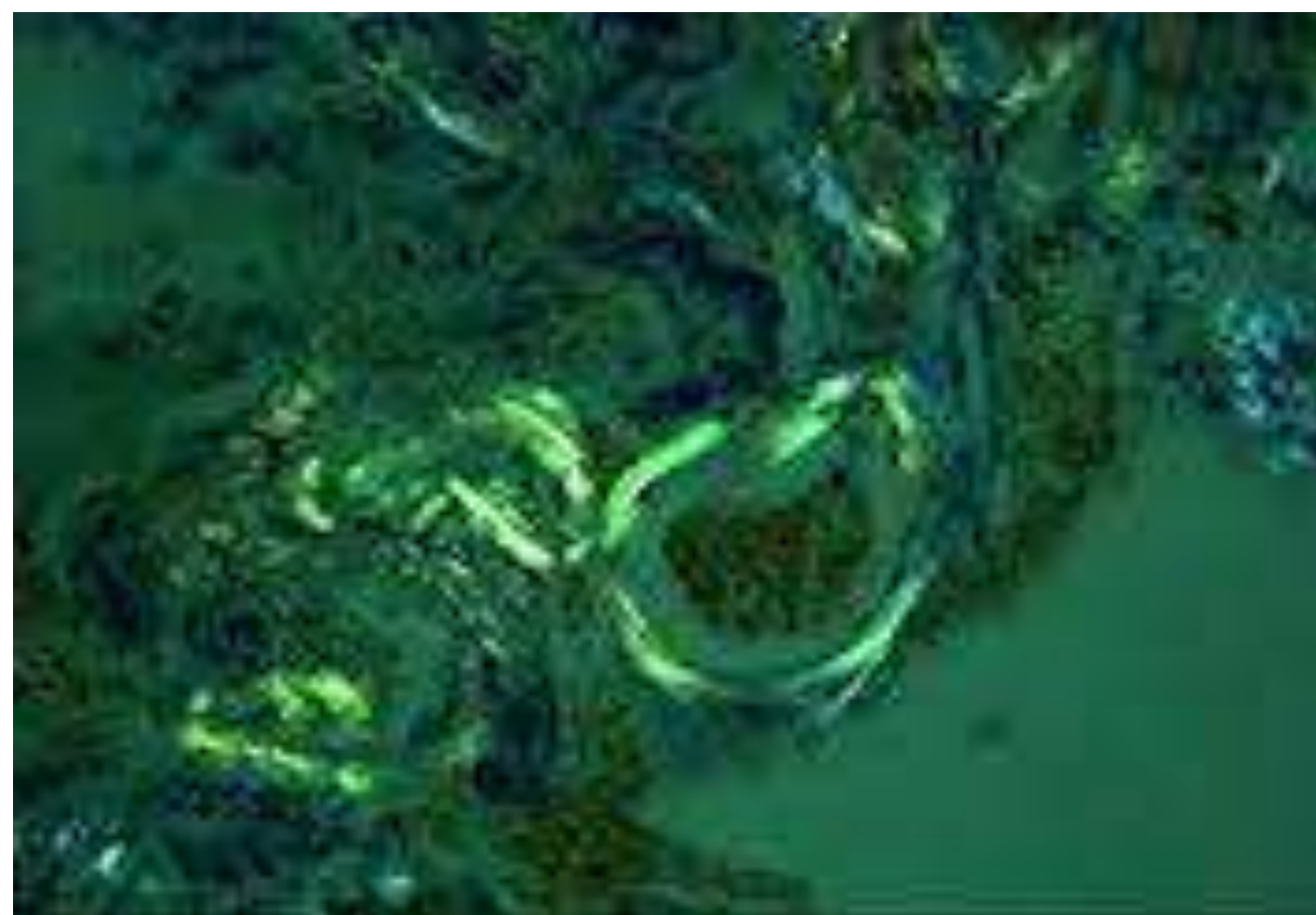
- Is the risk factor most commonly associated with lobar hemorrhages
- In CAA, amyloidogenic peptides usually the same ones in Alzheimer diseases ($A\beta$ amyloid) are deposited in the walls of medium-small caliber meningeal and cortical vessel

- This deposition can weaken the vessel wall and lead to hemorrhage

Morphology

- The underlying vascular abnormality in CAA is typically restricted to the leptomeningeal and cerebral cortical arteioles and capillaries





Subarachnoid Hemorrhage

Causes:

A. Saccular (berry) aneurysm rupture

- Is the most frequent cause of clinically significant subarachnoid hemorrhage

B. Vascular malformation

C. Trauma

D. Rupture of intraparenchymal hemorrhages into the ventricles

1. Berry (saccular) aneurysms

- Rupture of berry aneurysms is the most frequent cause of clinically significant subarachnoid hemorrhage

- Saccular aneurysm is the most common type of intracranial aneurysms
- Other types of aneurysms include:
 1. Atherosclerotic aneurysm , mostly of the basilar artery

2. Mycotic aneurysms
3. Traumatic aneurysms
4. Dissecting aneurysms

Note: Mycotic, traumatic and dissecting aneurysms

1. Arise in the anterior circulation
2. Cause infarction rather than hemorrhage