



Pathology

Cerebrovascular Diseases

Prof. Dr. Azza AbdelAziz Ali

azza3a@mans.edu.eg azza3a@yahoo.com

Learning Outcomes

By the end of the lecture, you will be able to:

- Identify and discuss the different cerebrovascular diseases .

Cerebrovascular Diseases

Hypoxia & Ischemia & Infarction

Intracranial Hemorrhage

Hypertensive Diseases

Aneurysms

Vascular malformations

Cerebrovascular Diseases

Brain disorders involving blood vessels.

1. Hypoxia, Ischemia, and Infarction

- A. Global cerebral ischemia
- B. Focal cerebral ischemia & infarction

2. Intracranial Hemorrhage

- A. Intra-parenchymal, B. Subarachnoid,
C. Subdural, D. Extradural

3. Hypertensive Cerebrovascular Diseases

4. Other Vascular Diseases:

- A. Aneurysms
- B. Vascular malformations
- C. Cerebral amyloid angiopathy
- D. Vasculitis

Hypoxia, Ischemia, and Infarction

Global cerebral ischemia:

Widespread ischemic-hypoxic brain injury.

Cause:

Severe systemic hypotension (systolic pressure below 50 mm Hg) as in cardiac arrest and shock.

Clinical outcome: Varies with severity and duration.

- Mild, transient → complete recovery.
- Moderate → damage to vulnerable areas.
- Severe, long-standing → widespread neuronal death.

Neurons are more liable to hypoxic injury than glial cells.

(most susceptible neurons are the pyramidal cells of the hippocampus and neocortex and Purkinje cells of the cerebellum.)

- Patients who survive suffer severe neurologic impairment.

Hypoxia, Ischemia, and Infarction

Global cerebral ischemia:

- N/E:**
- Brain swelling, wide gyri & narrow sulci
 - Poor grey /white matter demarcation

M/E:

Early changes:

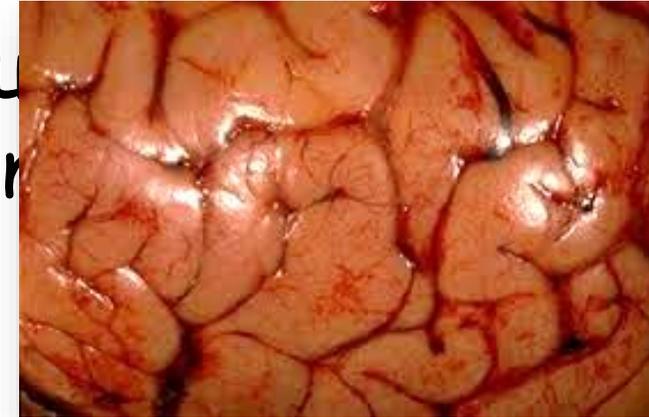
- **Red neurons** (cytoplasmic eosinophilia, nuclear dissolution).
- Similar changes occur in astrocytes and oligodendroglia.

Subacute changes:

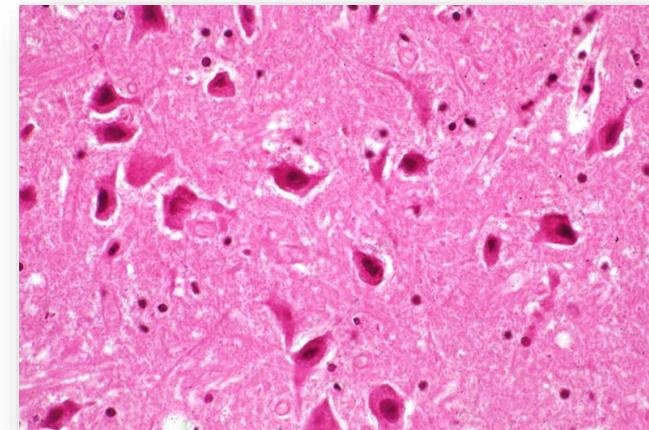
- Necrosis,
- Phagocytic cells remove necrotic tissue.

Repair:

- Loss of organized CNS structure.
- Gliosis.



Brain swelling



Red neurons

Hypoxia, Ischemia, and Infarction

B. Focal Cerebral Ischemia

Leads to infarction (cerebral stroke) in the distribution of the occluded vessel by embolus or thrombus.

*The presence of collaterals protects against infarction:

- **Thalamus, basal ganglia** and **deep white matter** are more affected due to deficient collaterals.
- **Circle of Willis** and **cortical leptomeningeal surface** are protected by collaterals.

***Clinically:** Neurologic deficit according to the area supplied by the affected vessel.

Hypoxia, Ischemia, and Infarction

B. Focal Cerebral Ischemia

Embolic infarcts: More common.

- Source of emboli:

1. Cardiac mural thrombi.

2. Valvular vegetations.

3. Atheromatous plaques within the carotid arteries or aortic arch.

Thrombotic infarcts: Less common.

- Sources of thrombi:

1. Thrombosis on top of atherosclerosis of the origin of the middle cerebral artery, and end of the basilar artery.

2. Thrombi of dural venous sinuses or deep cerebral veins (less common).

Hypoxia, Ischemia, and Infarction

B. Focal Cerebral Ischemia

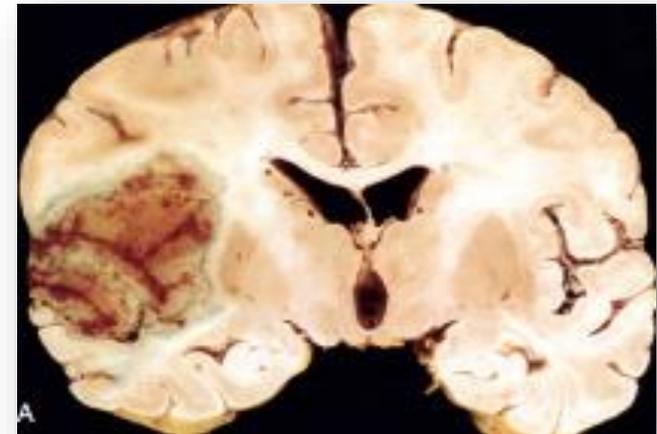
NE: Types and morphology of infarcts:

Non-hemorrhagic Infarct:

- Early: Pale, opaque.
- Liquefactive necrosis → creamy fluid-filled cavity.
- Healing → destruction of cortex + gliosis.

Hemorrhagic infarct: Due to:

- Reperfusion of ischemic tissue (collaterals or therapeutic/spontaneous dissolution of emboli).
- Infarcts caused by venous occlusion.

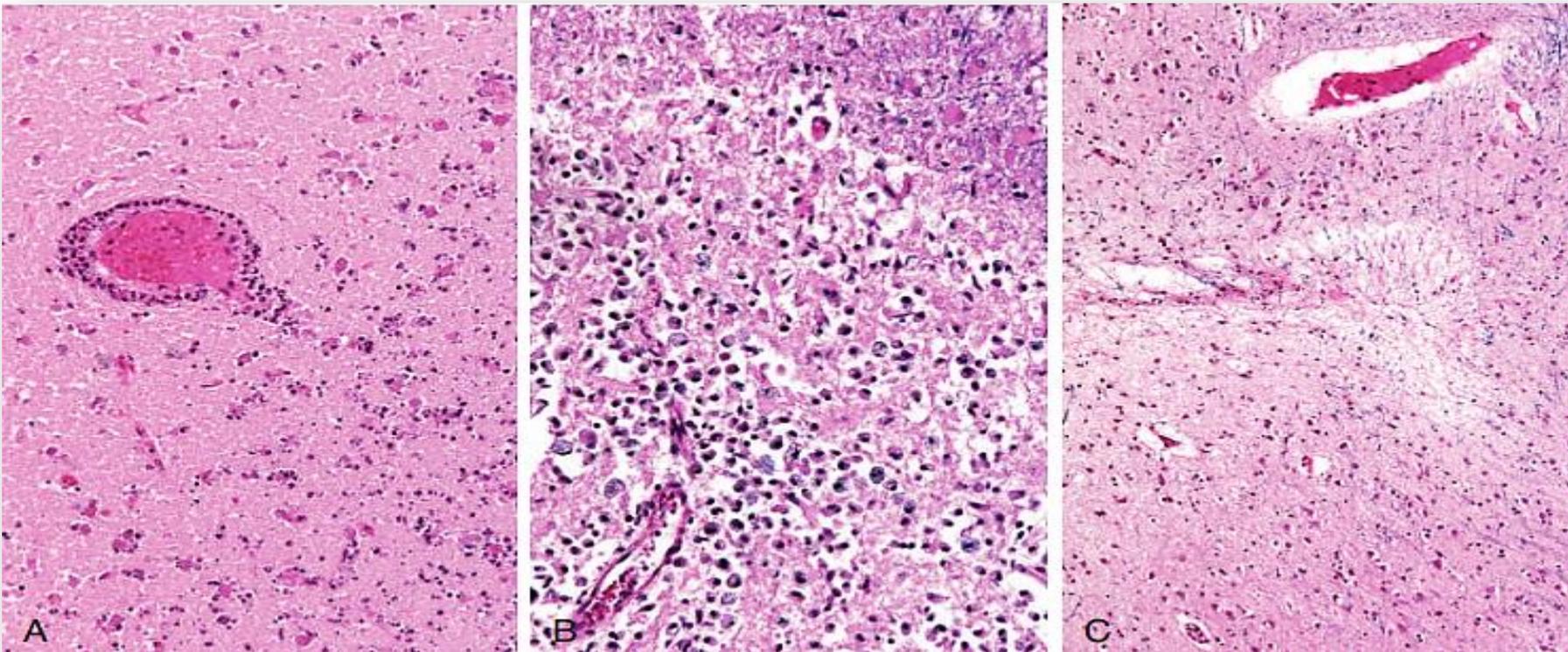


Hypoxia, Ischemia, and Infarction

B. Focal Cerebral Ischemia

ME: Cerebral infarction stages:

- A. Early infarct (red neurons) with neutrophil infiltration
- B. By day 10, influx of phagocytic cells
- C. Old infarct with tissue loss and residual gliosis



Intracranial Hemorrhage

A. Intra-parenchymal hemorrhage:

- Most common in hypertension.
- Atherosclerosis.
- Tumors.
- Cerebral contusions (trauma).
- Clotting disorders.
- Cerebral amyloid angiopathy.
- Vascular malformations.



B. Subarachnoid hemorrhage:

- Ruptured aneurysms
- Vascular malformations
- Brain trauma.



Intracranial Hemorrhage

C. Subdural hemorrhage (hematoma):

- Caused by rupture of venous sinuses or bridging subduralcerebral veins.
- Common in: old patients with brain atrophy, alcoholics.



D. Epidural (Extradural) hemorrhage :

- Caused by traumatic skull fracture, especially temporal or parietal bones (tear of middle meningeal artery).
- Expands rapidly (arterial bleeding) with rapid increase of ICP
- It is a neurosurgical emergency (drainage and repair to prevent death).

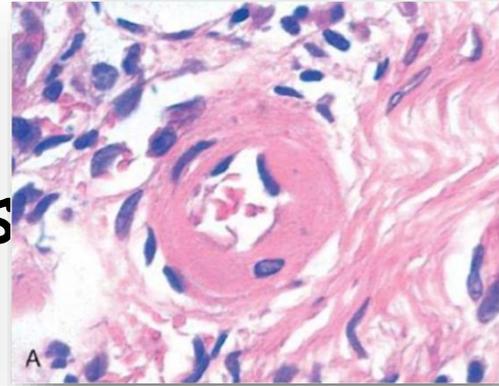


Hypertensive Cerebrovascular Diseases

Sites: Thalamus, basal ganglia, deep white matter, internal capsule and pons.

Lesions:

1. Hyaline arterio-sclerosis of cerebral arterioles
2. Charcot-Bouchard microaneurysms,
3. Lacunar infarcts: few millimeters in size,
4. Slit hemorrhages: Rupture of the small penetrating vessels,
5. Massive intra-parenchymal hemorrhage: arterial
6. Cerebral edema,
7. Acute hypertensive encephalopathy: if diastolic blood pressure exceeds 130 mm Hg. (mostly) → cerebral dysfunction, headaches, confusion, vomiting, convulsions, and sometimes coma.



**Hyaline
arteriosclerosis**

Cerebral Aneurysms

Types:

1. Congenital berry aneurysms:
2. Microaneurysms of essential hypertension (Charcot-Bouchard).
3. Atheromatous aneurysms.
4. Traumatic aneurysms.
5. Mycotic aneurysms: emboli of bacterial endocarditis.

Clinical effects:

- Pressure on the surrounding,
- Rupture and subarachnoid or intraparenchymal hemorrhage.
- Thrombosis and calcifications lead to acute ischemia (Infarction) or chronic ischemia (brain atrophy).

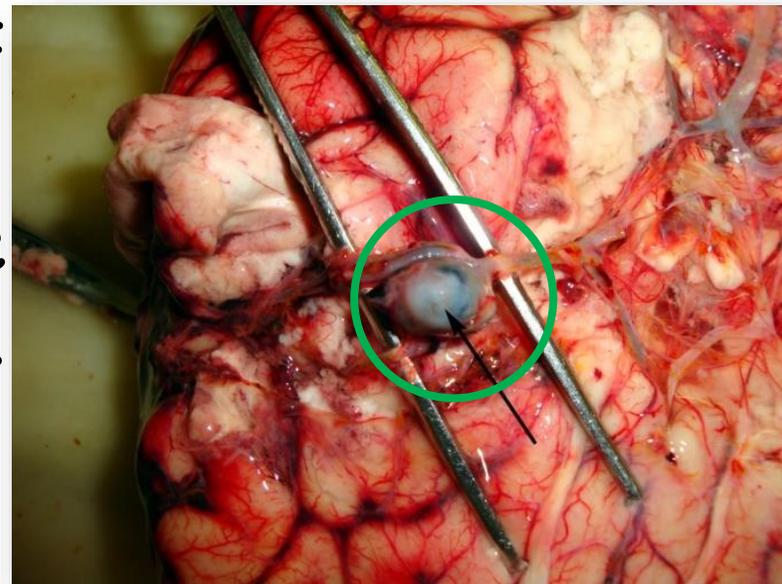
Cerebral Aneurysms

Congenital saccular berry aneurysms:

Site: circle of Willis (small, multiple).

Pathology: internal elastic lamina & medial weakness at bifurcation of arteries causing saccular aneurysm.

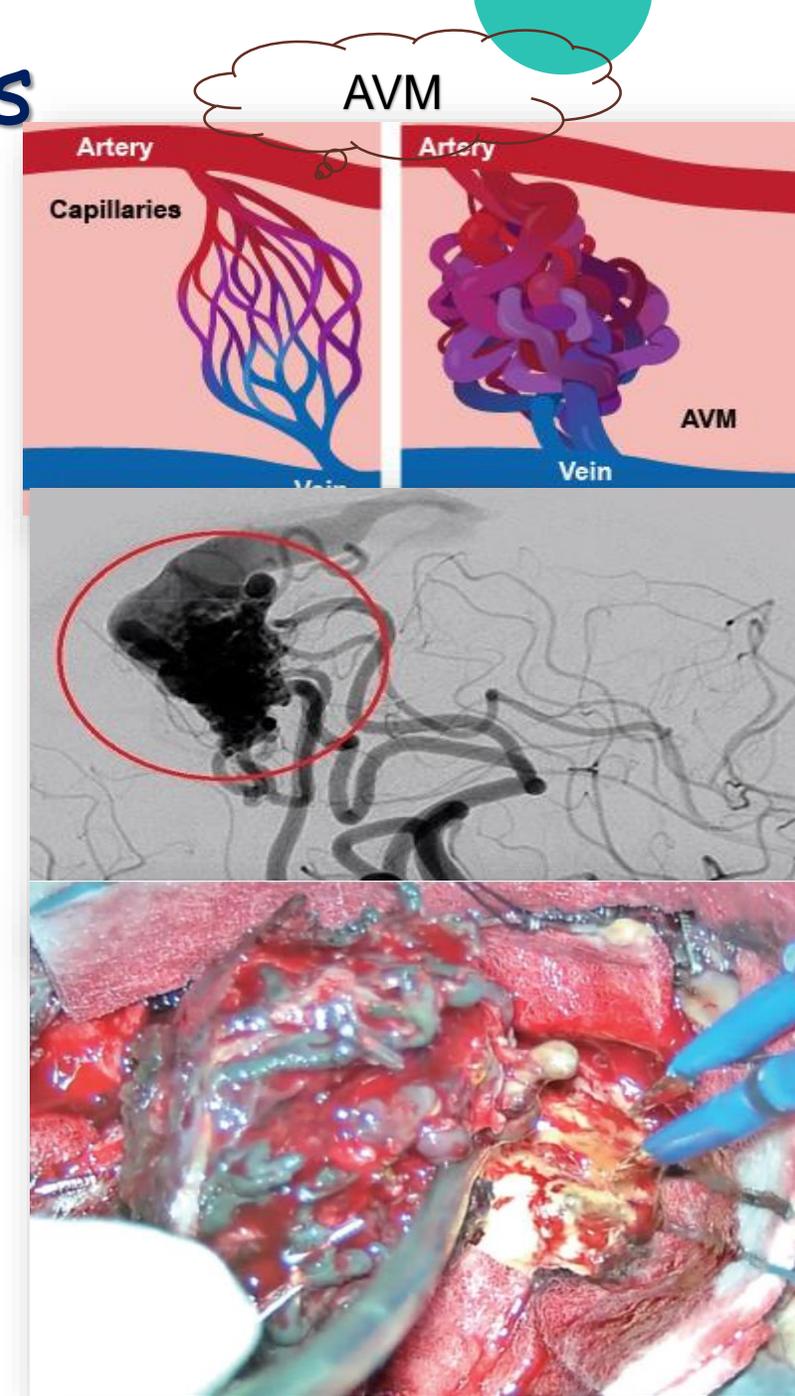
- Most frequent cause of spontaneous subarachnoid hemorrhage (sudden severe headache and loss of consciousness).
- Rupture associates straining (increase intracranial pressure. e.g. constipation, severe cough.....).



Vascular Malformations

Variants:

1. Arterio-venous malformations (AVMs)
 2. Cavernous malformations (hemangiomas),
 3. Capillary telangiectasias,
 4. Venous angiomas.
- The most dangerous malformation is **AVM**.
 - Appears as tangled network of worm-like vascular channels.
 - Leads to: mixed intra-parenchymal or subarachnoid hemorrhages



Now....Answer this

In focal cerebral ischemia (infarction), which of the following is less likely to be affected?

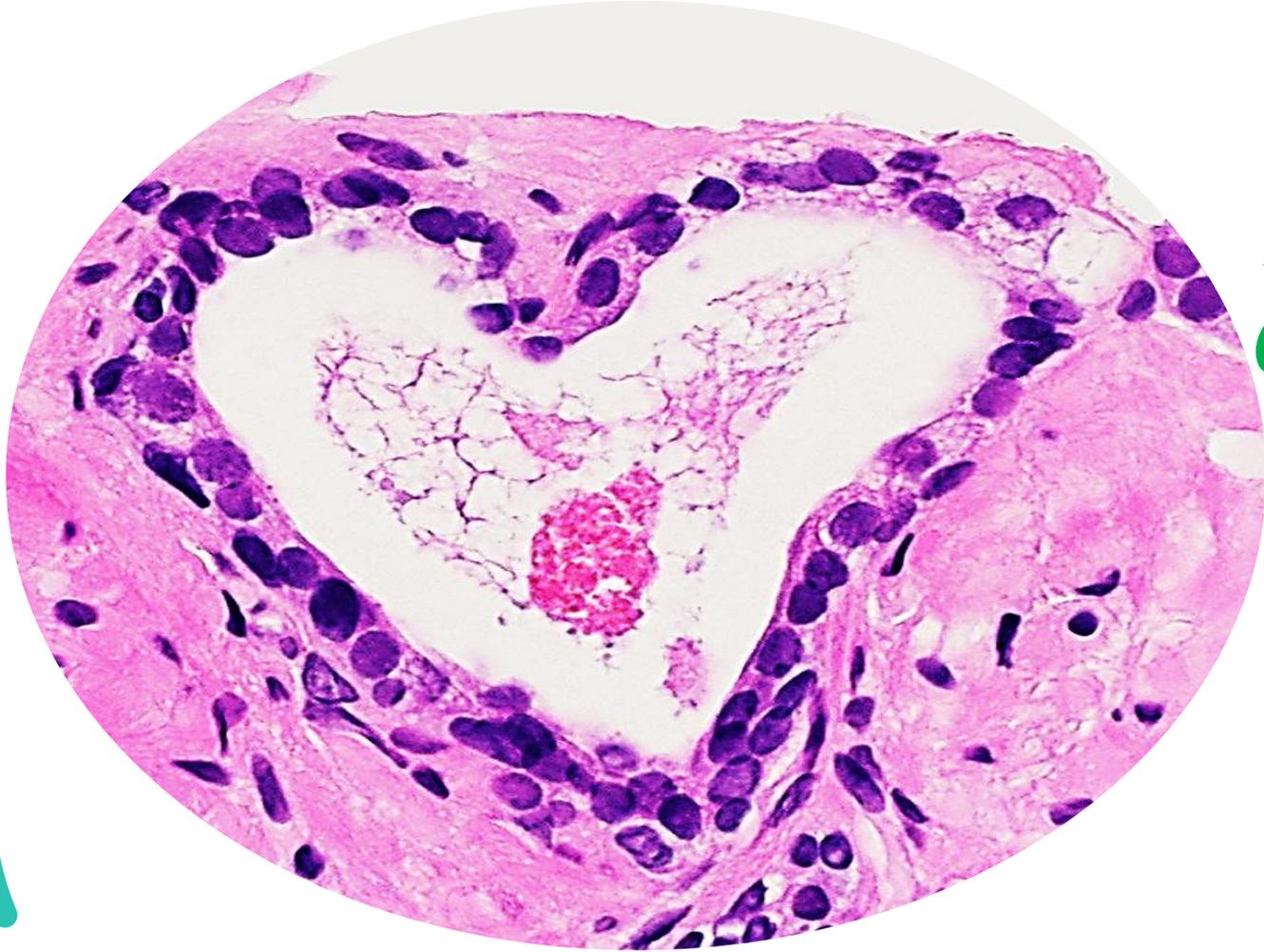
- A. Basal ganglia.
- B. Thalamic nuclei.
- C. The deep white matter.
- D. Submeningeal cortex.
- E. Deep nuclei.



Discussion & Feedback

References & recommended readings

1. Robbins & Cotran Pathologic Basis of Disease, (Robbins Pathology), 2018 ISBN: 978-0-323-35317-5, Edition: 10th
2. Webpath: <https://webpath.med.utah.edu/webpath.html>
<https://www.pathologyatlas.ro/index.php>



Thank you