Neuropathology

Cerebrovascular diseases (I)

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Cerebrovascular diseases

Major cause of death

3 main pathogenic mechanisms leading to stroke

- Thrombotic occlusion
- Embolic occlusion

} Infarction or ischaemic injury

- Vascular rupture

Ischaemic injury/infarction

- Focal
  - Blood vessel occlusion
- Global
  - Complete loss of perfusion
  - Severe hypoxaemia
  - Profound hypoglycaemia

Mechanisms of oxigen deprivation

- Functional hypoxia
  - low partial pressure of oxigen (high altitude)
  - Impaired oxigen-carrying capacity (anaemia, CO- poisoning)
  - Inhibition of oxigen use (cyanide poisoning)
- Ischaemia – transient/permanent
  - Hypoperfusion (hypotension, vascular obstruction)

Global cerebral ischaemia

- Caused by severe systemic hypotension (sys <50 mm Hg)
  - Cardiac arrest
  - Shock

- Outcome: complete recovery --- severe neurological impairment/death

Global cerebral ischaemia - macroscopy

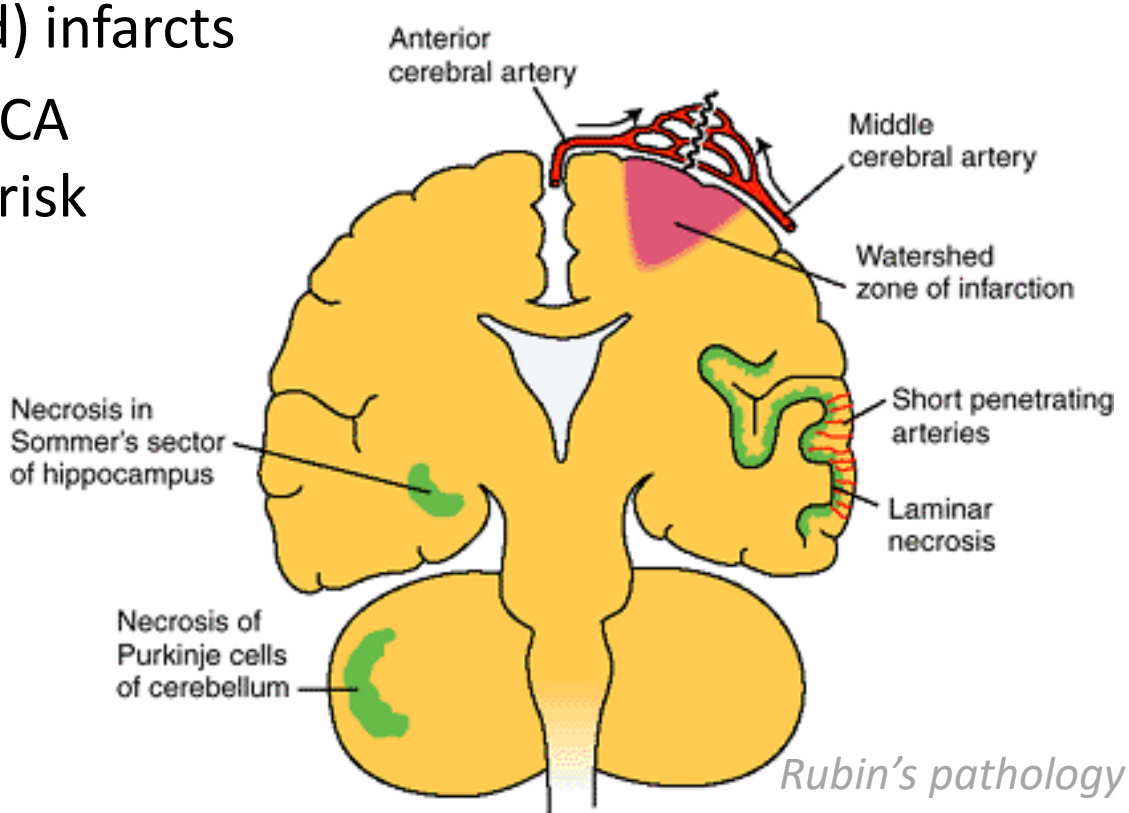
Most susceptible neurons

pyramidal cells of hippocampus and neocortex, Purkinje cells

Most susceptible areas

are the most distal portions of arterial territories (border zones).

- border zone (watershed) infarcts
- border zone between ACA and MCA is at greatest risk
- wedge shaped

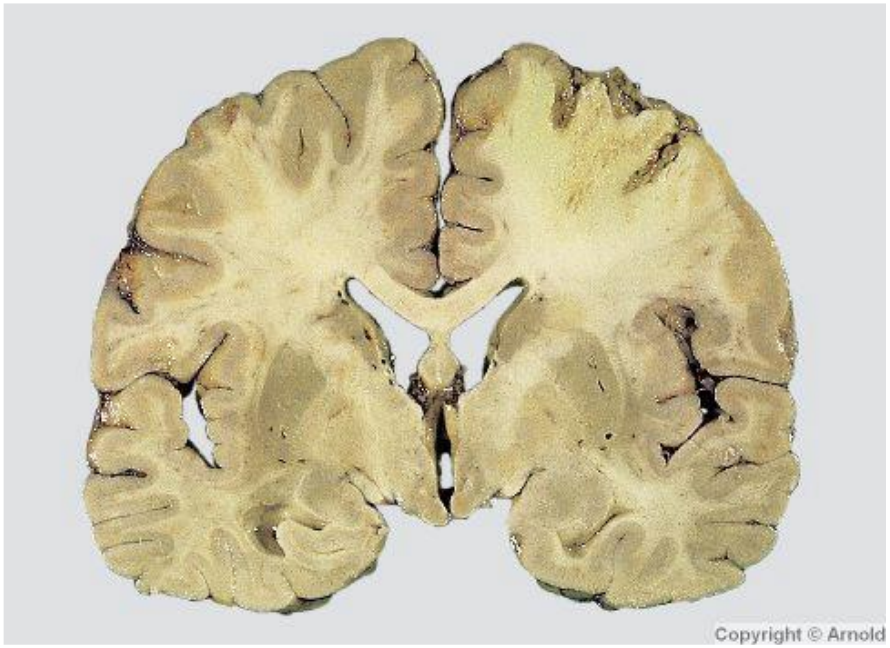


Rubin's pathology

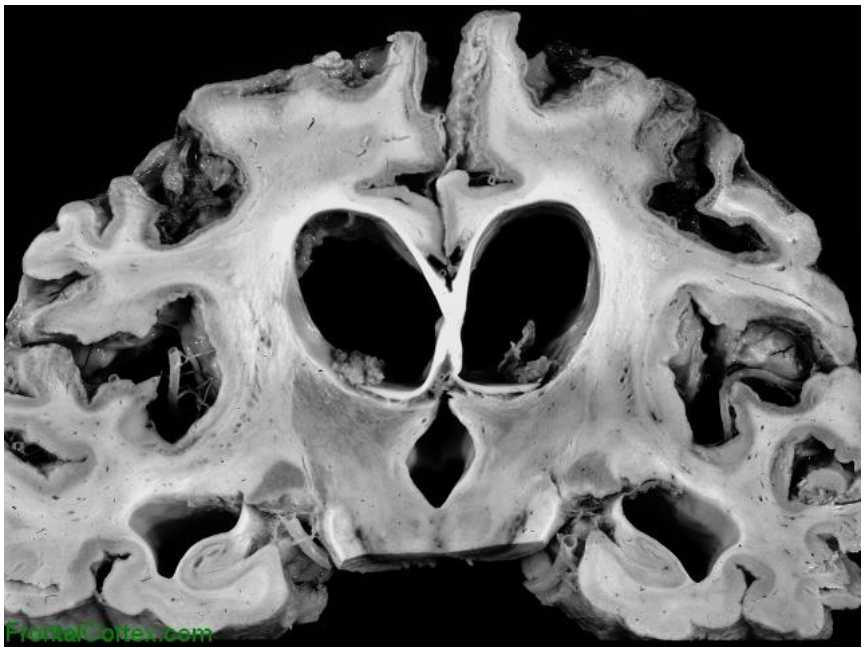
Global cerebral ischaemia - macroscopy

- Swollen brain
- Poor demarcation between gray and white matter

Watershed infarct



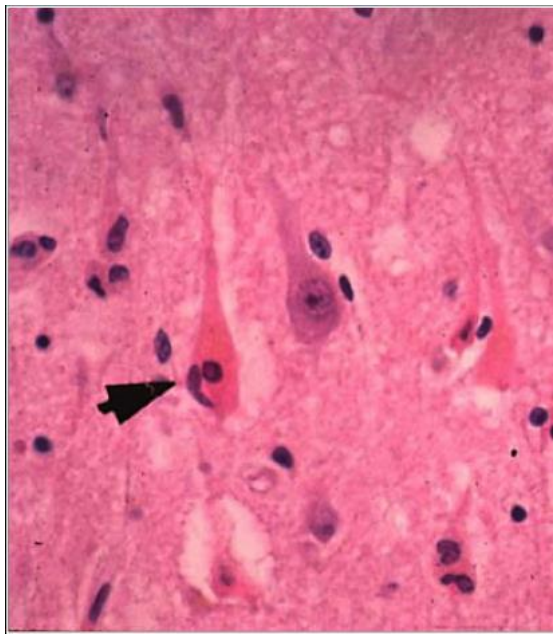
Laminar necrosis

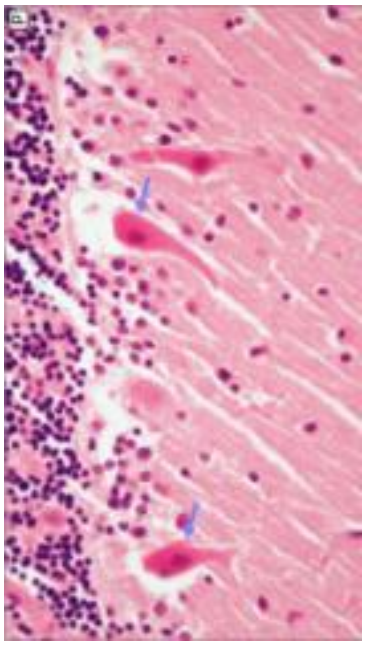


Global cerebral ischaemia - microscopy

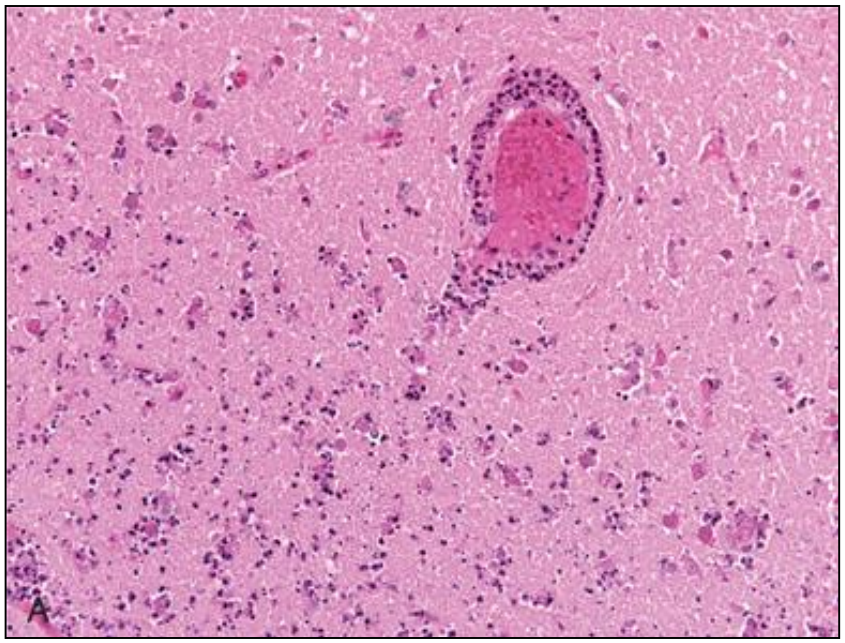
Early changes (12-24 hours)

red neurons
(microvesiculation, cytoplasmic eosinophilia, nuclear pyknosis and karyorrhexis)
similar changes in astrocytes & oligodendrocytes





neutrophil infiltration

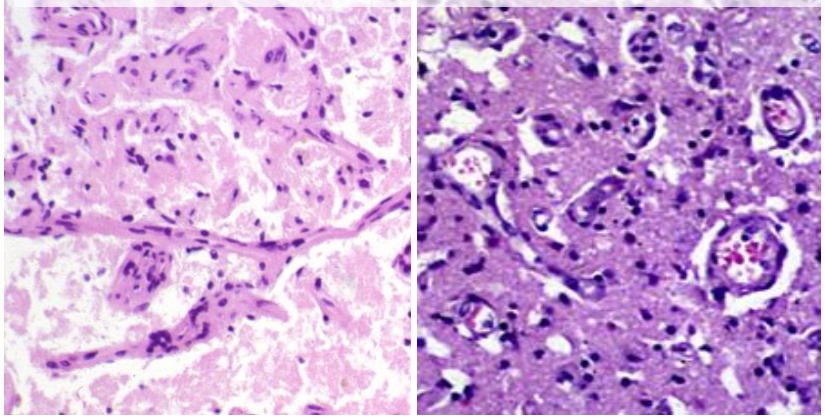


Robbins Basic Pathology

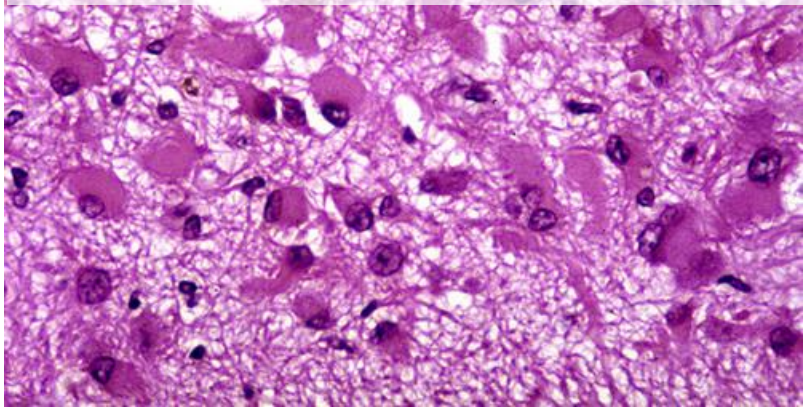
Global cerebral ischaemia - microscopy

Subacute changes (24h – 2 weeks)

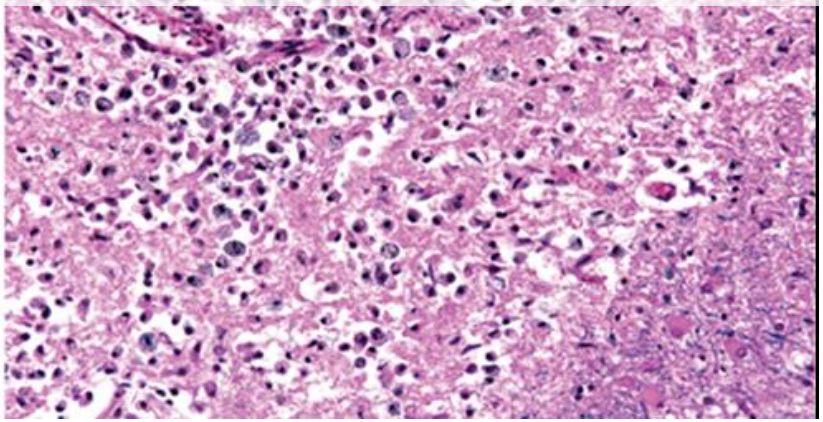
tissue necrosis, vascular proliferation



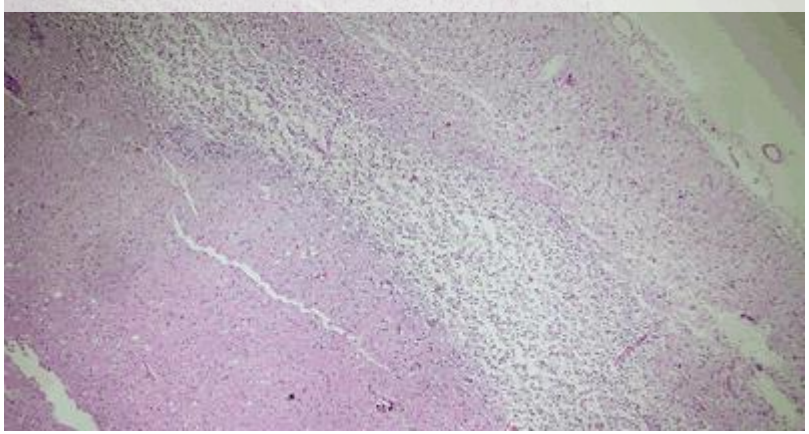
reactive gliosis



macrophage infiltration



pseudolaminar necrosis (deep layers, V-VI)



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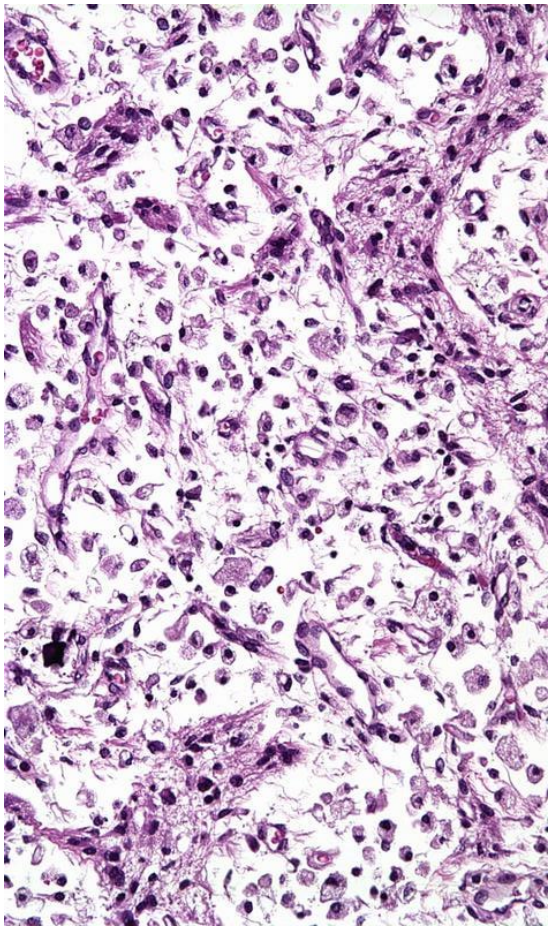
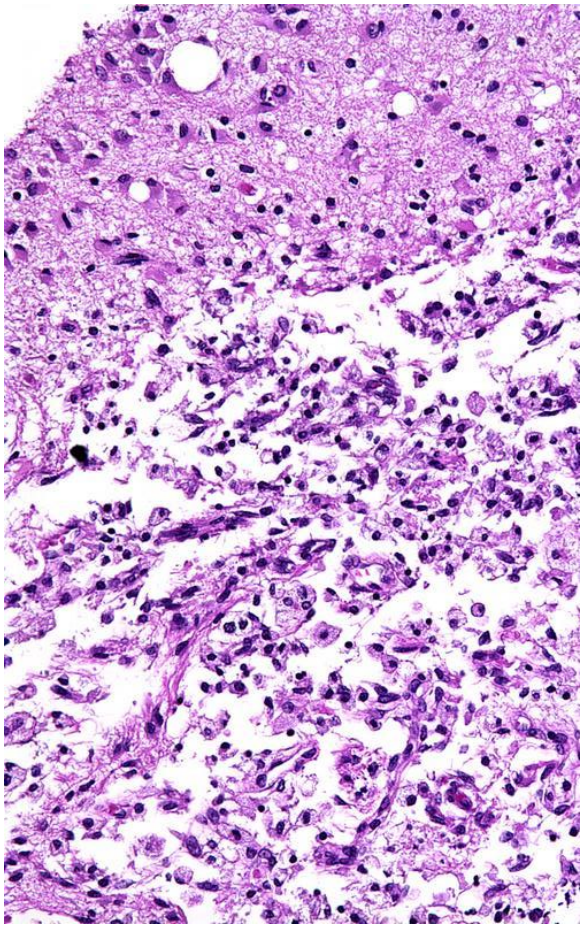
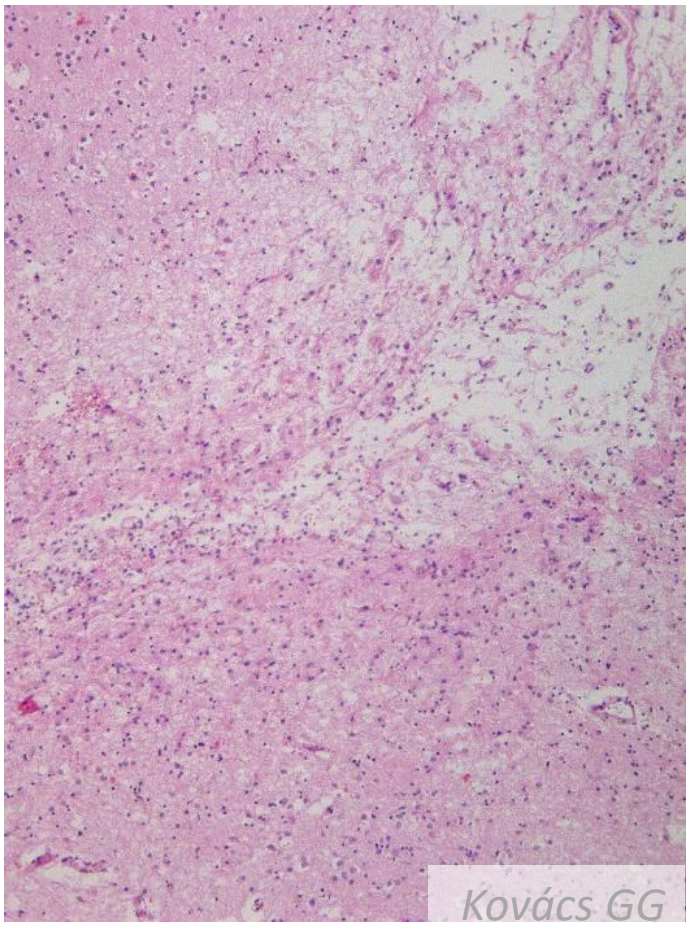
Global cerebral ischaemia - microscopy

Repair (after 2 weeks)

removal of necrotic tissue, loss of organized CNS structure, gliosis

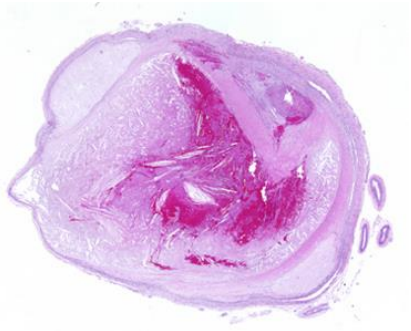






Focal cerebral ischaemia

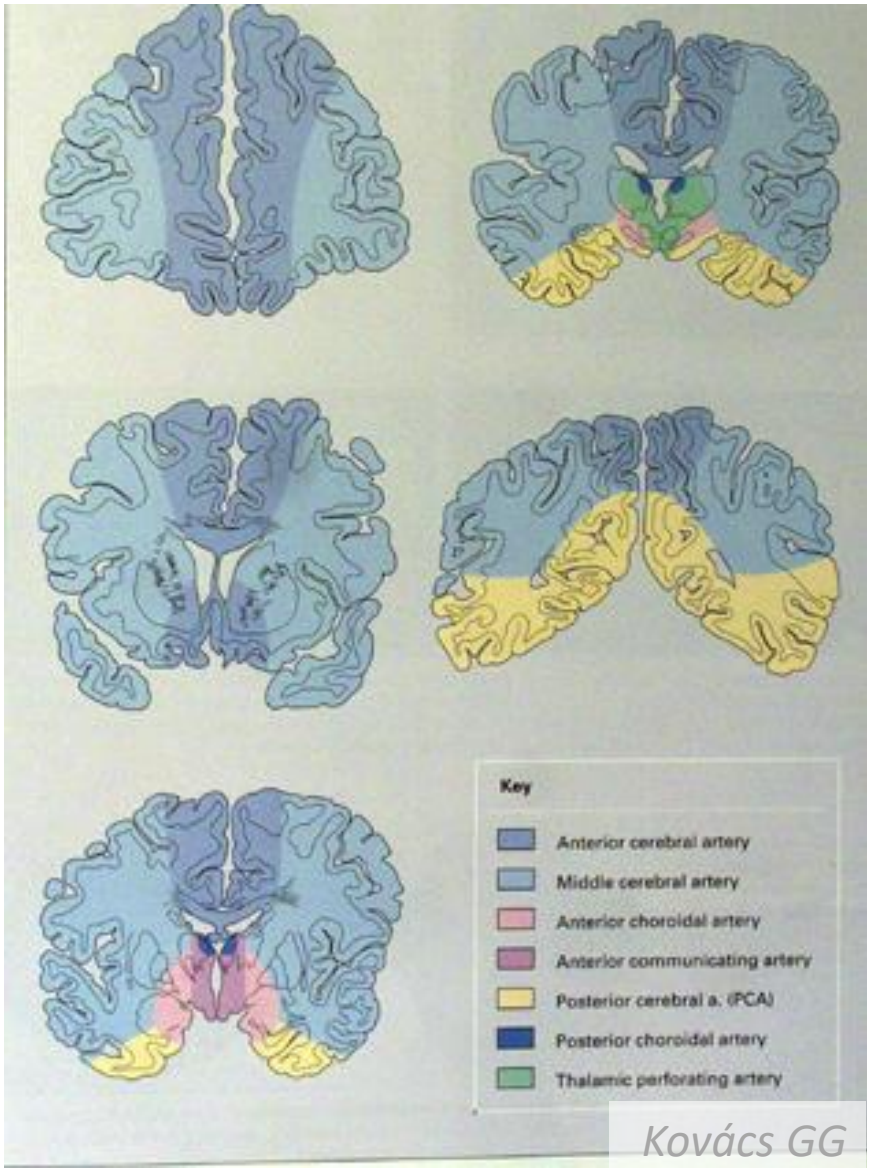
- Caused by vascular occlusion
  - Embolia
    - thromboemboli (heart, carotid arteries, paradoxical embolism from deep veins)
    - The territory or middle cerebral artery is most frequently affected
    - Fat emboli, air emboli, septic emboli, etc.
  - Thrombosis – superimposed on atherosclerotic plaques
    - Carotid bifurcation
    - Origin of MCA
    - Basilar artery



Focal cerebral ischaemia

- The extent of tissue damage may be modified by collateral blood flow (circle of Willis, cortical-leptomeningeal anastomoses)
- There is little collateral blood flow to the thalamus, basal ganglia, deep white matter

- Nonhaemorrhagic infarcts
- Haemorrhagic infarcts – reperfusion from collaterals or after dissolution of emboli (venous obstruction)



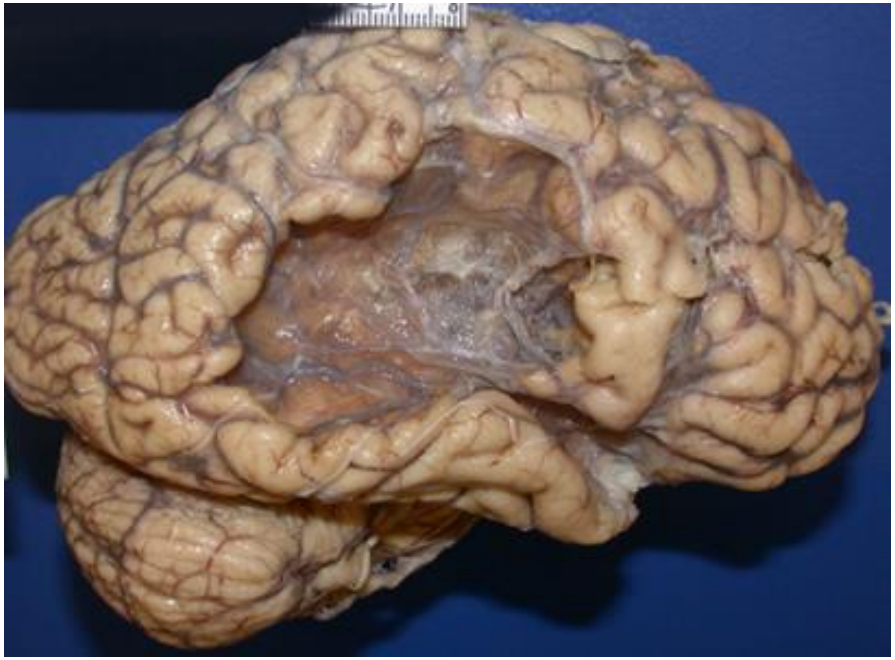
Nonhaemorrhagic infarct - macroscopy

Unchanged for 6 hours

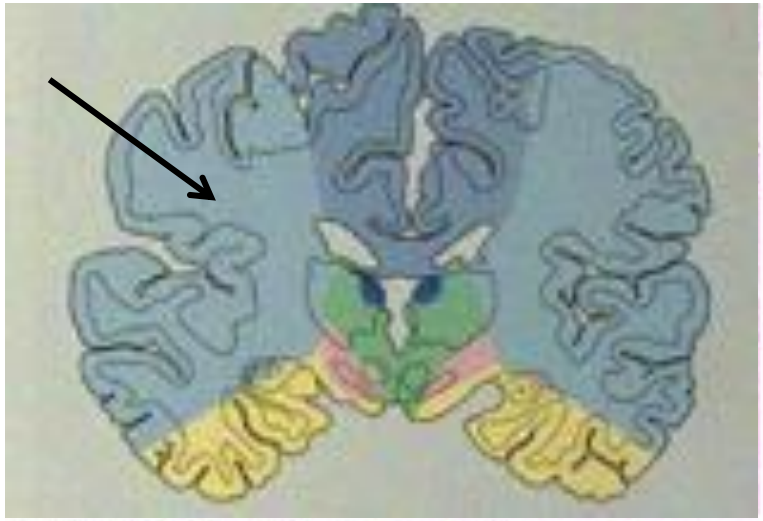
Pale, soft, swollen (6-48 hours)

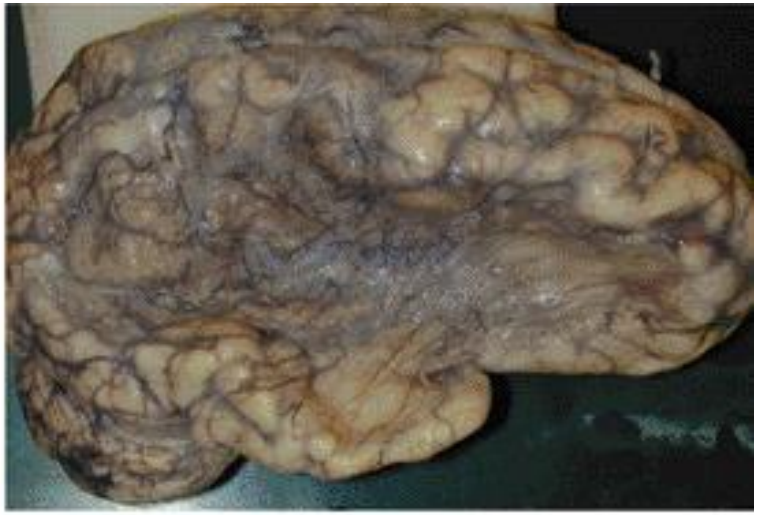
Friable and gelatinous (2-10 days)

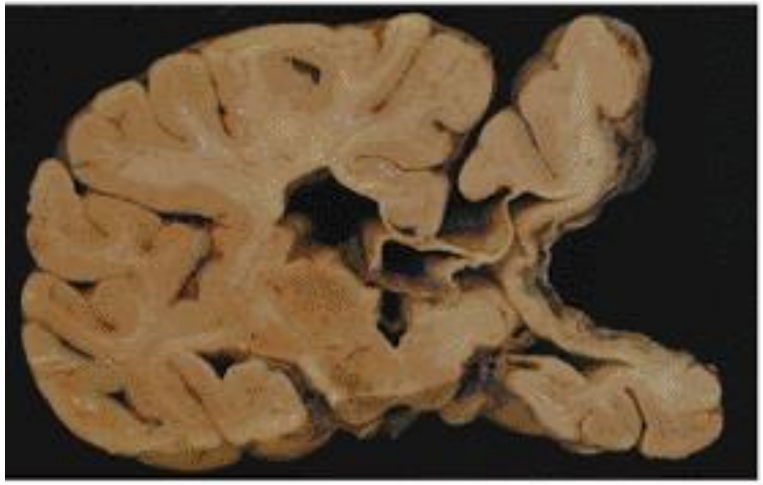
Liquefaction of tissue →
CSF filled cavity (10-21 days)

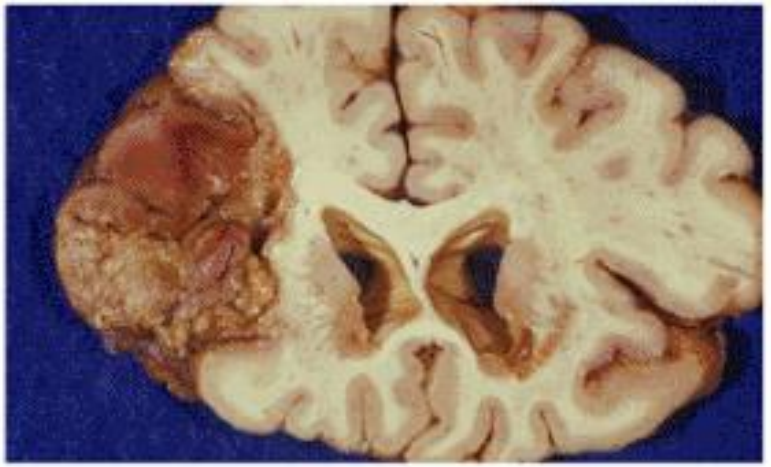


MCA infarct - macroscopy

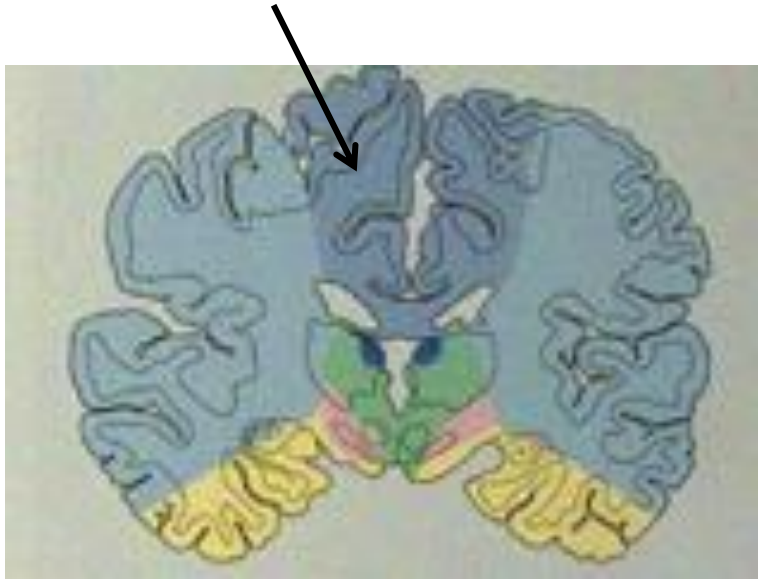


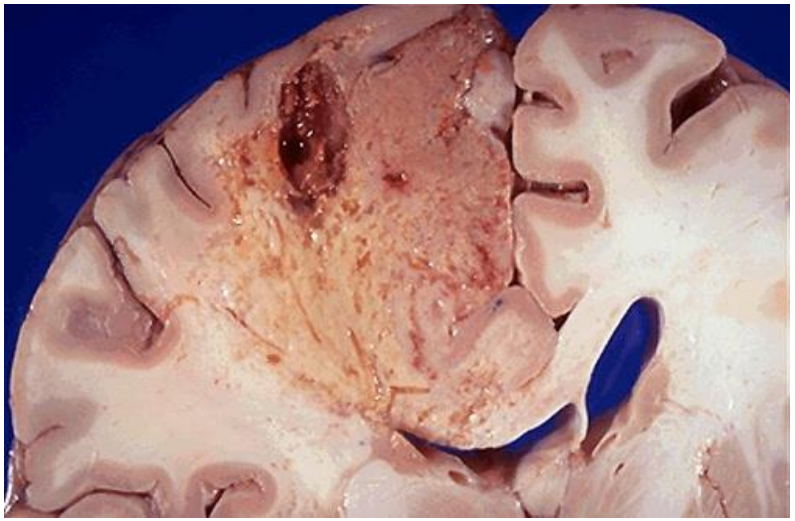


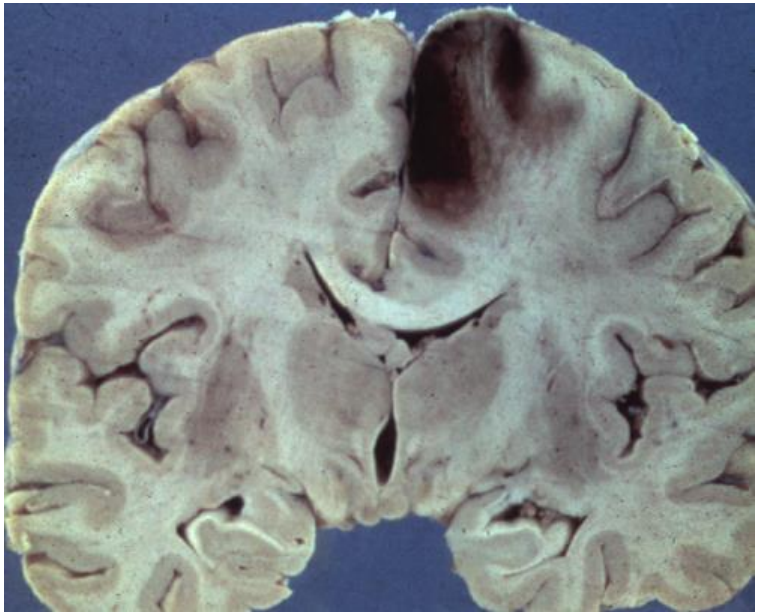


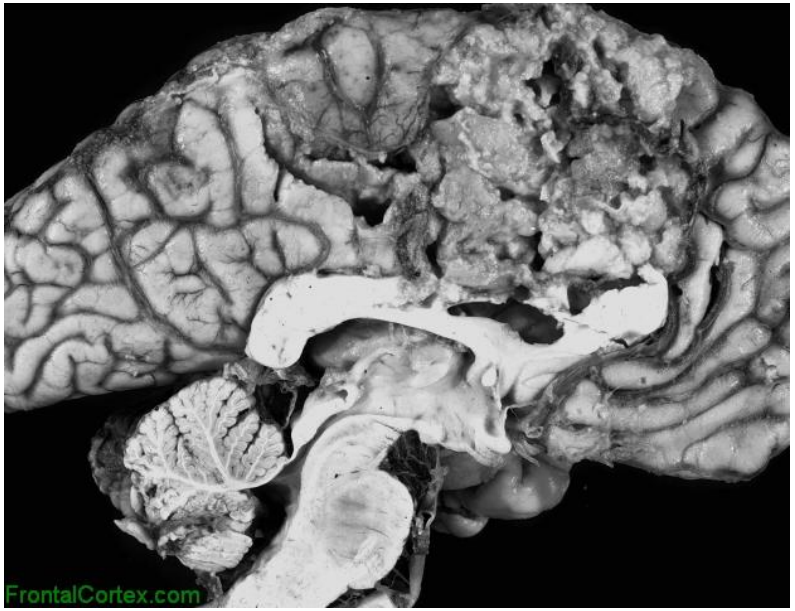


ACA infarct - macroscopy

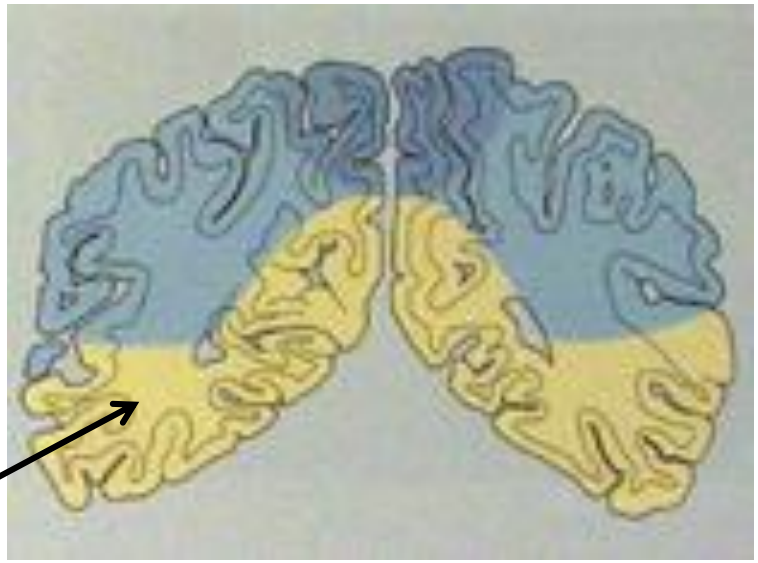


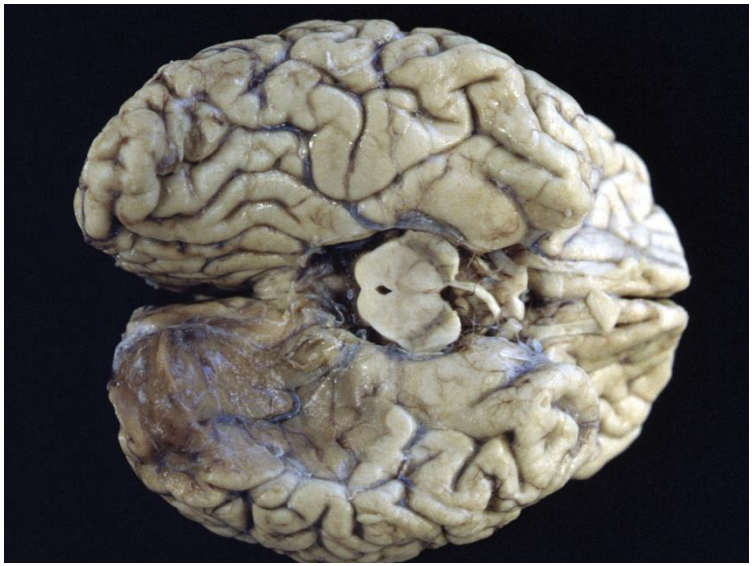


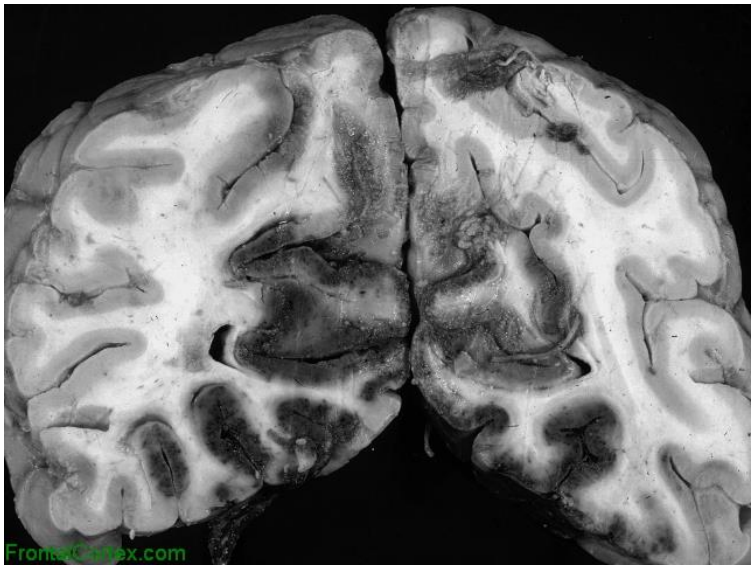


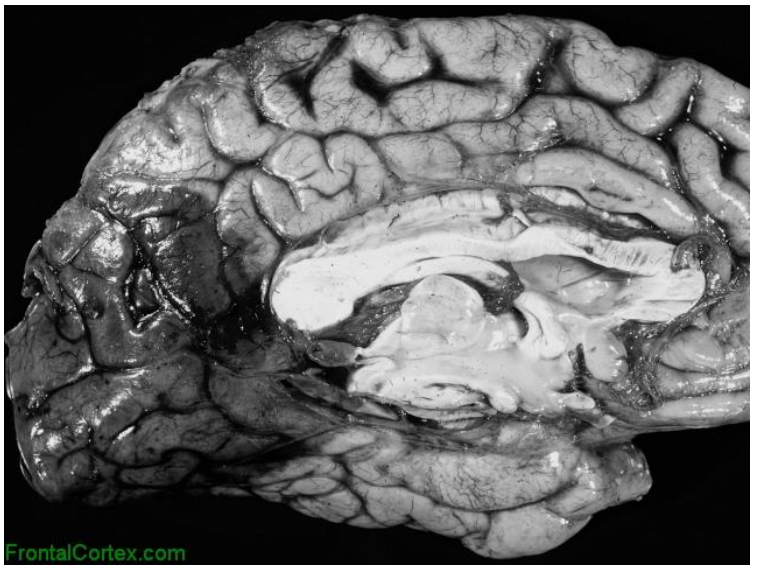


PCA infarct - macroscopy



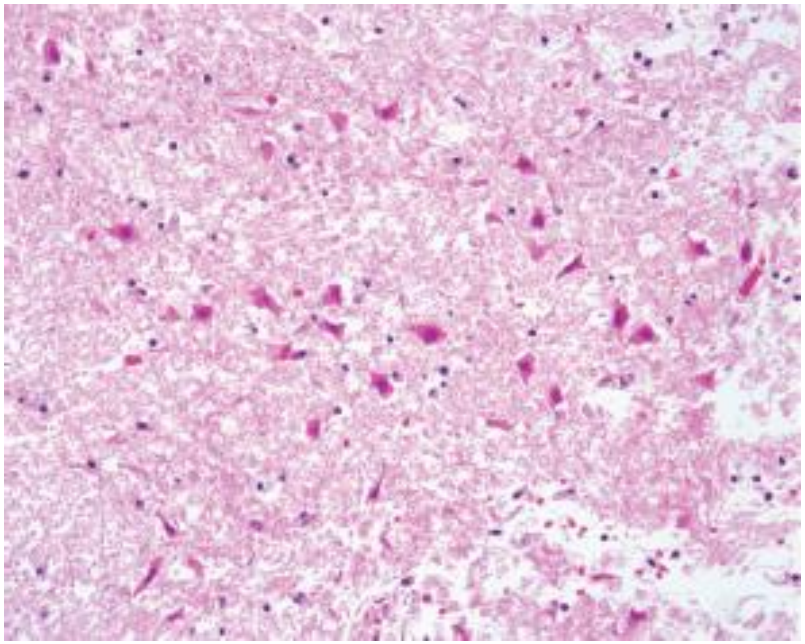


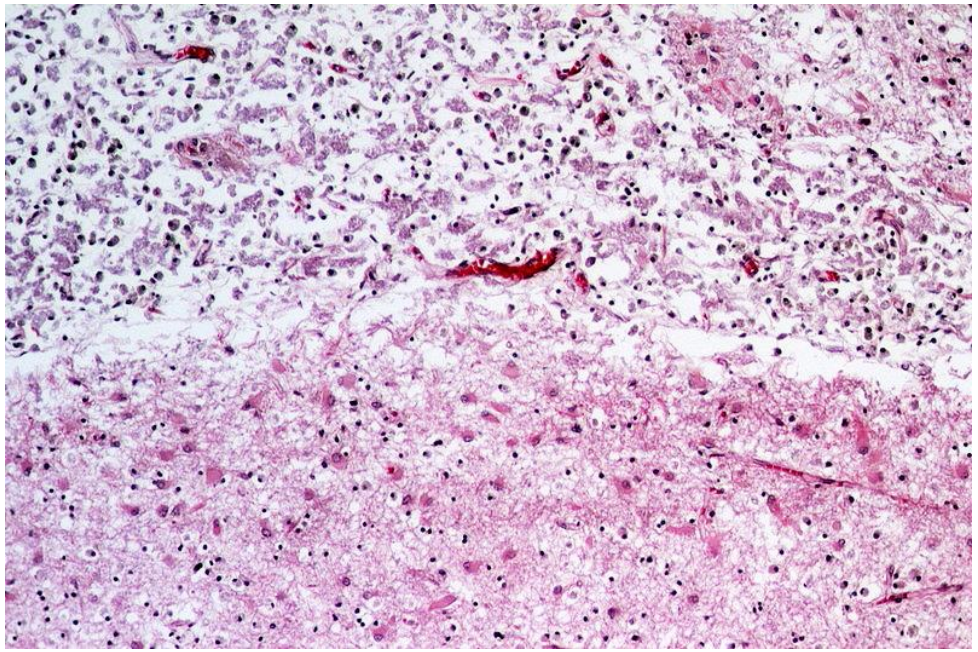




Nonhaemorrhagic infarct - microscopy

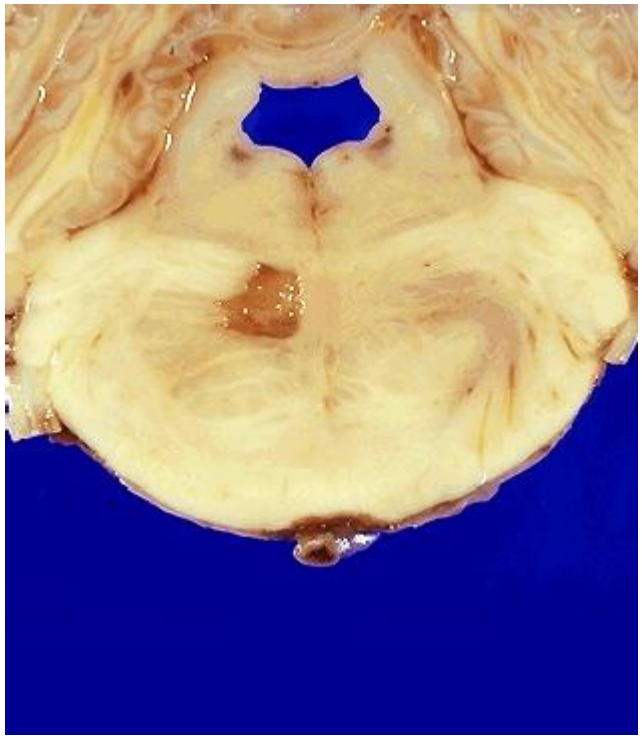
- Red neurons, cytotoxic and vasogenic oedema, swollen glial and endothelial cells, disintegration of myelinated fibers (after 12 hours)
- Neutrophilic infiltration (first several days)
- Macrophage infiltration (over the next 2-3 weeks)
- Increased numbers of enlarged astrocytes at the edge
- Liquefaction of tissue → CSF filled cavity

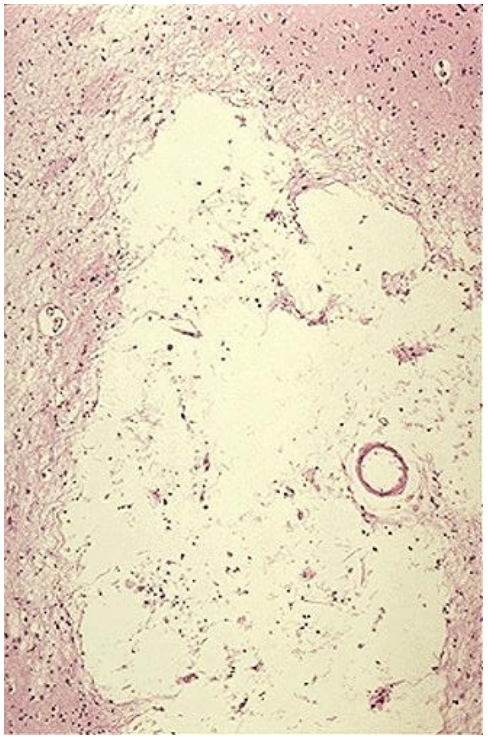


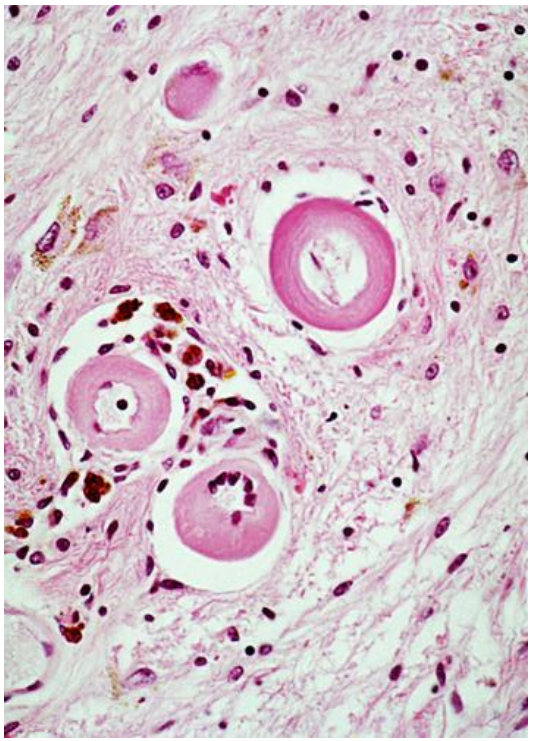


Lacunar infarcts

- Small cavitary infarcts (few millimeters)
- Deep gray matter, internal capsule, deep white matter, pons
- Caused by occlusion of a single penetrating branch of a large artery – due to hyaline arteriolar sclerosis in hypertension and diabetes

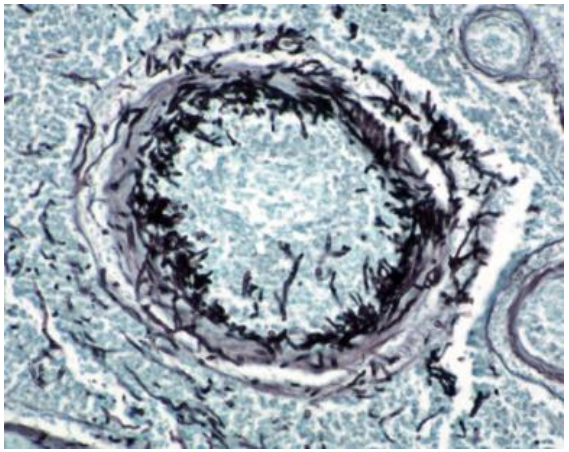






Vasculitis may lead to cerebral infarction

- Infectious arteritis
  - Aspergillosis, Herpes zoster, CMV (immunosuppression)
  - Syphilis, tuberculosis



- Non-infectious vasculitis
  - Polyarteritis nodosa
  - Primary angiitis of the CNS