Cerebrovascular diseases = CVA(cerebrovascular accident)= stroke

• Rapidly developing symptoms and signs of loss of focal CNS function(within seconds or minutes) but they persist for at least 24 hours.

TYPES → Ischemic stroke caused by vascular obstruction by a thrombus(Thrombotic occlusions: due to atherosclerosis of cerebral arteries……Common sites:1. Carotid bifurcation.2.Origin of middle cerebral artery. 3. Ends of basilar artery) or an embolus (More common than thrombotic infarcts…..Source: 1. cardiac mural thrombi. 2. arterial atheroma. 3. venous thrombi "cardiac defects = paradoxical embolism.."…Treatment: anticoagulants)

• Most common site of embolic occlusion : middle cerebral artery, a direct extension of the internal carotid.

→ Hemorrhagic stroke caused by vessel rupture secondary to several vascular diseases, like hypertension or vasculitis OR are due to reperfusion through collaterals or after dissolution of emboli….causes hemorrhagic infarcts.

Signs and symptoms of stroke= FAST

• Face – the face may have dropped on one side, the person may not be able to smile or their mouth or eye may have dropped.

• Arms – the person with suspected stroke may not be able to lift both arms and keep them there because of arm weakness or numbness in one arm.

• Speech – their speech may be slurred or garbled, or the person may not be able to talk at all despite appearing to be awake.

• Time – it is time to dial emergency team immediately if you see any of these signs or symptoms.

•If the symptoms last for less than 24 hours, it is called: transient ischemic attack: a warning sign that a full-blown stroke is imminent/ the supply of blood to the brain is temporarily interrupted, causing a "mini-stroke" often lasting between 30 minutes and several hours.

Morphology/ non-hemorrhagic infarcts/ macroscopic appearance:

• By 48 hours: pale, soft swollen area.

• Day 2-10: gelatinous and friable.

• Day 10 to week 3: liquefaction ending in a fluid filled cavity(Ischemic damage to the brain causes liquefactive necrosis).

Morphology / non-hemorrhagic/ microscopic appearance:

• Early changes(first 24 hours): red neurons(Acute neuronal cell damage) followed by cytoplasmic eosinophilia then pyknosis and karyorrhexis + edema + neutrophils…Similar changes later on glial cells.

• Subacute changes(24 hours to 2 weeks): necrosis, macrophages, vascular proliferation, reactive gliosis, gemistocytic astrocytes.
• Repair(after 2 weeks): gemistocytes regress, cavity persists, Gliosis(Astrocytes are the main cells responsible for repair and scar formation), Loss of organized CNS structure, Removal of necrotic tissue.
• Injury.. Causes: 1. hypertrophy and hyperplasia in astrocytes.
2. enlarged nuclei 3. prominent nucleoli.
4. increased pink cytoplasm. 5. increased, ramifying processes
These changes in astrocytes: gemistocytic astrocyte.

Intracranial pressure

• At rest, it is normally 7–15 mmHg for a supine adult.
• In adults, skull bones cannot expand
What's inside the cranium?
• 80% brain tissue (including intracellular and interstitial fluid which is around 75% of brain weight)
• 10% blood • 10% CSF (cerebrospinal fluid)
Monro- Kellie hypothesis: intracranial volume= $V_{CNS} + V_{CSF} + V_{Blood} + V_{lesion}$
• Space occupying lesions occur with all major brain diseases (except degenerative diseases). Examples: brain tumors, trauma, stroke, haemorrhage.
Causes of increased intracranial pressure:
• Mass effect : brain tumor, hematoma, or abscess.
• Generalized brain swelling :ischemia(causes edema), hypertension
• Increase in venous pressure : heart failure
• Obstruction to CSF flow and/or absorption or increased CSF production: hydrocephalus.
• Idiopathic or unknown
Clinical presentation:
• In the early stages: non specific symptoms/ Cushing reflex (Cushing response or Cushing triad) which manifests by: increased blood pressure, bradycardia and irregular breathing.
• In more advances cases : neurological manifestations including disturbed level of consciousness.
• Later, complications can occur, mainly herniation, seizures and ischemia.

Brain edema= cerebral edema

• Accumulation of excess fluid within the brain parenchyma.
• Two types: vasogenic and cytotoxic edema.. Usually coexist

• The distinction between gyri and sulci is diminished because the sulci are filled with fluid making them narrow and the gyri are widened by the fluid.

Herniation

- A complication of brain edema.
- Focal expansion of the brain tissue .

3 Types

Subfalcine = cingulate:

-Cingulate gyrus displaced under edge of falx.

-causes compression of anterior cerebral artery.

Transtentorial = uncinate:

• Medial aspect of temporal lobe compressed against the free margin of the tentorium.

• The brain tissue is forced from supra-tentorial towards the infra-tentorial compartment.

• Third cranial nerve compressed.. Dilated pupil, impaired ocular movement on the side of the lesion (ipsilateral side).

• Posterior cerebral artery can be affected >>> Ischemic injury to visual cortex.

• Can cause hemorrhage in the midbrain and pons (Duret Haemorrhage: The end result of temporal medial lobe " transtentorial" herniation/ small lineal areas of bleeding in the midbrain and upper pons of the brainstem), usually fatal.

Tonsillar:

• Displaced cerebellar tonsils through foramen magnum

• Brain stem compression... respiratory and cardiac centres in medulla compromised.

• LIFE THREATENING