Intracranial hemorrhage

Causes of non traumatic hemorrhage:

1. Primary brain parenchymal haemorrhage:

- Primary = spontaneous = non-traumatic.
-due to rupture of a small intra-parenchymal vessel.
- Most affected sites: basal ganglia, thalamus, pons and cerebellum.
-The leading cause: Hypertension → Charcot- Bouchard micro aneurysms

Hyaline arteriolosclerosis : Homogeneous pink hyaline thickening of the arteriolar walls with luminal narrowing and loss of underlying structural detail. Occurs due to leakage of plasma components across injured endothelial cells into vessel walls and increased extracellular matrix production by smooth muscle in response to chronic hemodynamic stress.

•Symptoms of parenchymal brain haemorrhage → neurological symptoms
→ symptoms of increased intracranial pressure

• morphology → Extravagated blood.
→ With time.. Resolution and cavity formation

• Old infarct or old hemorrhage; both will end up with a cavity!

• Effects of hypertension on the brain:

1) Massive intracranial haemorrhage.
2) Acute hypertensive encephalopathy= edema.
3) Rupture of small penetrating vessels → Slit haemorraghes
4) Lacunar infarcts: Small infarcts, mostly in deep grey matter (basal ganglia and thalamus), internal capsule, deep white matter and pons. Caused by occlusion of penetrating branches of a large cerebral artery.

2. Vasculitis: Inflammation → rupture of vessel wall → hemorrhage.

Causes of vasculitis:

1)Infectious arteritis:

• previously seen with syphilis and TB.
• Now in association with: CMV, herpes, aspergillosis.....immunosuppression

2)Polyarteritis nodosa.
3)Primary angiitis of CNS cause diffuse encephalopathy with cognitive dysfunction

3. Cerebral amyloid angiopathy:

• Amyloid deposition in the walls of arteries → Bleeding , usually in the lobes of cerebral cortex (lobar hemorrhage)

• Amyloidosis: Deposition of extracellular fibrillary proteins (which are produced by the aggregation of misfolded proteins) ⟶ tissue damage and functional compromise.

-By electron microscope: continuous, non-branching fibrils with a cross-β-pleated sheet conformation.

4. Ruptured berry (Secular)aneurysm:

• Thin walled outpouching of an artery

• 90% in the anterior circulation

• Near major arterial branching points

↑Intracranial pressure ⟶ rupture

• Sudden severe headache followed by loss of consciousness

• 25-50% die • Survivors: risk of recurrent bleeding

-Mainly causes subarachnoid hemorrhage but also can cause hemorrhage within the brain paranchyma.

(Most common cause: ruptured berry aneurysm/ Other causes: vascular malformations, trauma, tumours, haematological disturbances)

5. Vascular malformations:

• Cavernous malformations • Capillary telengectasia • Venous angioma

• Arteriovenous(AV) malformations; Symptoms: seizures and intracranial hemorrhage; Morphology: network of disorganized vascular channels

Other (rarer) causes of intra-cerebral hemorrhage:

• Bleeding disorders • Drug related: anti-coagulants • Cocaine use

• Tumors.. Can encroach on a vessel and cause bleeding.

Head injury

• Blunt or penetrating. • Open or closed.

• Severe brain damage can occur without external signs of head injury.

• Lacerations and even skull fractures are not necessarily associated with brain damage.

Traumatic parenchymal injury

When an object impacts the head:

• Injury of brain at site of impact: coup injury

• Injury opposite to site of impact: countercoup

• Both are contusions

• Repetitive episodes of trauma can later lead to neurodegenerative process e:g Alzheimer.

Brain injury

1.concussions:

• Reversible altered consciousness after head injury in the absence of contusions

• Transient dysfunction in the form of: loss of consciousness, temporary respiratory arrest, loss of reflexes.

• Pathogenesis: unknown

• Recovery is complete but amnesia of the episode.

2.contusion:

• Caused by rapid tissue displacement , disruption of vascular channels with subsequent haemorrhage, tissue injury and edema.

• Common in areas overlying rough and irregular bone surface: orbitofrontal region, temporal lobe tips.

Morphology: • Wedge shaped, widest aspect closest to point of impact.

• Edema and extravasated RBCs.

• Superficial aspects of cortex affected more (contrary to ischemic injury)

3.lacerations:

• Penetrating injuries cause skull fractures and brain lacerations

• Laceration: tissue tearing and hemorrhage.

• Old traumatic injury: depressed, retracted, yellow brown patches involving the gyri.

• Larger lesions: cavity, resembling remote infarcts

4.Diffuse axonal injury:

• Subtle widespread injury to axons within the brain.

• Movement of one region of the brain relative to another.. disrupt axonal integrity.

• Under LM :axonal swelling

• Can lead to severe irreversible neurologic deficit.

Traumatic vascular injury: 1.Intaparenchymal 2.Subarachnoid

	3.Epidural hematoma	4.Subdural hematoma
Cause	Fracture	Rapid movement of brain during trauma
Ruptured vessels	middle meningeal artery	bridging veins
Blood location	Between dura & skull	Between dura & brain tisssue
Shape	Biconvex	crescentic