Lec 1

1. Stroke ≡ CVA ≡ cerebrovascular disease
   - Major cause of Death (Accident)
   - Most common cause of ∴ Acute
     Neurologic Morbidity
   - ↳ clinical term applies to all 3 when symptoms are Acute

* Mechanisms (types) *

→ Ischemic (85%) — Thrombi / Emboli (More common) — Hemorrhagic — vascular rupture

Within sec or min's

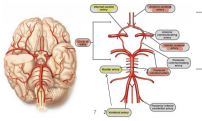
* Stroke : rapidly developing symp & signs of loss of focal CNS function
  (Lasting for 24 hr or Leading to Death)
  Must at least
  - ↳ if symp last <24 hr ∴ its (TIA)
    Transient ischemic attack

* Types of Stroke *

Ischemic

- Caused by vascular obstruction

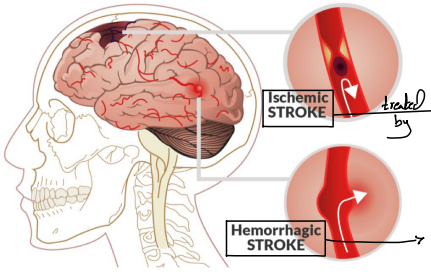
Brain blood supply



- Thrombotic occlusions
  - * Atherosclerosis of cerebral Arteries causing Thrombosis
    - * Common Sites * (remember: - Bifurcations - Narrow areas)
      1. Carotid bifurcation.
      2. origin of Middle cerebral artery.
      3. End of basilar artery.
- Embolic infarcts (Lodge in branches or stenotic ← Arises caused by Atherosclerosis)
  - Sources
    - Cardiac Mural Thrombi Due to
      - Myocardial Dysfunction
      - valvular Disease
      - Arterial fibrillation
    - Arterial Atheroma in
      - Carotid Arteries or
      - Aortic Arch
    - Venous thrombi crossing to Arterial circulation through Cardiac defects (Defect in Heart septum) (Paradoxical embolism) (More common) VSD or ASD
      - DVT
      - Fat emboli

Hemorrhagic

- by vessel rupture 2° to several vascular Diseases
  Ex: Hypertension, vasculitis
  * Causes hemorrhagic infarcts



treated by → Anticoagulants Within 1st 3hrs

if used here it Might Kill the patient xx

Exam Q: Most common site of Embolic occlusion : Middle cerebral A
↳ Direct extension of the internal carotid A.

∴ very important first to distinguish between the 2 Types !!

* Stroke risk factors *

- Same risk factors of Atherosclerosis

* clinical features *

- Signs & symp = "FAST"

- Face May have Dropped on one side : pt unable to smile or their Mouth or eye May have Dropped
- arm weakness or numbness in one arm ∴ unable to lift both arms & keep them there
- Slurred/garbled speech or unable to talk at all Despite appearing to be awake

* Transient ischemic attack (TIA) *

- Sometimes, Stroke is preceded by TIA ∴ Should be treated seriously !!
- ∴ important to be recognized clinically as they are a Warning sign that a full-blown Stroke is imminent !! (in the near future)
- Means that the supply of blood to brain is temporarily interrupted => causing "Mini-Stroke" often last (30 min – several hours)

*Note: Time difference here isn't that important as we can't by 100% accurate about these changes

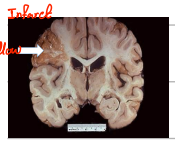
*gliosis: type of scar formation in the Brain & spinal cord.

Morphology / Non-Hemorrhagic infarcts

Macroscopic

Acute (By 48hrs)

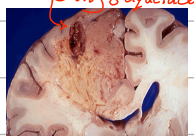
Infarct yellow



- pale, soft swollen area

Subacute (2d - 10d)

Cavity & liquefaction



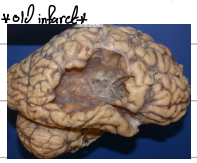
- gelatinous & friable

*ischemic Damage to brain
⇓ Causes
yellow liquefactive Necrosis

Chronic/repair (2w - 3w)

after >2w

*old infarct



- resolved & Ending in a fluid filled Cavity

Microscopic

*Note: No fibrosis in CNS

Early changes (First 24hrs)

@ acute neuronal cell damage (Red Neurons)
@ Cytoplasmic eosinophilia
@ pyknosis & Karyorrhexis
then
@ Neutrophilic infiltrate.

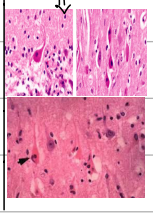
Subacute (24hrs - 2w)

- Necrosis
- Macrophages
- Reactive gliosis
- gemistocytic astrocytes

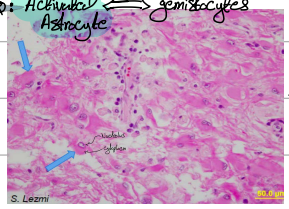
Repair (after 2w)

- removal of necrotic tissue
- gliosis - Astrocytes are the Main cells responsible for repair & scare formation
- Loss of organized CNS structure.

Red neurons + Edema + Neutrophils



Exam Q: Activated Astrocyte ⟺ gemistocytes



- gemistocytes regress
- Cavity persists

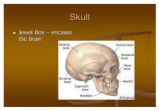
Summary regarding Stroke.

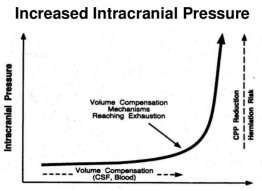
- Stroke = CVA, is a clinical term describing acute neurological symptoms caused by vascular disease.
- Stroke can be ischemic or hemorrhagic. Ischemic is commoner
- Ischemic stroke can be embolic or thrombotic. Embolic is commoner.
- Most common site of embolic occlusion is the middle cerebral artery.
- Ischemic strokes might be preceded by TIA= vascular occlusion causing symptoms lasting from minutes to several hours.
- TIAs predict a full stroke and should be treated promptly.
- Ischemic infarcts in the brain cause liquefactive necrosis.
- In the acute stage we see red neurones and neutrophilic infiltrate
- In subacute stage we see macrophages ,gemistocytes and gliosis.
- In the late stages, gemistocytes disappear leaving a cavity behind.

2 Intracranial pressure

The cranium..

- The brain is enclosed within the skull, which is a rigid box that protects it.
- In adults, skull bones cannot expand
- So if the material within the cranium increases.. Pressure will increase= increased intracranial pressure





if any of them ↑ => intracranial pressure will ↑

10% Blood ← * Cranium Content * (Roughly speaking)
10% CSF ↓ ↳ Brain tissue 80%
*including intracellular & interstitial fluid
Which is around 75% of Brain weight

Monro- Kellie hypothesis

- Monro- Kellie hypothesis: intracranial volume= V_{Brain} + V_{CSF} + V_{Blood} + V_{lesion}
- V= volume.

- This hypothesis indicates that any space occupying lesion in the brain will increase the volume inside the cranium and this will result in increased intracranial pressure.
- Space occupying lesions occur with all major brain diseases (except degenerative diseases). Examples: brain tumours, trauma, stroke, haemorrhage.

OK, so what is intracranial pressure (ICP)???

- It is the pressure inside the skull and is measured in millimetres of mercury (mmHg)
- At rest, it is normally 7-15 mmHg for a supine adult.

- If pressure in the cranium is higher than this upper limit= increased intracranial pressure (= intracranial hypertension.

clinical presentation of increased ICP

Early stages

@ non specific symp (Headache & vomiting) ↓ With no Nausea as the cause isn't from GI tract
@ Might have Cushing reflex
↑BP ← (Cushing triad) → irregular Breathing
↓
Bradycardia

More advance

- Neurological Manifestations ⇓ including (Disturbed level of consciousness)

Later

- complications ⇓ Mainly
*Herniation
&
* Seizures

* Causes of increased ICP*

Mass effect

- Brain tumor
- Hematoma
or
- abscess

generalized Brain swelling

- ischemia
- Hypertension

↑ Venous Pressure

- Heart failure

obstruction to CSF flow & / absorption or ↑ CSF production

- Hydrocephalus

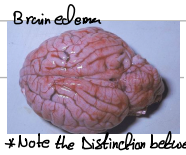
idiopathic or unknown

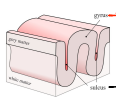
* Brain Edema = cerebral edema

↳ accumulation of excess fluid within the Brain parenchyma.

Types

Vasogenic





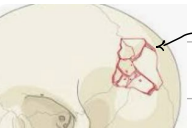
*Note the Distinction between the gyri & sulci is Diminished Due to

Cytotoxic edema

usually Coexist

Herniation : a complication of brain edema

- ↑ Vol of tissue inside the skull → ↑ ICP → Focal expansion of Brain tissue



Cranial vault ⇒ subdivided by rigid Dural folds (Falx & tentorium)

the expanded brain tissue is displaced in relation to these folds

- Expansion= herniation
- SO: herniation is a complication of increased intracranial pressure and it occurs in relation to margins of the dural folds.

Herniation types

Life threatening

Subfalcine (Cingulate)

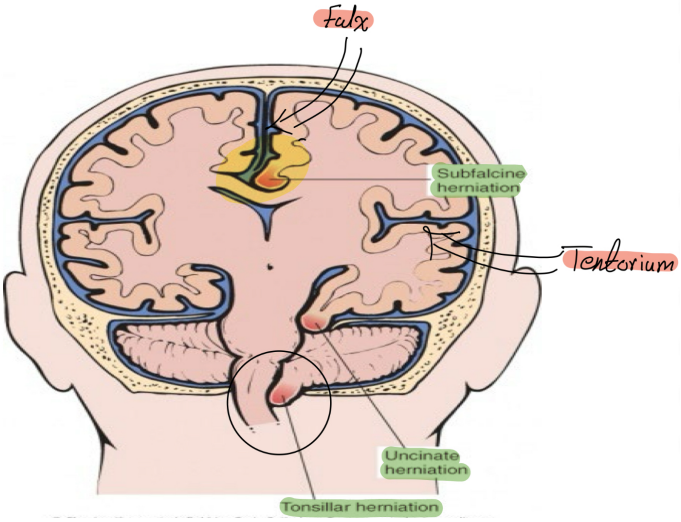
- cingulate gyrus displaced under edge of falx
- Can cause compression of Ant. cerebral A
- ∴ the territory supplied by this Artery can suffer ischemic Damage & infarction if severe

Transtentorial (uncinate)

- Medial aspect of temporal lobe compressed against the free Margin of the tentorium
- ∴ brain tissue forced from Supratentorial → infratentorial compartment

Tonsillar

- Displace cerebellar tonsils through foramen Magnum causes
- ↓
- Brainstem Compression
- ↓
- respiratory & cardiac centers in the Medulla are compromised



Complications

3rd cranial N (oculomotor nerve)

compressed ↓ causing

- Dilated pupil
- impaired ocular Movement on the side of lesion

(ipsilateral side)

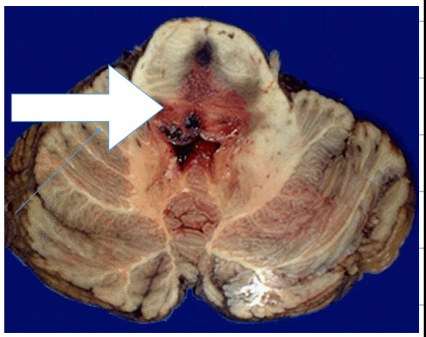
post. cerebral A

Can be affected ↓

- ischemic injury to tissues supplied by it ↓ including visual cortex ∴ blured vision

Hemorrhage in the Midbrain & pons (usually fatal)

(Duret hemorrhage)



Duret hemorrhage

The end result of temporal medial lobe (transtentorial) herniation is compression of the brainstem (midbrain and pons) and stretching of small arterial branches to cause Duret haemorrhages

Duret haemorrhages are small lineal areas of **bleeding** in the midbrain and upper pons of the brainstem. They are caused by downward displacement of the brainstem. They are named after Henri **Duret**.

Summary

- The skull protects the brain but leaves little space to accommodate for any increase of the cranial contents.
- increased cranial contents cause increased intracranial pressure. This can be due to increased brain tissue (tumours), fluid (edema due to hypoxia or inflammation or other causes), CSF (hydrocephalus) or blood (haemorrhage)
- increased ICP manifests as headache and vomiting, Cushing triad (hypertension, bradycardia, irregular slow breathing)) and can progress to coma.
- increased ICP can be complicated by herniation; which is a displacement of brain tissue from a compartment to another.
- Subfalcian herniation displaces the cingulate gyrus under edge of falx, it causes compression of anterior cerebral artery
- Transtentorial herniation displaces the medial aspect of temporal lobe against the free margin of the tentorium. This compresses the third cranial nerve, posterior cerebral artery and can cause Duret haemorrhage in the midbrain which is usually fatal.
- Tonsillar herniation displaces the cerebellar tonsils through foramen magnum causing brain stem compression, which is usually fatal.

Question

- A 54 year old man complained of severe headache and vomiting. imaging studies showed a large subdural hematoma. Two days later he had dilated pupil of the right eye with and his visual acuity decreased. Which of the following is incorrect about his condition?
- A. Can be complicated by haemorrhage in the pons.
- B. His eye symptoms could be related to ischemic injury to the visual cortex
- C.The medial aspect of his temporal lobe is compressed against the free margin of the tentorium
- D. The dilated pupil indicated damage of the left third cranial nerve
- E. He might develop fatal brain stem complications.

Answer

- The scenario describes increased ICP due to hematoma. The complications he had indicate herniation, and the symptoms are those of transtentorial herniation.
- the answer is D: the lesion is related to the ipsilateral nerve (at the same side of the lesion).. so his right third cranial nerve is compressed.
- A is correct, it describes Duret hemorrage . Also E is correct , again it describes Duret haemorrhage.
- C :correct, it simply describes his main complication: transtentorial herniation
- note that the decreased visual acuity is due to effect on the visual cortex (ischemic damage due to compression on the posterior cerebral artery) , however, the dilated pupil and impaired ocular movement are effects of compression on the third cranial nerve.

☑ تم بحمد الله