

# Head CT Criteria and Interpretation

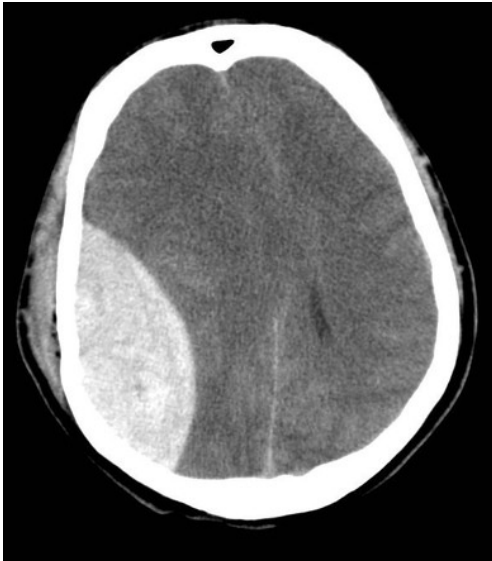
**Key teaching points – cover as much as you can in the allotted time**

1. Criteria for requesting a CT Head
  - “When would you want a CT head for a patient?”
  - CT Head is required if:
    - Any high risk factor
      - Vomiting since the injury
      - Current anticoagulant treatment
      - GCS < 13
      - Sign of basal/open/depressed skull fracture
      - Seizure since the injury
      - Focal neurological deficit
    - Loss of consciousness or amnesia PLUS a medium risk factor
      - Age 65+
      - Dangerous mechanism
      - More than 30mins amnesia of events immediately before head injury
      - Bleeding or clotting disorder
    - GCS < 15 and more than 2 hours since injury
  - In trauma, patients needing one will usually get a CT Head within 1 hour
2. CT Head interpretation
  - Show students the different images below - ask them to describe each image, what is the cause of each?



### **Normal CT Head:**

- Normal grey and white matter attenuation
- Normal sulci and gyri
- Sagittal fissure is in the midline
- Ventricles are of normal size
- Bony calvarium appears normal
- Hyperdensity noted in the lateral ventricles – consistent with normal calcification of the choroid plexus



#### Extradural (epidural) haemorrhage:

- Biconvex sharply-demarcated hyperdense collection in the right temporal region with mass effect
- Poor grey and white matter differentiation consistent with cerebral oedema
- Sulcal effacement consistent with raised ICP
- Sagittal fissure is deviated to the left
- Ventricles are effaced, consistent with raised ICP
- Bony calvarium appears normal
- Subcutaneous fluid collection noted in the right temporal region

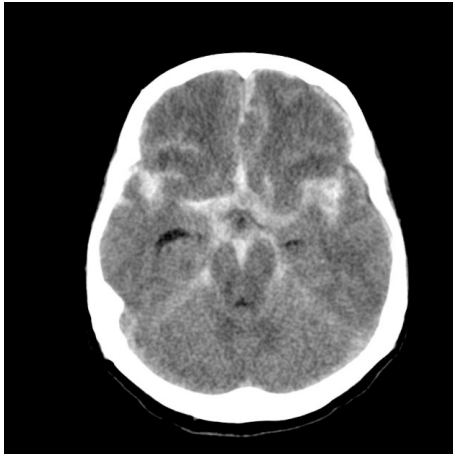
Likely cause: usually due to traumatic injury, commonly rupture of middle meningeal artery into space between inner table of the skull and dura mater. Bi-convex in shape as it does not cross skull suture lines.



#### Subdural haemorrhage:

- Crescent-shaped hyperdense collection in the left frontotemporal region
- Central hypodense areas within hyperdense region – represent active bleeding
- Sulcal effacement consistent with raised ICP
- Sagittal fissure is deviated to the right
- Ventricles on left are effaced, consistent with raised ICP
- Bony calvarium appears normal
- Hyperdensity noted in the lateral ventricles – consistent with normal calcification of the choroid plexus

Likely cause: usually due to traumatic injury causing tearing of the bridging veins. Bleeds between dura and arachnoid so can cross suture lines (therefore crescent shaped). In the elderly, the brain is often smaller (cerebral atrophy) → bridging veins are under more tension → rupture at lower impact.



#### **Subarachnoid haemorrhage:**

- Hyperdense collection around the brainstem, in all basal cisterns and the lateral fissures
- Poor grey and white matter differentiation consistent with cerebral oedema
- Sulcal effacement consistent with raised ICP
- Ventricles are effaced, consistent with raised ICP
- Bony calvarium appears normal

Likely cause: bleeding between the arachnoid and pia mater – follows the contours of the sulci/gyri and circle of Willis – classic star pattern. Most common cause is trauma, but can also be spontaneous (e.g. aneurysm rupture).



#### **Intraparenchymal (intracerebral) haemorrhage:**

- Hyperdense collection in the posterior aspect of the right cerebral hemisphere, consistent with haemorrhage
- Extension of haemorrhage into both ventricles
- Marked midline shift to the left
- Sulcal effacement consistent with raised ICP
- Ventricles are effaced, consistent with raised ICP
- Bony calvarium appears normal
- Small crescent-shaped peripheral hyperdensity in anterior right hemisphere - ? small subdural

Likely cause: bleeding within the brain tissue itself due to a ruptured blood vessel. Lobar haemorrhage.

### 3. Monro-Kellie doctrine

- The cranium, enclosing the brain, forms a fixed space comprising three components: blood, cerebrospinal fluid, and brain tissue.
- Dynamic equilibrium, any increase in one of them results in a compensatory decrease of the other two.
- Once the other compartments have reached their point of maximum compensation, any further increase in the size of one results in increased intracranial pressure.

