SPONTANEOUS SUBARACHNOID AND INTRACEREBRAL HAEMORRHAGE

SUBARACHNOID HAEMORRHAGE:

ALTHOUGH ONLY A SMALL PROPORTION OF E.D. VISITS, A MISSED DIAGNOSIS CAN PRODUCE DEVASTATING RESULTS

75% OF SUBARACHNOID HAEMORRHAGES ARE CAUSED BY RUPTURED ANEURYSM.

IN 20% CASES, A CAUSE IS NOT IDENTIFIED

5% → MISCELLANEOUS CAUSES → A.V.M., DRUGS, PERIMESENCEPHALIC VENOUS BLEED

2% OF FAMILY MEMBERS WITH S.A.H. WILL DEVELOP THE DISEASE, BUT THE RISK RISES WITH ↑'g NUMBER OF FAMILY MEMBERS INVOLVED OR THOSE WITH ADULT POLYCYSTIC KIDNEY DISEASE

ADDITIONAL RISK FACTORS SHOWN BELOW:

Table 160-1 Risk Factors for Subarachnoid Hemorrhage
Hypertension
Smoking
Excessive alcohol consumption
Polycystic kidney disease
Family history of subarachnoid hemorrhage
Coarctation of the aorta
Marfan syndrome
Ehlers-Danlos syndrome type IV

CLINICAL FEATURES:

- Classically → THUNDERCLAP HEADACHE, or a severe headache of acute onset that reaches maximal intensity within minutes → high rate of SAH in this population (11-25%)
- Even if a patient is not experiencing their "worst ever headache", a headache that is different in intensity of quality from past headache raises concern for SAH

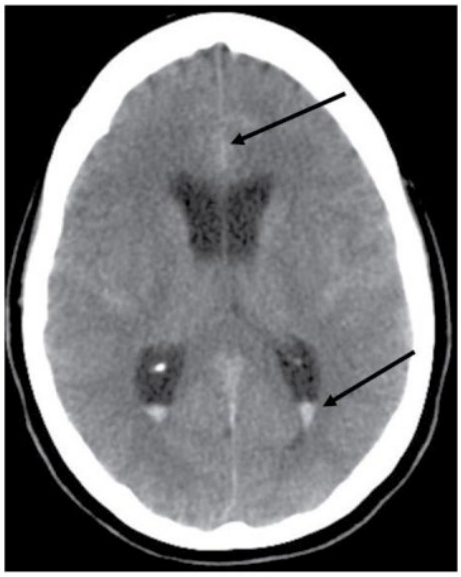
- Headaches associated with LOC, diplopia, neurologic signs, nuchal rigidity → high risk
- Consider in those with headache and N+V or altered mentation
- Approximately 20% develop their headache whilst engaged in activities that raise the BP → sex, exercise, defecation

DIAGNOSIS:

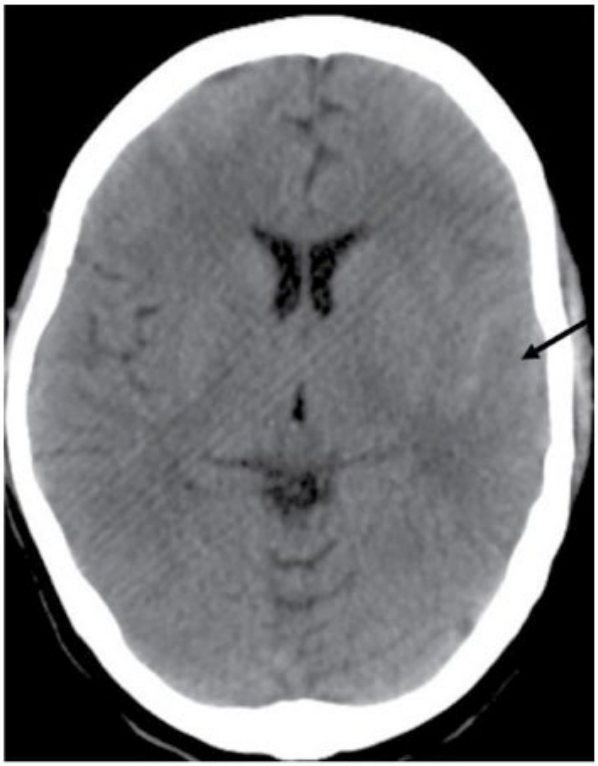
- An aggressive work-up is warranted in patients in whom SAH is considered
  - However, it is misdiagnosed in 5-12% cases on their initial visit to ED
- Differential is extensive:

Table 160-2 Differential Diagnosis of Subarachnoid Hemorrhage
Other intracranial hemorrhage
Drug toxicity
Ischemic stroke
Meningitis
Encephalitis
Intracranial tumor
Intracranial hypotension
Metabolic derangements
Venous thrombosis
Primary headache syndromes (benign thunderclap headache, migraine, cluster headache)

- **Diagnostic modality of choice is NON-CONTRAST CT BRAIN**
  - Sensitivity of modern CT is highest shortly after symptoms (see Stiel *et al*) → ~98% when performed within 12 hours of onset → 93% by 24 hours, 10 days → blood is reabsorbed
  - Prior to Stiel's work, most authorities agree that CSF analysis is needed in those with suspected SAH with a normal CT brain



Diffuse SAH with intraventricular extension and hydrocephalus



Small SAH in left sylvian fissure (subtle)

- CSF analysis:
  - Looking for RBC and XANTHOCHROMIA → bilirubin breakdown products of blood → previously by visual inspection for yellow colour, now done by spectrophotometry → takes about 12 hours for it to develop
  - One study of traumatic LP showed that xanthochromia may develop within 2 hours, hence mandating rapid processing of samples to avoid false positives
  - The number of RBCs to diagnose SAH has never been defined

        - On study showed 10-15% of LP were traumatic, whilst another (small study) showed that there may even be a 25% drop-off in those with confirmed SAH
    - Normal CT, no xanthochromia and zero or few RBCs confirms no SAH → literature remains vague as to the cut-off for number of RBCs

CLASSIFICATION OF S.A.H.:

- **MOST WIDELY USED SYSTEM IS "HUNT AND HESS":**

Table 160-3 Grading Scales for Subarachnoid Hemorrhage

Grade	Hunt-Hess Scale	World Federation of Neurosurgical Societies Scale[22]
1	Mild headache, normal mental status, no cranial nerve or motor findings	GCS of 15, no motor deficits
2	Severe headache, normal mental status, may have cranial nerve deficit	GCS of 13 or 14, no motor deficits
3	Somnolent, confused, may have cranial nerve or mild motor deficit	GCS of 13 or 14, with motor deficits
4	Stupor, moderate to severe motor deficit, may have intermittent reflex posturing	GCS of 7–12, with or without motor deficits
5	Coma, reflex posturing or flaccid	GCS of 3–6, with or without motor deficits

TREATMENT OF S.A.H.:

- **MEDICAL MANAGEMENT AIMS TO PREVENT COMPLICATIONS**
- Regular GCS and pupilliary monitoring
- Complications of SAH include:
    - VASOSPASM (days later)
    - REBLEEDING (a massive problem in ED)
    - Cerebral infarction
    - Cerebral oedema
    - Hydrocephalus
    - Intracranial hypertension
    - Fluid and electrolyte abnormalities
    - Respiratory failure
    - Myocardial dysfunction (autonomic disturbances)
    - Thromboembolism
    - Sepsis
- Risk of re-bleeding is GREATEST IN THE FIRST 24 HOURS → can be reduced by adequate BP control → ideal target BP remains unclear → MAP <130 is broad guideline → use titrateable IV agent (labetalol, nitroprusside)
    - Pain medications and antiemetics remain important and aid reduction of BP
    - AVOID HYPOTENSION
- Vasospasm is most common 2 days to 3 weeks post SAH → modest protective benefits seen with administration of NIMODIPINE → 60mg q4h, initiated within 96 hours unless contraindicated (liver disease, allergy, non-functioning GIT)
- Delayed cerebral ischaemia is associated with extremes of temperature and ↑BSL → avoid these conditions
- Approximately 5-20% of patients with SAH have at least one seizure but SEIZURE PROPHYLAXIS REMAINS CONTROVERSIAL

- ALL PATIENTS SHOULD BE ADMITTED TO I.C.U. IN CONSULTATION WITH A NEUROSURGEON
    - Patients who have normal CT and LP findings within 2 weeks of initial symptoms may be safely discharged from ED

INTRACEREBRAL HAEMORRHAGE:

- Causes 8-11% of all acute strokes and is twice as common as SAH
- ICH carries high morbidity and mortality
- Anticoagulation with warfarin is a significant RF for ICH → annual incidence of 0.3-0.6% in those taking the drug, and plays a role in 6-16% of cases of ICH
    - In those taking warfarin, the risk of ICH nearly doubles for each o.5 increase in INR above 4.5!
    - ICH occurs in ~3-9% of patients administered TPA for ischaemic stroke

PATHOPHYSIOLOGY:

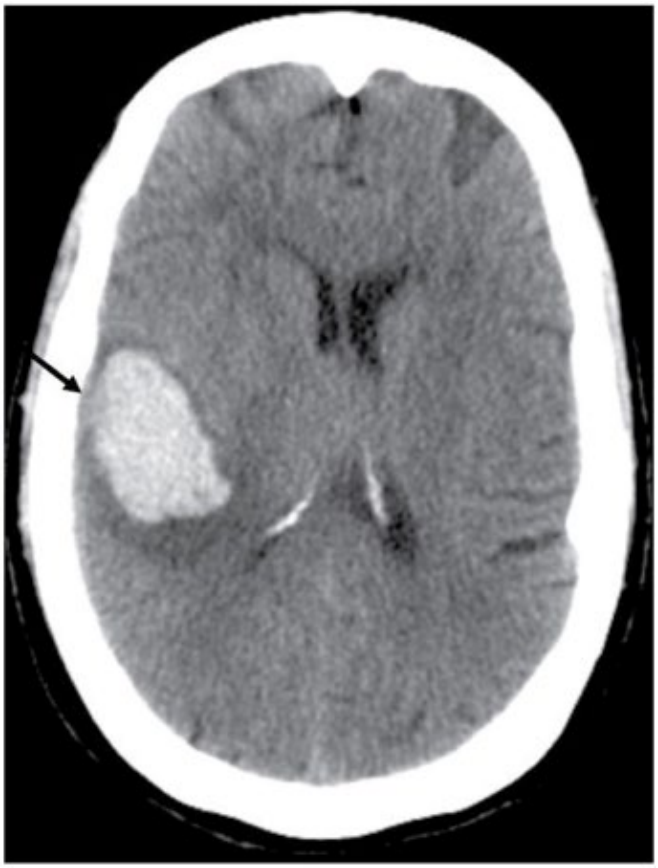
- RF for ICH → long-term HT, AVM, arterial aneurysm, anticoagulant use, sympathomimetic abuse (cocaine), intracranial tumours, smoking

CLINICAL FEATURES:

- ICH may be indistinguishable initially from cerebral infarction, SAH and ischaemic stroke
- Headache, N+V often precede neurologic deficit
- In hypertensive ICH → bleeding is usually localised to the putamen, thalamus, pons or cerebellum (DECREASING ORDER OF FREQUENCY)
- Patients with ICH are more likely to have rapidly progressive symptoms

DIAGNOSIS:

- Differential is similar to SAH (see above)
- CT is optimal for demonstrating haemorrhage extension into the ventricles, whereas MRI is superior for demonstrating underlying structural lesions



Large right parietal intraparenchymal bleed with local mass effect and MLS

TREATMENT:

- In critical care area (resus/ICU)
- Close attention to airway, neurologic status
- AVOID HYPERTHERMIA
- Antiepileptics if seizures occur
- Aggressive management of hyperglycaemia
- Reversal of coagulopathy
- BP management as below but evidence is scarce:

Table 160-4 Suggested Guidelines for Treating Elevated Blood Pressure in Spontaneous Intracranial Hemorrhage

Clinical Circumstances	Management
SBP >200 mm Hg or MAP >150 mm Hg	Consider aggressive reduction of blood pressure with continuous IV infusion.
SBP >180 mm Hg or MAP >130 mm Hg and evidence or suspicion of elevated ICP	Consider monitoring ICP and reducing blood pressure using intermittent or continuous IV medications to keep cerebral perfusion pressure >60–80 mm Hg.
SBP >180 mm Hg or MAP >130 mm Hg and no evidence or suspicion of elevated ICP	Consider a modest reduction of blood pressure (e.g., MAP of 110 mm Hg or target blood pressure of 160/90 mm Hg) using intermittent or continuous IV medications.

- MANAGEMENT OF RAISED ICP:
    - Raising head of bed 30 degrees
    - Appropriate analgesia and sedation

- OSMOTIC DIURETICS (mannitol 0.5g per kg)
- INTUBATION AND MILD HYPERVENTILATION (aim normocarbia 30-35 at lowest)
- INVASIVE ICP MONITORING USUALLY NEEDED