Subarachnoid Haemorrhage

Non-traumatic SAH is usually the result of bleeding from a mostly acquired (berry) aneurysm at artery junctions in the Circle of Willis.

Epidemiology

- 5-10% of cases of all strokes, 6% of first strokes.
- 75% from intracranial arterial aneurysms (70% berry), 20% from a non-aneurysmal peri-mesencephalic haemorrhage and 5% from other vascular abnormalities including AVMs.
- 50% <55y and of those ~50% die acutely.
- 10 to 15% fail to reach hospital.
- 1% of headaches presenting to ED

Risk factors

- Previous SAH or 1st or 2nd degree relative with SAH
- Hypertension
- F>M esp if oestrogen deficiency.
- Connective tissue diseases e.g. Marfan's, Ehler-Danlos IV, NF type 1, AD adult polycystic kidney disease
- Excessive alcohol intake, smoking, cocaine
- Anticoagulants ↑severity but little evidence to say risk of SAH
- ↑Aneurysm size, most that bleed are <1cm in diameter.

Presentation

Warning bleed:

- May occur in >50% - headache, vomiting, spont resolution, CN lesion

Acute presentation:

- Severe (or worst ever), and often sudden, headache. (Similar to thunderclap headache)
- Vomiting, seizures (~20%), sentinel bleeds (by 1-3wks: ~40% headache or TIA-like)
- ↓LOC (66%).
- Meningeal neck stiffness (3-6hrs)
- Intraocular haemorrhages on ophthalmoscopy (15%)
- May be focal neurological signs, suggestive of a stroke ± CN palsy
- Reflex rise in BP (Cushing response)

Differential diagnosis

- Other causes of stroke
- Meningitis
- Trauma
- Thunderclap headache

Investigations

Bloods: ABG, UEC (Na), Coags. A mild trop rise may be found.
ECG: ST & T wave changes (usually inferior leads: ST↓, T↓ or peaked), also ↑QRS, ↑QTc. If these are wrongly interpreted as AMI & thrombolysis given, the result may be disastrous.
Imaging: Plain CT (~97.5-100% sensitive if <12h. ↓Accuracy with time.) If +ve CT angio/MRA.
LP: If CT negative and no CI. ↑Opening pressure, ↑RBCs but beware bloody tap. Xanthochromia detectable after 4hr, always if >12hr (95% sens by spectrophotometry for bilirubin).
Angiography: Consider if CT negative & LP negative yet still high suspicion or high risk factors.

Grading

Number of systems. World Fed of Neurological Surgeons use GCS+motor deficit for 5 grades.

World Federation of Neurosurgical Societies (WFNS) SAH grade

WFNS Grade	Glasgow Coma Score	Major Focal Deficit
0 (unruptured)	—	—
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present/absent
5	3–6	Present/absent

Management

Aims: prevent further bleeding and ↓the rate of 2° Cx e.g. cerebral ischaemia or hydrocephalus.

Resuscitation:

- ABCD, O_2, intubate if coma
- IVC, analgesia (not aspirin), antiemetic
- Maintain CPP & ↓ICP (head up 30°, hypervent, **mannitol/3% saline**, ?**lignocaine** pre-ETT),
- Careful BP control (?ideal target - patient's norm, MAP<130mmHg, sysBP<160mmHg).
- Anticonvulsants for seizures

Cease/reverse all anticoagulants/anti-platelet drugs.

Calcium channel blocker: anti-vasospasmodic (evidence for use based on single trial)

- **Nimodipine** 60mg PO/NG q4h x 7d (IV not proven and sig SE but sometimes used)

Neurosurgery: aneurysm >7mm or wide neck, otherwise coiling

Endovascular embolisation/coiling: indicated when aneurysm <7mm or narrow neck

Statins: may have some benefit.

Treat Cx:

- Acute: Electrolyte imbalance, seizures and hypotension.
- Delayed: Rebleeding (50% mort), cerebral ischaemia/vasospasm (30%), hydrocephalus

Prognosis

10% die pre-hospital, 30% die if untreated, ~35% make good recovery, ~35% devastating.

Best guides:

- Level of consciousness and neurologic grade on admission
- Patient age (inverse correlation)
- Amount of blood on initial head computed tomography (CT) scan (inverse correlation)
- Also cerebral infarction, fever, and symptomatic vasospasm , anaemia and/or red blood cell transfusions