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CLINICAL UPDATE

Non-Traumatic Subarachnoid Haemorrhage

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Abstract :

This update is a basic review of epidemiology, clinical presentation, diagnosis and management of subarachnoid haemorrhage and its complications. A neurological emergency with high morbidity and mortality, we present the common and potentially life threatening neurologic and medical complications of SAH to increase the awareness and promote early recognition, management and intervention which will reduce early mortality and prevent secondary brain damage and morbidity. Management of these patients adhering to published guidelines is emphasized and references to clinical trials is to elucidate the role of controversial management approaches. We have avoided great in depth into genetics and rare complications and presentations as this review is targeted to help undergraduates and junior doctors.

Intoduction

Subarachnoid haemorrhages (SAH) account for 3% of all strokes (1). They usually result due to rupture of saccular (berry) aneurysms. Mortality due to SAH has reduced in the recent past due to rapid recognition and improved management. However, morbidity due to SAH is high, with survivors being left with permanent disability, cognitive deficits and psychological effects such as depression and anxiety.

Average age of aneurysm rupture is in the 5th or 6th decade of life. Ruptured cerebral aneurysms are the commonest cause (85%) with other vascular malformations such as arteriovenous malformations, arteriovenous fistulas and reversible cerebral vasoconstriction syndrome contributing to about 5% of SAH (2). Ten percent of SAHs may not reveal a source of bleeding.

Risk factors:

Modifiable risk factors
• hypertension • smoking • heavy alcohol use • sympathomimetic drugs such as cocaine

Table 1: Modifiable Risk Factors for SAH

Non-modifiable risk factors
• increasing age • female gender • African American, Hispanic, Japanese and Finnish ethnicity • prior history or family history of subarachnoid haemorrhage • history of aneurysm in two or more first degree relatives • autosomal polycystic kidney disease • type IV Ehlers-Danlos syndrome • cerebral aneurysms more than 7 mm in diameter.

Clinical presentation:

The most common clinical symptoms on presentations include

- Sudden severe headache
  (described as the worst headache ever experienced)
- Loss of consciousness
- Nausea
- Vomiting
- Photophobia
- Neck pain

Some patients may experience a headache without any other associated symptoms and is known as the sentinel headache.

If the diagnosis is not suspected there is a high risk of a life threatening re-bleeding within hours to days. Other rare symptoms at presentation that should also raise the suspicion of SAH include seizures, acute encephalopathy and concomitant subdural haematoma.

Physical examination should include

- Level of consciousness (Glasgow coma scale)
- Evaluation for meningism
- Presence of focal neurological deficits
- Fundus examination may reveal intraocular haemorrhage (Terson syndrome – intraocular haemorrhage associated with SAH is associated with increased mortality and may occur in up to 40% of patients with SAH (3)).

Diagnosis:

Several diagnostic modalities are used to confirm the diagnosis of SAH.

01. Head Computed Tomography: A non-contrast CT (NCCT) head is the most rapidly available initial diagnostic test. Sensitivity of the NCCT changes with time of headache onset from 93% in the first 6 hours to approximately 100% in the first 12 hours and then begins to drop to 93% in the first day to less than 60% at 7 days (4). The characteristic appearance of hyperdense blood in the basal cisterns or sylvian, interhemispheric and interpeduncular fissure is suggestive of aneurysmal aetiology. NCCT Brain also helps evaluate for presence of complications such as hydrocephalus, intraventricular haemorrhage and intracerebral haemorrhage.
02. Lumbar puncture: Lumbar puncture is recommended as the immediate next recommended investigation in the presence of a negative or equivocal NCCT with clinical findings highly suggestive of a SAH. Opening pressure should be measured and is usually high. CSF is spun and visually inspected for xanthochromia. Spectrometry for detection of xanthochromia is superior to visual inspection however not always available. Xanthochromia develops 12 hours after onset of SAH thus it is recommended that lumbar puncture is performed after 12 hours.
03. Magnetic resonance imaging: NCCT head and MRI are equally sensitive in detecting SAH in the first 2 days, with MRI brain being slightly superior in the first 6 hours. Due to the availability and high sensitivity NCCT brain is the diagnostic investigation of choice. Hemosiderin sensitive MRI sequences (gradient recalled echo(GRE) and susceptibility-weighted imaging(SWI)) or fluid attenuated inversion recovery (FLAIR) sequences are superior in detecting subacute and chronic SAH when compared with NCCT(5). MRI is also helpful in detecting alternative pathologies such as arteriovenous malformations, inflammatory, infective and neoplastic aetiologies.
04. Identifying the bleeding source: Vessel imaging should follow a positive NCCT, MRI or lumbar puncture. The gold standard for vessel imaging is cerebral digital subtraction angiography(DSA). CT angiography (CTA) is widely available and is preferred as the first line vascular imaging modality as it is less invasive compared to DSA.
05. Perimesencephalic subarachnoid haemorrhage: Thirty eight percent of patients with negative imaging will have a nontraumatic SAH with blood isolated to the perimesencephalic cisterns.

Management:

SAH is a neurological emergency. Management of the acute phase of SAH is divided into 2 phases –

01. Prompt evaluation, recognition and diagnosis and immediate transfer to an appropriate centre for rapid treatment of the bleeding source
02. Close monitoring in a centre with neurological services and expertise to prevent secondary neurologic and medical complications.

Initial management:

01. As in any critical care situation the initial management focuses on airway breathing and circulation. Patients unable to protect their airway (coma, stupor from hydrocephalus, seizures, agitated patients who are sedated) should be intubated.
02. Re-bleeding:
    - The main focus of management during the first few hours until definitive treatment of the bleeding source.
    - Re-bleeding is a life threatening complication, with the highest mortality rate within the first 72 hours after SAH. Majority of the bleeds occur within the first 6 hours (2).
    - Re-bleeding rates are about 3% per year after the first month.
    - Risk factors for re-bleeding include poor grade SAH, hypertension, large aneurysm and the use of antiplatelets or anticoagulants.
    - Blood pressure fluctuations and blood pressure peaks should be avoided.
    - Current guidelines recommend maintaining systolic blood pressure below 160mmHg (6,7) with continuous infusion of intravenous antihypertensives (nicardipine 5mg/h to 15mg /h or labetalol 5 mg/h to 20mg/h)
    - Continuous intravenous infusion is preferred over bolus administration.
    - Hydralazine is best avoided due to rebound hypertension.
    - Pain management with short acting opiates.
    - Meningeal irritation is managed with dexamethasone.
03. Antifibrinolytics: Short term use of antifibrinolytics eg. Tranexamic acid, up to a maximum of 72 hours until aneurysm is secured is recommended. (7,8,9). Prolonged infusion results in dep vein thrombosis, venous thromboembolism, stroke and myocardial infarctions and is best avoided.
04. Disease severity scoring: The most common scales used are the World Federation of Neurological Surgeons Scale (WFNSS)

and the Hunt and Hess Scale. They are strong predictors of outcome (10,11). The modified Fishers Scale is the most reliable and validated radiological scale which is linearly associated with worse cerebral vasospasm and delayed cerebral ischaemia.

05. Admission to high volume centre: A high volume centre is defined as centres which handle more than 35 SAH cases per year with experienced cerebrovascular surgeons, endovascular specialist and neurocritical care services. (12)

06. Aneurysm treatment: Currently used definitive treatment modalities are clipping and coiling. The ISAT (International Subarachnoid Aneurysm Trial) which compared endovascular coiling versus surgical clipping showed a trend preferring endovascular coiling (13). The endovascular coiling group had a significantly higher survival, less disability and a lower risk of epilepsy compared to surgical clipping. With better outcomes in most parameters being better even at 10 years endovascular coiling is preferred over clipping. However the final choice of treatment is affected by patient characteristics as well as aneurysm characteristics.

Endovascular coiling is preferred in

- Older age
- Poor clinical grade
- Multiple comorbidities
- Top of basilar aneurysm
- High surgical risk
- Aneurysm suitable for coiling or clipping

Surgical clipping is preferred in

- Aneurysms with wide neck to body ratio
- Crucial arteries arising from the aneurysm dome
- Middle cerebral artery aneurysm
- Aneurysm with large parenchymal haematoma

07. Critical Care Management

- SAH is a systemic disease and is associated with systemic inflammatory response in syndrome (SIRS) in 75% of cases.
- SAH is also associated with several neurological complications such as hydrocephalus, brain oedema, delayed cerebral ischaemia, re-bleeding, seizures and neuroendocrine disorders and cardiac and pulmonary complications.

Neurological Complication	Management
Re-bleeding: • Life threatening complication\	• Early and rapid treatment of unsecured, ruptured aneurysm • Aggressive blood pressure control
Hydrocephalus: • Acute symptomatic hydrocephalus occurs in 20% of cases • Occurs within minutes to days after SAH onset • Presents as decreased level of consciousness, impaired up gaze, hypertension, delirium • Resolves spontaneously in 30% but can also rapidly deteriorate	• NCCT brain to confirm diagnosis • Insertion of external ventricular drain (EDV). • Lumbar drain can be used for communicating hydrocephalus • Risk of EDV include infection and bleeding in addition to changes in transmural pressures precipitating bleeding in an unsecured aneurysm. • Rapid weaning of EDV is recommended after securing the aneurysm or within 48 hours in neurologically stable patients. • If weaning is unsuccessful a chronic ventriculoperitoneal shunt is recommended.
Seizure and seizure prophylaxis: • Seizures occurring prior to securing aneurysm is a sign of early re-bleeding • Long term epilepsy occurs in 2% of patients • Non-convulsive seizures and non-convulsive status is common (3-18%). • Non-convulsive status is associated with delayed cerebral ischaemia and worse prognosis	• Treatment with antiepileptics is limited to the period before and around aneurysm clipping or coiling. • Antiepileptics have a negative effects on recovery • Phenytoin has a negative effect on neurocognitive recovery after SAH and is best avoided. • Stop antiepileptics in patients where clinical examination can be done reliably as soon as aneurysm is secured • Prophylaxis recommended only for 3 to 7 days unless patient presented with seizures at the onset of SAH. • Levetiracetam is used frequently because of its high bioavailability, favourable side effect profile and minimal drug interaction.

Delayed cerebral ischaemia: • Defined as any neurological deterioration that persists for more than 1 hour and cannot be explained by any other neurologic or systemic conditions, such as fever, seizures, hydrocephalus, sepsis, hypoxemia, sedation and other metabolic causes. • A diagnosis of exclusion • Occurs 3 to 14 days after SAH • Risk factors *SAH thickness *intraventricular haemorrhage *high score on the modified Fisher Scale *poor clinical grade *loss of consciousness at ictus *cigarette smoking *cocaine use *systemic inflammatory response *hyperglycaemia *hydrocephalus *non-convulsive seizures	• Calcium channel blockers (nimodipine) and maintenance of normal intravascular volume status are prophylactic interventions used. • Nimodipine 60mg every 4 hours for 21 days • Hypotension (which may lead to hypoperfusion) is a common side effect requiring a dose reduction with an increase in frequency to 30 mg every 2 hours • Hypervolemia must be avoided • In the presence of cerebral salt wasting fludrocortisone may need to be used every 12 hours.
Medical Complications	Management
Cardiopulmonary complications: • Range from minor ECG changes to severe stress cardiomyopathy and neurogenic pulmonary oedema. • Troponin can be elevated • Pulmonary oedema can lead to hypoxia	• Appropriate medical management as per recommended guidance
Fever: • Most common complication (in up to 70% of patients) • More likely to occur in patients with high grade SAH and poor neurological status	• Monitor temperature • Antipyretics • Rule out or treat infectious aetiology • Shivering should be strictly avoided
Thromboembolism: • Deep vein thrombosis occurs in 2 to 20% of screening method • Patients with high grade SAH are at greatest risk	• Pneumatic compression devices • Chemoprophylaxis is initiated immediately after endovascular aneurysm repair and within 24 hours after craniotomy and clipping.
Glycaemic dysfunction: • Common due to stress • Associated with delayed cerebral ischaemia and poor clinical outcome	• Maintain a blood glucose level between 80mg/dl and 200 mg/dl (6,7)
Hyponatremia: • Most common electrolyte disorder in SAH • Most common is cerebral salt wasting • Syndrome of inappropriate secretion of antidiuretic hormone is rare	• Cerebral salt wasting is managed with fluid administration and sometimes with continuous infusion of hypertonic saline (1.5% to 3%) and fludrocortisone if diuresis and natriuresis impede maintenance of adequate volume status • SIADH is managed with fluid restriction and sometimes diuresis with loop diuretics.
Anaemia: Haemoglobin less than 9g/dl is associated with delayed cerebral ischaemia and poor clinical outcomes	• Goal haemoglobin at least 10 g/dl to 11.5g/dl

Table 3: Neurological and Medical Complications

Prognostication after SAH

World Federation of Neurological Surgeons Scale and the Hess and Hunt Scale help discriminate high risk from low risk patients in the population but they cannot be applied for individual patients. Factors such as patient values and preferences, comorbidities, social networks and resilience affect outcome. All efforts should be made to save patients even with high grade SAH unless they present with bilaterally dilated pupils and a head CT or DSA is inconsistent with brain perfusion and life. Adequate protocolized neurocritical care should be provided for the first 2 weeks before advanced directives and relatives wishes are taken into consideration.

The recently published Functional Recovery Expected After Subarachnoid Haemorrhage (FRESH) scale prognosticates expected 12 month cognitive outcome and quality of life after SAH (14).

Conclusion:

SAH is a life threatening, neurological emergency carrying a high morbidity and mortality. Early recognition and prompt protocolized management along published guidelines with early detection and management of life threatening complications help reduce mortality.

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