Abstract

Introduction
Aneurysm subarachnoid haemorrhage is a leading cause of mortality occurring in about 10 per 100,000 persons annually. It is characterised by three commonly occurring complaints: ‘worst headache of my life’, photophobia and meningismus. It is imperative that emergency department and primary care physicians understand how to diagnose and initially manage aneurysm subarachnoid haemorrhage.

Discussion
This review details cerebrospinal fluid analysis and the current imaging techniques for identifying aneurysm subarachnoid haemorrhage and intracranial aneurysms. It will cover the initial management and treatment options along with considerations in delayed SAH presentation.

Conclusion
A high suspicion for SAH during the initial evaluation increases the patient’s chance for early intervention and reduces mortality and disability.

Introduction
Aneurysm subarachnoid haemorrhage (SAH) is a leading cause of mortality (40%–50% of SAH), occurring in about 10 per 100,000 persons annually; 30,000 SAH in the United States alone each year. SAH is characterised by three commonly occurring complaints: ‘worst headache of my life’, photophobia and meningismus. These symptoms often can be confused with bacterial or viral meningitis and migraine headaches. As a result, aneurysm SAH is initially misdiagnosed in nearly 12% of patients, a potentially lethal mistake. Misdiagnosis increases the risk of re-bleeding and subsequent death. The rule of thirds dictates that one-third of the patients with SAH die before reaching the hospital, another third present with irreversible neurological deficits and the last third make it to treatment; half of the last third will have long-term disabilities (Figure 1). In short, only about 16% of patients will be left with minimal or no permanent neurological sequelae after SAH.

A high suspicion for SAH during the initial evaluation in the Emergency Department or by the Primary Care Physician increases the patient’s chance for early intervention and reduces mortality and disability. Standardised protocols for the evaluation of patients with ‘headache’ in the Emergency Department currently does not exist, further complicating the initial evaluation and disposition of these patients; such a protocol may save lives. Here, we review the diagnosis and initial management of suspected aneurysm in patients with SAH.

Discussion

Aetiology
Trauma is the most common cause of SAH, followed by ruptured intracranial aneurysm (75%–80%) and arteriovenous malformations (4%–5%). Other causes to consider include: central nervous system vasculitis, cerebral artery dissection, coagulopathy, dural venous sinus thrombosis, spinal cord arteriovenous malformation or dural fistula, sickle cell disease and sympathomimetic drugs such as cocaine. In 14%–22% of SAH, no cause can be identified on angiography even with multiple modes of imaging. In this case, the aetiology may be from a venous bleed (perimesencephalic haemorrhage) or small aneurysm that auto-thrombosed and no longer fills with blood; this is referred to as angiographic negative SAH. Patients with SAH who are suspected of having a ruptured aneurysm should be evaluated for risk factors associated with brain aneurysm formation, a spinal tap to look for blood in the cerebrospinal fluid and radiographic imaging.

Risk Factors
Many risk factors for brain aneurysm formation and haemorrhage are modifiable with medication or behavioural changes; cigarette smoking and hypertension being the most important (Table 1). Identification of risk factors in men and women aged 18–49 years concluded that SAH in this age group is mostly associated with cigarette smoking, illicit drug use and hypertension. A 70%–75% of patients with SAH had a prior history of smoking and 50%–60% are current smokers. Other independent risk factors in this study included a low Body Mass Index, family history of haemorrhagic stroke, and low educational achievement; the increased risk of SAH persists even after cessation of cigarette smoking. The odds ratio (OR) of SAH in previous smokers (OR, 4.1; 95% CI: 2.7–6.0) and current smokers (OR, 5.4; 95% CI: 3.7–7.8) suggests that the haemodynamic, but not structural changes induced by smoking, may resolve after smoking cessation.

Genome linkage studies observing 104 siblings affected with intracranial
aneurysms mapped the occurrence of brain aneurysms to chromosome 7q11. The best evidence for linkage was detected at D7S2472 in the vicinity of the elastin gene (ELN). The haplotype between the intron-20 and intron-23 polymorphisms of ELN was strongly associated with brain aneurysms ($P < 0.000$) and homozygous patients were at high risk ($OR = 4.39$, $P = 0.002$) suggesting a genetic placement near the ELN locus on chromosome 7. Ethanol ingestion may also be an important contributor to SAH. Drinking 100–299 g of ethanol per week accounted for 11% of the SAH cases while >300 g per week accounted for 21% and smoking for 20%. An additional 17% of the cases were attributed to hypertension, 11% to a positive family history of SAH. A prospective population-based cohort from the Nord-Trøndelag Health Study and the Tromos Study in Norway examined a potential synergism between cigarette smoking, hypertension and regular alcohol consumption. There was an additive relative excess risk between hypertension and current smoking (6.40; 95% CI: 0.88–11.92) in 122 cases of SAH followed for 977,895 person-years. These population studies suggest that screening and preventive treatment of patients with a family history of SAH will cause a modest reduction in the incidence of SAH. Reducing the prevalence of modifiable risk factors such as ethanol ingestion, smoking and hypertension will also reduce the incidence of SAH.

**Warning Signs and Symptoms**

The hallmark symptom of SAH is a thunderclap headache, often referred to as the ‘worst headache’ of a person’s life. Photophobia, meningismus, vomiting and syncope soon ensue. If severe headache is the only symptom, the chance of SAH is only 10%. Other causes of similar headaches in the differential diagnosis include severe acute paroxysmal headache, benign thunderclap headache (or crash migraine), reversible vasoconstrictive syndrome and benign orgasmic cephalgia. In 30%–60% of patients presenting with SAH, a transient headache may occur briefly for a day or so before clearing; these are called warning headaches from a sentinel haemorrhage. Warning headaches may occur without SAH and be due to aneurysmal enlargement or haemorrhage confined within the aneurysm wall.

The hallmark symptoms of SAH are the result of blood in the subarachnoid space irritating the meninges and spinal nerve roots, which may produce Kernig’s or Brudzinski’s sign. Fundoscopic examination often unveils ocular haemorrhages (20%–40%) classified into three categories: subhyaloid or preretinal haemorrhage (11%–33%), vitreous haemorrhage also called Terson syndrome (4%–27%), and intra-retinal hemorrhage. Other less specific and less commonly found symptoms include transient loss of consciousness, cranial nerve palsies, diffuse weakness, diplopia, ptosis and seizures. When aneurysm SAH is suspected based on these symptoms and findings from physical examination, lumbar puncture (LP) and radiographic imaging help to clinch the diagnosis. The initial clinical severity of acute SAH should be determined rapidly by using simple validated scales, including the Hunt and Hess Grading Scale and Fisher Scale, due to their usefulness as an indicator of potential outcome.

**Lumbar Puncture for cerebrospinal fluid analysis**

Analysis of the cerebrospinal fluid (CSF) for suspected SAH can be
helpful in making the diagnosis of brain aneurysm bleeding. Various methods can be used to obtain CSF but the simplest, fastest and safest way typically is with an LP. An LP can be performed at the bedside with local analgesia. Generally accepted guidelines dictate that an LP should be performed at least 6 h after symptom onset; this timing has been suggested because circulation of the blood cells in the CSF from the brain to the lumbar region will not be immediately present and can be missed if performed too early. The CSF should be collected in four separate sterile tubes and sent for analysis specifically looking for red blood cell (RBC) count in the first and fourth tubes to help distinguish between blood from SAH and a traumatic tap; spectrophotometric analysis is also performed to test for the presence of xanthochromia. RBCs lyse in the CSF, releasing haemoglobin. Heme-oxigenase-1 then degrades the haemoglobin into bilirubin in the CSF and this can be identified as a yellowish discoloration of the CSF, or xanthochromia. Sometimes xanthochromia can be seen with the naked eye against a white background, but spectrophotometric analysis is more sensitive and more reliable. Spectrophotometry has a specificity of 97% (95% CI: 92%–99%) but only 29% specificity with visual inspection (95% CI: 23%–35%).

Xanthochromia will not be apparent until the second to fourth hour after SAH and is not seen with a single traumatic tap. It is present in almost 100% by 12 h after the SAH, 70% at 3 weeks, and is still detectable at 4 weeks. A traumatic tap would result in a large number of RBCs in the first CSF tube and far fewer in the fourth tube once the blood from the tap has cleared through the needle. Characteristic findings of CSF analysis with SAH include xanthochromia and a high number of RBCs in all four collection tubes. Critical to note is that lowering the CSF pressure during the diagnostic process increases the risk of subarachnoid haemorrhage re-bleeding. It is therefore standard practice to carry out diagnostic lumbar punctures with the patient in the supine position, with the legs dependent.

Table 1  Risk factors for aneurysm formation (Reproduced with permission from Frontera®)

<table>
<thead>
<tr>
<th>Modifiable risk factors</th>
<th>Non-modifiable risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cigarette smoking (dose-dependent effect on aneurysm formation)</td>
<td>Previous subarachnoid haemorrhage (new aneurysm formation rate 1%–2% per year)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Polycystic kidney disease</td>
</tr>
<tr>
<td>Moderate to heavy ethyl alcohol use</td>
<td>Connective tissue disease (Ehlers–Danlos syndrome, Marfan syndrome)</td>
</tr>
<tr>
<td>Cocaine use</td>
<td>Aortic coarctation</td>
</tr>
<tr>
<td>Endocarditis (mycotic aneurysm)</td>
<td>Pseudoxanthoma elasticum</td>
</tr>
<tr>
<td></td>
<td>Moyamoya disease</td>
</tr>
<tr>
<td></td>
<td>Arteriovenous malformation</td>
</tr>
<tr>
<td></td>
<td>Fibromuscular dysplasia</td>
</tr>
<tr>
<td></td>
<td>Dissection w/pseudoaneurysm</td>
</tr>
<tr>
<td></td>
<td>Vasculitis</td>
</tr>
<tr>
<td></td>
<td>Neurofibromatosis 1</td>
</tr>
<tr>
<td></td>
<td>Glucocorticoid remediable hyperaldosteronism</td>
</tr>
<tr>
<td></td>
<td>Family history</td>
</tr>
</tbody>
</table>

Table 2  Hunt and Hess Grading Scale for subarachnoid haemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical exam</th>
<th>Assoc. mortality (%)</th>
<th>Mean Glasgow Outcome score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic, mild headache, slight nuchal rigidity</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Cranial nerve palsy, mod-severe headache, severe nuchal rigidity</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>Mild focal deficit, lethargy, confusion</td>
<td>19</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, mod-severe hemiparesis, early decerebrate rigidity</td>
<td>40</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity, moribound appearance</td>
<td>77</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 3  Modified Fisher and Fisher Scale for subarachnoid haemorrhage

<table>
<thead>
<tr>
<th>Grade</th>
<th>Modified Fisher</th>
<th>% with vasospasm</th>
<th>Fisher</th>
<th>% with vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No SAH or intraventricular haemorrhage (IVH)</td>
<td>—</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Thin SAH, no IVH</td>
<td>24</td>
<td>No SAH or IVH</td>
<td>21</td>
</tr>
<tr>
<td>2</td>
<td>Thin SAH w/IVH</td>
<td>33</td>
<td>Diffuse or vertical layers ≤1mm thick</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>Thick SAH, no IVH</td>
<td>33</td>
<td>Local clot and/or vertical layers &gt;1mm</td>
<td>37</td>
</tr>
<tr>
<td>4</td>
<td>Thick SAH w/IVH</td>
<td>40</td>
<td>Intracerebral or IV clot with diffuse or no SAH</td>
<td>31</td>
</tr>
</tbody>
</table>
an LP can precipitate re-bleeding by increasing the transmural pressure across the aneurysm dome. A second cautionary measure is to confirm radiographically that there is no contraindication to spinal tap such as non-communicating hydrocephalus from blood in the fourth ventricle or early signs of brainstem herniation, both of which could result in further neurological compromise and death. Therefore, it is recommended that only a small amount of CSF be removed and a small (≤20G) spinal needle be used to avoid post-LP CSF leakage and delayed herniation. Patients arriving to the hospital later than day three after symptom onset should always have an LP even if computed tomography (CT) scan does not show SAH; CT scan loses its sensitivity for SAH detection beyond this time period.

**Imaging Analysis**

**CT for SAH**

CT is a very sensitive radiographic means of detecting acute SAH but should not be relied on as the sole investigational tool (Figure 2). Older generation CT scanners detected acute SAH 93%–95% of the time within the first 24 h after onset of symptoms. The sensitivity of CT scans for SAH decreases with time as the blood products disperse within the CSF, clots dissolve and blood gets reabsorbed from the spinal fluid. Modern CT scanners are more sensitive detectors of SAH with reports of 100% sensitivity up to 5 days after symptom onset and an overall sensitivity of 99.7% (95% CI: 98.1%–99.9%) and specificity of 100% (99.2%–100%)\(^{16}\). Despite the high sensitivity and specificity, suspected SAH with a negative CT scan still requires LP to confirm the absence of SAH\(^{16}\).

Other studies report that CT sensitivity is slightly lower, between 95% and 100% on the first day, falling to about 58% at 5 days and less than 50% after 1 week\(^{17}\). By day 10, the blood may have been totally resorbed. Some controversy exists about the utility of LP if CT scans are negative for SAH within 6 h of ictus\(^{18}\). A more recent study completed in 2012 found that the sensitivity of head CT within 6 h of ictus was 98.5% (95% CI: 92.1%–100%), diagnosing all patients with aneurysmal and perimesencephalic SAH; sensitivity of CT performed after 6 h was only 90.0% (95% CI: 76.3–97.2)\(^{19}\). Thus, this group concluded that there was no added value of CSF analysis in patients presenting with acute headache and normal head CT within 6 h after ictus. In patients with an atypical presentation however, it is still recommended that CSF analysis be performed to look for RBCs and xanthochromia. It is our opinion that patients presenting with hallmark signs of aneurysm SAH and a negative CT scan of the brain get an LP for CSF analysis, as this remains the standard of care.

**CT Angiography**

The accuracy of CT angiography (CTA) for identifying brain aneurysms, particularly helical CTA, is approaching that of catheter-based digital subtraction angiography (DSA).

DSA remains the gold-standard study for the radiographic diagnosis and pre-treatment planning for patients with brain aneurysms. Advantages of CTA over DSA include its non-invasive nature, fewer resources, lower risk of complications and morbidity, and suitability for critically ill or unstable patients\(^{20}\).

The negative predictive value of CTA for detecting aneurysm ranges from 82% to 96% on a multi-detector CT scanner with sensitivity and NPV approaching 100% on a ‘per aneurysm’ basis\(^{17}\). The lower sensitivity is associated with aneurysms measuring less than 3 mm but may be improving with newer techniques.

**Figure 2:** Non-contrast enhanced computed tomography (CT) reveals subarachnoid haemorrhage. Arrows indicate blood in the left sylvian fissure.
and the 64-slice multi-detector CT scanner. The introduction of spiral CTA has led to improved resolution and prompted some claims that CTA achieves equivalent diagnostic accuracy to that of DSA for brain aneurysms. However, the sensitivity for the detection of aneurysms smaller than 3 mm is still felt to be less than 90%\(^1\). Depending on the history and pattern of blood on the non-contrasted brain CT scan, a negative result on CTA still warrants further investigation.

**Magnetic Resonance Imaging and Angiography**

In cases of proved SAH where CTA and DSA are negative magnetic resonance imaging (MRI) is used to search for other causes of SAH. MRI is useful for detecting cavernous angio- mas of brain or spinal cord and cerebral venous sinus thrombosis. MRI is more sensitive than CT in detecting SAH (91%–100%) after day five using a gradient echo sequence\(^1\). Magnetic resonance angiography (MRA) is used mainly to monitor already diagnosed aneurysms and those treated with surgical clip ligation and endovascular embolisation. It has a sensitivity of about 94% when the aneurysm is greater than 3 mm in size but only 38% for smaller ones; gadolinium-enhanced MRA may increase the sensitivity\(^2\). For medium- and large-sized aneurysms, the sensitivity of MRA is comparable to CTA. CTA remains superior to MRA for aneurysms smaller than 5 mm, with MRA sensitivity dropping to 56% for aneurysms of this size range\(^3\).

**Catheter-based DSA**

DSA remains the gold-standard radiographic test for detecting pathology of the intracranial vasculature (Figure 3). If SAH is confirmed and CTA is negative, then DSA should be considered depending on the blood pattern on plain CT. DSA is minimally invasive and associated with <1% risk of complications. More specifically, it carries up to a 0.8% risk of serious non-neurological complications and a 0.5% rate of transient neurological complications. In addition, one study reported a 2.6% risk of a second haemorrhage from a ruptured aneurysm during DSA performed within 6 h after initial SAH\(^4\).

The risk associated with DSA, though minimal, has been the impetus for further evaluation of the efficacy of three-dimensional (3D)-CTA compared with DSA for aneurysm diagnosis of brain aneurysm after SAH. 3D-CTA was found to have an overall sensitivity of 96.3%, specificity of 100% and accuracy of 94.6%, comparable to that of DSA. However, DSA is better for detecting smaller aneurysms. Aneurysms less than 3 mm in diameter were more likely to be detected on DSA (90.9% sensitivity) than with 3D-CTA (81.8% sensitivity)\(^5\). In a comparative study on diagnostic validity of cerebral aneurysm by CTA vs. DSA after SAH, CTA was 89% sensitive and 100% specific, whereas DSA was 74% sensitive with the same specificity. The positive predictive value of both methods was 100%, but the negative predictive value was higher for CTA (85%) than DSA (69%)\(^6\). If the surgery is carried out based on CTA alone, aneurysms less than 4 mm may be missed and not repaired during surgery.

**Evaluation of Radiographically Negative SAH**

Previous studies suggest that the overall incidence of DSA-negative SAH ranges from 10% to 20%\(^7\). A definitive diagnosis in this sub-
group is only obtained 2%–21% of the time. Some possible causes of a confirmed SAH on CT or LP with negative radiographic imaging include auto-thrombosed aneurysms, venous perimesencephalic haemorrhage, venous sinus thrombosis, vascular lesions of the spine, spinal neoplasms, pregnancy-induced hypertension, sympathomimetic drug abuse, bleeding dyscrasias, antiplatelet agents and anticoagulants. Failure to detect a vascular lesion places the patient at risk for recurrent haemorrhage. Strategies for managing these patients with radiographically negative SAH include subsequent DSA approximately 1 week after the ictus in an effort to detect missed vascular lesions such as thrombosed aneurysms that may recur, microarteriovenous malformation (AVM), and dural or pial arteriovenous fistula (AVF)\textsuperscript{11}. Further outpatient vascular imaging should be performed when the index of suspicion remains high despite previous negative studies. Con-trasted MRI of the brain and cervical spine, diffusion weighted imaging (DWI) and fluid attenuated inversion recovery (FLAIR) of the brain, should be considered to look for subtle intracranial lesions such as neoplasms, vascular and cavernous malformations. Laboratory analysis should also be considered to evaluate for vasculitis, coagulopathies, drug use, and pregnancy-related hypertension.

**Initial Management and Treatment**

**Blood Pressure**

Control of hypertension is very important to lower the risk of re-bleeding from the tenuous aneurysm wall\textsuperscript{26}. Maintaining normotension, systolic blood pressure (SBP) < 130 in patients presenting with SAH can be performed quickly with calcium-channel blockers or β-blockers. We commonly use nicardipine or labetalol, sometimes as a combined therapy in difficult-to-manage situations. The American Heart and Stroke Associations recommend nicardipine, labetalol and esmolol because of their short half-life, reliable dose response and favourable safety profile. They recommend avoiding nitroprusside in neurological emergencies because of its tendency to raise intracranial pressure and cause toxicity with prolonged infusion\textsuperscript{26}. A retrospective study evaluated the use of nicardipine or labetalol for blood pressure control in patients with SAH and concluded that nicardipine was superior\textsuperscript{27}. After the aneurysm has been secured, less stringent blood pressure parameters are tolerable.

**Re-bleeding**

For untreated ruptured aneurysms, the re-bleed rate is 4% on the first day then 1%–1.5% everyday thereafter until the aneurysm is secured. The longer term re-bleed rate for unsecured brain aneurysms is 15%–20% in 2 weeks, 50% in 6 months and then drops to ~3%/year with a mortality rate of 2%/year. About 50% of the patients who re-bleed will die\textsuperscript{1}. A recent retrospective review found that there was a higher risk of re-bleeding in patients with intracerebral or intraventricular haematoma, high serum glucose level (>113.8 mg/dl) on admission, posterior circulation aneurysms, male gender, hypertension and worse clinical status\textsuperscript{28}. The best method for preventing aneurysm re-bleeding is early treatment of the aneurysm with surgical clip ligation (Figure 4) or endovascular embolisation (Figure 5)\textsuperscript{26}.

Antifibrinolytic agents have been used to reduce the incidence of early re-bleeding in patients with SAH. Early systemic heparinization appears to be the best method to prevent early re-bleeding\textsuperscript{29}. The dose and the duration of heparinization remain to be defined. A recent meta-analysis found that intravenous heparin therapy is associated with lower risk of early re-bleeding, decreased intracranial pressure, and reduced mortality but increases the risk of systemic complications\textsuperscript{30}. The best heparin regimen for the prevention of early re-bleeding remains to be determined.

![Figure 4: Aneurysm clip on the base of a posterior communicating artery aneurysm. The arrow points to the neck of the aneurysm pinched closed by the clip. The filled arrow-head indicates the location of the right optic nerve.](image)
re-bleeding when early definitive treatment is not available. Because antifibrinolytics can cause vasospasm, their use should be limited to within 72 h of ictus and should not be used in patients with coagulopathy, history of myocardial infarction, ischemic stroke, pulmonary emboli or deep venous sinus thrombosis.

The antifibrinolytic, tranexamic acid can reduce early re-bleeding rates and adverse outcomes (from 10% to 2%) when the drug was administered immediately after the diagnosis of SAH. However, because of the significant prothrombotic side effects of antifibrinolytic agents and the advent of endovascular therapy, these drugs are rarely used today.

**Hydrocephalus**

Acute hydrocephalus can result from SAH blood obstructing the flow of CSF through the ventricular system or by altering reabsorption by the arachnoid granulations. Radiographic evidence of hydrocephalus will occur in 15% of patients with SAH, 40% of these will become symptomatic (Figure 6). Hydrocephalus is more prominent in those of increased age, poor clinical grade and more extensive blood on CT scan. For symptomatic hydrocephalus, an external ventricular drain (EVD) may be needed emergently to drain CSF and reduce intracranial pressures that would otherwise result in brainstem herniation syndrome. Care must be taken not to overdrain the CSF, because this would change the transmural pressure across the aneurysm dome and increase the risk of re-rupture; EVDs are adjusted to maintain intracranial pressures at 15–20 cm of H$_2$O.

**Seizures**

The incidence of seizure activity at the time of SAH is 25%, most commonly seen after an MCA aneurysm rupture. A seizure at the onset of aneurysm rupture is a predictor of poor outcome. Seizures increase the risk of aneurysm re-rupture in a patient with an unsecured aneurysm.

**Figure 5:** Arrowhead points to a basilar tip aneurysm occluded with coils after subarachnoid haemorrhage. Arrow indicates narrowing of the basilar artery indicative of severe cerebral vasospasm.

**Figure 6:** Computed tomography slice reveals subarachnoid haemorrhage with hyperintense areas in the centre corresponding to blood in the subarachnoid basal cisterns. Laterally, there is a discrete widening of the temporal horns of the lateral ventricles, indicating that the patient has hydrocephalus.
Prophylactic use of antiepileptic drugs in patients with SAH is controversial. A retrospective study investigating the impact of prophylactic phenytoin use in SAH patients resulted in worse cognitive outcomes at 3 months after SAH\textsuperscript{33}. However, the development of safer drugs with fewer side-effects may be beneficial in high risk patients with SAH suffering from intraparenchymal bleeds or those who are comatose and close neurological monitoring is not possible.

**Considerations in Delayed SAH Presentation**

**Hyponatraemia**

The most common electrolyte imbalance in patients with SAH is hyponatraemia. The reported incidence of hyponatraemia after SAH ranges from 10\% to \textasciitilde{}30\%. It is more common in patients with poor clinical grade, anterior communicating artery aneurysms and hydrocephalus; and it may be an independent risk factor for poor outcome\textsuperscript{26}. Both syndrome of inappropriate antidiuretic hormone (SIADH) and cerebral salt wasting (CSW) are common causes of hyponatraemia in the setting of SAH. These electrolyte imbalances usually do not occur immediately after SAH and are not dealt with during the initial assessment. However, patients who present in a delayed fashion may have hyponatraemia needing correction with hypertonic saline solution. It is important to recognise the aetiology of hyponatraemia because SIADH represents positive water balance while CSW is associated with a negative total body water balance, the latter of which can exacerbate cerebral vasospasm (CV) and cerebral ischaemia if treated inappropriately\textsuperscript{34}. Current guidelines recommend avoidance of large volumes of hypotonic fluids and intravascular volume contraction.

**Cerebral Vasospasm**

CV is a constriction of the cerebral vasculature that results from blood products circulating in the CSF from SAH. It is believed that the SAH causes an inflammatory reaction in the basal cisterns of the brain inducing CV, the most common cause of delayed cerebral ischaemia and neurological deficits after SAH. The risk of CV increases with the amount of blood in the subarachnoid space and typically does not begin before the fourth day after SAH and can last for 14–21 days\textsuperscript{10,35}. CV can be detected on radiographic imaging and also by bedside transcranial Doppler studies (TCDs). Although the specificity and sensitivity of TCDs are operator dependent, severe CV can be identified with fairly high reliability\textsuperscript{26}. DSA remains the gold standard for the diagnosis of CV and it assesses vessel calibre and cerebral blood flow (Figure 5).

Other ancillary tests that may aid in the diagnosis of CV include static radiographic studies such as CTA and MRA. Cerebral blood flow evaluation with CT and MR perfusion (Figure 7), and SPECT scans may be more useful for clinical management, but most important is continuous neurological assessment with careful neurological examinations.

**Treatment Options**

Treatment options for ruptured brain aneurysms consist of craniotomy with surgical clip ligation and endovascular embolisation. A detailed discussion of the benefits and risks of each treatment option are beyond the scope of this text. In brief, however, a neurosurgeon must be consulted to assess the patient with SAH and...
discuss treatment options and perform immediate intervention to stabilise the patient’s intracranial pressures and secure the aneurysm. If no neurosurgical services are available at the institution, the patient should be stabilised for transfer to a facility that can provide all treatment options and post-treatment intensive care unit services (Figure 8). Early aneurysm treatment significantly reduces the risk of lethal rebleeding.

When deciding surgical or endovascular treatment of a ruptured aneurysm, sac morphology, aneurysm location, presence of an intraparenchymal clot, medical co-morbidities and experience of the treating neurosurgeon or endovascular specialist are all important factors.

**Conclusion**

It is imperative that emergency department and primary care physicians understand how to diagnose and initially manage aneurysm SAH. Recent evidence-based literature recommends non-contrasted head CT scan as the initial first step in evaluation of SAH followed by LP if radiographic imaging is non-diagnostic. CSF analysis with spectrophotometry provides a specificity of 97% for xanthochromia. Advancing technology has improved the sensitivity and specificity of CTA for detecting brain aneurysms and approaches that of DSA. However, DSA remains the gold-standard and serves as an important tool for planning treatment. It is important to control hypertension immediately following SAH to reduce the risk of an aneurysm re-bleed. A high suspicion for SAH during the initial evaluation increases the patient’s chance for early intervention and reduces mortality and disability.

**Abbreviations list**

3D, three-dimensional; CI, confidence interval; CSF, cerebrospinal fluid; CSW, cerebral salt wasting; CTA, CT angiography; CV, cerebral vasospasm; DSA, digital subtraction angiography; EVD, external ventricular drain; LP, lumbar puncture; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; OR, odds ratio; RBC, red blood cell; SAH, subarachnoid haemorrhage; SIADH, syndrome of inappropriate antiuretic hormone; TCD, transcranial Doppler studies.

**References**


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**FOR CITATION PURPOSES:** Simpson VM, Deshaies EM. Diagnosis and initial management of subarachnoid haemorrhage. OA Emergency Medicine.
Figure 8: Modifications to current diagnostic protocols arranged according to the distribution of haemorrhage on admission CT scans. (a) Proposed diagnostic algorithm for patients with classic haemorrhage distribution. Detailed follow-up testing is recommended given the likelihood of finding a causative lesion. (b) Diagnostic algorithm for patients with perimesencephalic haemorrhage. Delayed follow-up vascular imaging is performed to detect posterior circulation aneurysms. Laboratory studies (e.g. inflammatory markers) and MRI are of low yield in this subgroup and have been excluded. (c) Diagnostic algorithm for patients with an abnormal lumbar puncture. Laboratory studies (e.g. inflammatory markers) are of low yield in this subgroup and have been excluded. Follow-up MRI of the brain with FLAIR could be considered in patients in whom the lumbar puncture is non-diagnostic or to confirm a positive result; follow-up MRI of the cervical spine is recommended in patients with signs and symptoms of spinal pathology. (Reproduced with permission from Little et al.)