



## Subarachnoid Haemorrhage: A Systematic Review

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**ABSTRACT:** Subarachnoid haemorrhage arises from the accumulation of blood between the arachnoid and pia mater resulting from an aneurysmal rupture or traumatic head injury. Subarachnoid haemorrhage is a life-threatening emergency that requires prompt treatment. The characteristic presenting symptom is the thunderclap headache, which patients may describe as the "worst headache of life" and may prompt further imaging. Associated symptoms include nausea, vomiting, and diplopia. Often signs of meningismus are present due to the presence of blood in the fourth ventricle and further down the spinal cord, causing neck and back pain. Findings of focal deficit increase the grade of subarachnoid haemorrhage and patients with a high-grade subarachnoid haemorrhage report to the Emergency room in a state of coma requiring evaluation and urgent treatment, as the coma can be reversible. Sometimes concurrent intraocular haemorrhage, known as Terson syndrome may be present warranting an ophthalmology service. Eighty per cent of patients that develop Terson syndrome require regular follow-up visits but may not require further intervention. This activity reviews the evaluation and management of subarachnoid haemorrhage (SAH).

**KEYWORDS:** Aneurysm, Hunt Hess scale, Subarachnoid haemorrhage.

### INTRODUCTION

Subarachnoid haemorrhage is a type of head bleed requiring fast and appropriate care for each patient. Subarachnoid haemorrhage is defined as blood between the arachnoid membrane and the pia membrane. Trauma remains the most common cause though Aneurysmal subarachnoid haemorrhage comprises a small portion of this patient population, but is the most worrisome type of subarachnoid haemorrhage. Spontaneous subarachnoid haemorrhage should raise concern for rupture of aneurysm. Aneurysmal ruptures mostly occur in patients older than 50 years of age. Typically seen after strenuous exercise or postcoital activity. 30% of subarachnoid haemorrhages occurs during sleep. Further imaging should be done for intense headaches in a patient that is not prone to headaches. While subarachnoid haemorrhage is also associated with intraventricular haemorrhage, intracerebral haemorrhage, and subdural hematoma depending upon the force of the rupture and location of an aneurysm. Mental status changes should be noted because patients are prone to seizures as well as hydrocephalus and vasospasm, both potentially devastating factors.<sup>[1][2][3]</sup>

### AETIOLOGY

The majority of the cases of SAH (85%) arise due to a ruptured saccular aneurysm. The circle of Willis and its branches are the common sites of these aneurysms. Precipitating factors of SAH include cocaine abuse, dissection of a vertebral artery, sickle cell anemia and anticoagulation disorders. Positive family history with two or more first-degree relatives who have had an aneurysmal SAH warrants preventive screening. Also autosomal dominant polycystic kidney disease (ADPKD) is known to be associated with up to 8% of patients with cerebral aneurysms.<sup>[4]</sup> Trauma remains another significant cause of SAH. Initially, the surgical correction of cerebral aneurysms was introduced back in the 1930s, and later in the 1990s coiling, and clipping became popular less-invasive treatment options for patients with SAH. Studies have concluded that aneurysms that are smaller than 10 mm or an aneurysm that has never bled are highly unlikely to have a SAH and may require no surgical or preventative intervention.<sup>[5]</sup>

### PATHOPHYSIOLOGY

Soon after blood is released into the subarachnoid space after an inciting event it comes close to the brain surface acting as an irritant, and many complications of subarachnoid haemorrhage are due to the irritant effect of blood on the brain. Primarily brain responds



with seizures, vasospasm, and confusion. Subarachnoid haemorrhage has effects outside the brain such as neurogenic pulmonary oedema and neurogenic stunned myocardium. Blood products eventually circulate with cerebral spinal fluid impeding normal fluid clearance in arachnoid granulations resulting in hydrocephalus. If left untreated fluid collection in the ventricular system will eventually create enough pressure to cause brain herniation syndromes and possible death. Neurosurgical intervention should be done on priority when hydrocephalus is identified.<sup>[6]</sup>

**CLINICAL FEATURES**

Patients classically present with a severe rapidly progressing headache that develops within seconds to minutes and has a maximal intensity at its onset. Associated symptoms include neck stiffness, vomiting, decreased level of consciousness, hemiparesis, and occasionally, seizures. The typical headache pattern is characterized by pulsatile pain. Seizures are more common in aneurysmal SAH. Neck stiffness typically occurs around 6 hours after the onset of a SAH. Rising intracranial pressure due to brain herniation causes asymmetrical pupil size and loss of pupillary light reflex. Vitreous haemorrhage as a result of a severe SAH is called Terson syndrome and occurs in 3% to 13% of cases.<sup>[7]</sup> Increased intracranial pressure can cause sympathetic storm due to activation of the descending sympathetic nervous system at the medulla resulting in an increase in blood pressure, cardiac arrhythmias, and/or cardiac arrest. In addition electrocardiographic changes such as large U waves, T wave abnormalities, QT prolongation, Q waves, cardiac arrhythmias and ST changes (elevation or depression) may be seen. Neurogenic pulmonary edema may occur as a secondary complication.<sup>[8]</sup> Patients may reveal a history of a head injury before symptoms ensue or a known history of a cerebral aneurysm. Additional risk factors comprise high blood pressure, smoking, family history, and alcohol/drug abuse. Patients having a history of a prior severe headache and/or a history of a small bleed with settling symptoms within the past month are considered to have a sentinel bleed which often precedes a more severe SAH. A headache from a sentinel bleed is very difficult to diagnose, as CT and LP can be inconclusive in these patients. Findings such as oculomotor abnormality/palsy may indicate the posterior communicating artery as the source of the bleed. Meningeal irritation from the SAH may cause neck stiffness.<sup>[7]</sup> In this case, the Kernig sign (defined as the inability to fully extend the knees when the thigh is flexed at the hip and the knee is at 90-degree angles) and Brudzinski sign (demonstrated by hip and knee flexion with passive neck flexion) may also be positive.<sup>[7]</sup> Ottawa SAH clinical decision rule is designed to facilitate the identification of sub arachnoid haemorrhage.

Inclusion criteria: alert, adult (>15 years old) patients with new, severe, non-traumatic headache reaching maximal intensity within one hour. Exclusion criteria: new neurologic deficits, prior history of aneurysm, subarachnoid hematoma, or brain tumor, or history of recurrent headaches (≥ 3 episodes in ≥ 6 months)

- Age ≥ 40
- Neck pain/stiffness
- Witnessed loss of consciousness
- Onset with exertion
- Instantly peaking/thunderclap headache
- Limited neck flexion

**Table 1:** HUNT HESS scale

Grade	Clinical description
(1)	Asymptomatic or mild headache
(2)	Moderate to severe headache , nuchal rigidity, can have oculomotor palsy
(3)	Confusion, drowsiness or mild focal signs
(4)	Stupor or hemiparesis
(5)	Coma, moribund or extensive posturing

**EVALUATION AND TREATMENT**

Hunt Hess and Cooperative aneurysm study scales respectively are used for the severity of SAH table 1 and 2. Diagnostic modalities available to assess for SAH include non-contrast Computed Tomography(NCCT), Lumbar Puncture(LP), Computed Tomographic Angiography(CTA), and Magnetic Resonance Imaging(MRI). Realizing the high mortality trends in missed SAH cases, a diagnostic



strategy with the highest sensitivity is employed which is currently accepted to be non-contrast CT followed by LP if NCCT is negative.<sup>[9][10][11]</sup> This is supported by both the American Heart Association (AHA) and the American College of Emergency Physicians (ACEP). While CT/LP remains the most accepted rule-out method, other approaches do exist. Many practitioners have endorsed the recent publications showing non-contrast CT to be a sufficient stand-alone study if completed within six hours.<sup>[12]</sup> Once the diagnosis of SAH is confirmed, the most important measures to be taken include confirmation of airway security and stabilization of hemodynamics. Intubation should be attempted in cases of low Glasgow Coma Scale Score or inability to safeguard the airway, but care should be taken to mitigate rise in mean arterial pressure (MAP) during the intubation process. Cardiac monitoring is important, as patients with severe brain injury are at risk for neurocardiogenic stunning.<sup>[13]</sup> Reversing anticoagulation should be done on priority. Vitamin K antagonists can be reversed with phytonadione (vitamin K) and 4-factor prothrombin complex concentrate (PCC) or fresh frozen plasma. Direct thrombin inhibitors such as dabigatran can be reversed with idaricizumab.<sup>[14]</sup> In addition to efficient reversal of anticoagulation, measures to reduce the risk of aneurysmal re-rupture include controlling pain, nausea, and valsalva effect by treatment with analgesics, antiemetics, and stool softeners as needed. Many patients with SAH will require ventriculostomy drainage, either for hydrocephalus or periprocedurally to help with ICP complications.

**Table 2:** Cooperative aneurysm study scale.

Grade	Clinical description
(1)	Free of symptoms
(2)	Mildly ill, alert and responsive, headache present
(3a)	Moderately ill - lethargic, headache, no focal signs
(3b)	Moderately ill - alert, focal signs present
(4a)	Severely ill - No focal signs, stuporous
(4b)	Severely ill - drowsy, major focal signs present

The ultimate therapeutic goal, once a bleeding aneurysm is identified, is to secure it surgically by coiling or clipping. While coiling is the preferred method,<sup>[15]</sup> since it is less invasive than open surgical clipping. Once the aneurysm is secured, the greatest risk to patient outcome is that of vasospasm and delayed cerebral infarction (DCI). Treatment can be catheter-directed calcium channel blocker administration, such as nicardipine or verapamil, or vessel angioplasty.<sup>[16,17,18]</sup>

**CONCLUSION**

Most patients with subarachnoid haemorrhage report to the emergency room. Thus, it is crucial that the emergency department physician and nurse practitioner know the workup for these patients. Despite advances in the diagnosis and treatment of aneurysmal subarachnoid haemorrhage, mortality remains high. Unfortunately, despite the optimal care, majority of patients die within 30 days. Disabling severe complications is often seen in those who survive SAH.

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*Cite this Article: Wani Mubashir, Sara Khalid, Mehraj Sharik, Aman Ala, Nayarah Nisar, Khibrat Peerzada, Waseem Ahmad, Aleena Ahmad, Azra Khan, Aamir Shafi (2022). Subarachnoid Haemorrhage: A Systematic Review. International Journal of Current Science Research and Review, 5(8), 3216-3219*