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Chapter

Aneurysmal Subarachnoid Haemorrhage (aSAH) and Hydrocephalus: Fact and Figures

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Abstract

Hydrocephalus (HCP) occurs due to the injurious effect of subarachnoid haemorrhage (SAH). It causes increased morbidity and mortality. It can be acute and frequently occurs within 48 hours and up to 7 days. Subacute hydrocephalus may occur up to 14 days and is chronic if remained or develops after 2 weeks of the subarachnoid haemorrhage. Acute hydrocephalus after aneurysmal subarachnoid (aSAH) bleeding is non-communicating or obstructive and occurs due to physical obstruction by a clot, the effect of blood in the subarachnoid space, and inflammation. Chronic hydrocephalus is due to fibrosis and adhesion, which hampers cerebrospinal fluid (CSF) absorption and increased secretion of CSF from gliosis. Various risk factors for developing hydrocephalus in aneurysmal subarachnoid haemorrhage patients range from female gender to high severity scores. Acute hydrocephalus frequently requires diversion drainage of CSF by external ventricular drain (EVD); it usually subsides within a week, and EVD is removed. Fewer patients will develop or continue to have hydrocephalus, requiring either short or longer shunting of the CSF namely by ventriculoperitoneal shunt or other modes of CSF drainage.

Keywords: aneurysmal subarachnoid haemorrhage, cerebrospinal fluid (CSF), hydrocephalus, communicating, non-communicating, external ventricular drain (EVD), ventriculoperitoneal (VP) shunt

1. Introduction

Subarachnoid haemorrhage (SAH) is blood in the space between the arachnoid membrane and pia matter around the brain (subarachnoid space). Hydrocephalus after aSAH (aneurysmal subarachnoid haemorrhage) is a frequent complication that can be acute, subacute, or chronic, requiring a diversion procedure such as external ventricular drain (EVD) or ventriculoperitoneal shunt insertion.

Up to 85% of subarachnoid haemorrhage occurs due to the rupture of a cerebral aneurysm, and SAH (subarachnoid haemorrhage) accounts for 5% of strokes. The overall incidence of aneurysmal subarachnoid bleeding is decreasing trend. However, aSAH (aneurysmal subarachnoid haemorrhage) remains a devastating clinical emergency with increased morbidity and mortality. aSAH varies according to geographic variation, with Finland and Japan having higher cases of SAH [1].

2. Epidemiology

The incidence of hydrocephalus (HCP) after aSAH is reported from 6 to 67%, and this wide range is due to various backgrounds and clinical situations of reporting. More recently, the incidence of hydrocephalus has been reported to be around 20–30% of subarachnoid haemorrhage [2].

One of the tertiary cares centre from the Indian subcontinent reported that 18.6% of their aSAH had hydrocephalus. Posterior circulation aneurysms were found to cause more frequent hydrocephalus than anterior circulation aneurysms [3].

3. Classification and types of hydrocephalus in aSAH

Hydrocephalus (HCP) in aSAH is divided into acute, subacute and chronic. In most of these patients, HCP is acute and occurs within 3 days of the bleeding. Subacute HCP occurs within 4 to 14 days of bleeding, and hydrocephalus after 2 weeks of aSAH is called chronic HCP, and it occurs in up to 20% of aSAH patients.

Further to it, HCP can be communicating or non-communicating. In the initial days of aSAH, it is non-communicating due to blockage and obstruction to the free flow of CSF due to narrowing and obstruction of the cerebral aqueduct. However, when it becomes chronic, it becomes communicating due to fibrosis of subarachnoid granulation. In this type, the flow of CSF is obstructed or blocked after the cerebral ventricles and usually results from a thickened arachnoid layer. Details are described in the etiopathology section.

4. Risk factors

Various reports suggest that the following are the risk factors for the development of acute hydrocephalus (obstructive or non-communicating) following aSAH are mentioned as follows: [4]

1. Female gender
2. Higher grade of aSAH
3. Higher modified fissure grade
4. Location of aneurysm
5. Meningitis

6. Known hypertension

According to Chen S et al., following are risk factors for communicating, non-obstructive chronic hydrocephalus [5]

1. Poor neurological grades
2. Massive intraventricular haemorrhage
3. Rebleed
4. Increased CSF secretion
5. Impaired CSF absorption
6. Advanced age

HCP in aSAH with the increased risk for shunt requirements [6]:

1. Larger aneurysm
2. Posterior circulation ruptured aneurysm
3. Intraventricular haemorrhage
4. Higher Hunt and Hess, Modified Fisher grade, and low admission Glasgow comma score (GCS)
5. Rebleeding and more significant intraventricular haemorrhage
6. Elderly patients (age > 60 years)

Other factors, like economy, medical development and techniques for aneurysms obliteration also detect the requirement of temporary or permanent shunts [7].

5. Pathophysiology of HCP in aSAH

The hydrocephalus after aSAH is either obstructive (non-communicating) or non-obstructive (communicating) hydrocephalus. Acute hydrocephalus is usually obstructive as the blood in the subarachnoid space causes both physical obstruction and direct effect on neurons (**Figure 1**). Blood clots in the CSF circulation cause obstruction to the flow, and platelets stimulation TGF (transforming growth factor) initiate fibrosis in the subarachnoid space. Thrombin of the blood in CSF causes epithelial ciliary damage and obstruction of CSF flow, and the product of blood formation also contributes to the ciliary damage. The direct effect of the blood in subarachnoid space leads to direct injury to neurons and releases HMGB1 (High Mobility Group Protein B1), which stimulates toll-like receptors (TLRs) and RAGE (*Receptor* for Advanced Glycation End products), leading to an acute surge of pro-inflammatory mediators (IL1, IL6 and TNF-9), and this contributes to ciliary

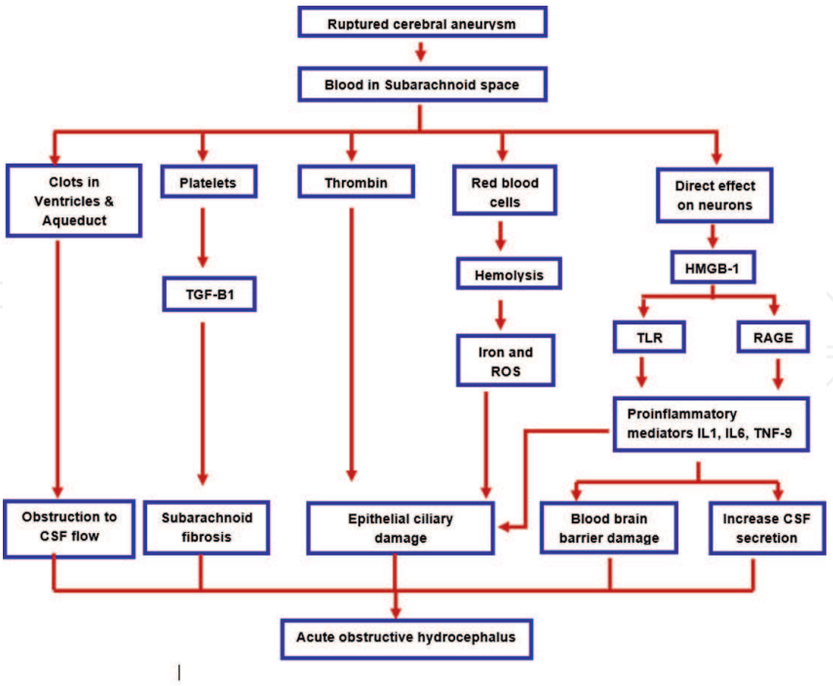

Figure 1.
Pathophysiology of acute hydrocephalus in aSAH.

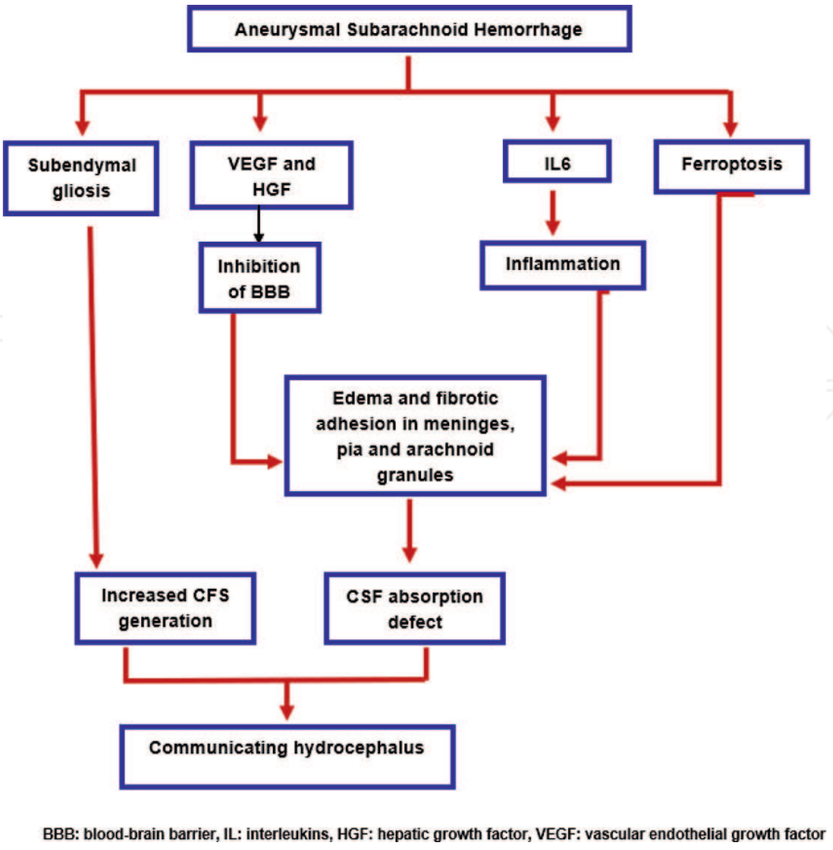

Figure 2.
Chronic hydrocephalus pathophysiology.

damage as well as subarachnoid fibrosis and ultimately contributes to the acute hydrocephalus formation (**Figure 1**) [8].

Chronic hydrocephalus after aneurysmal subarachnoid haemorrhage is either communicating or non-obstructive type. The pro-inflammatory biomarkers surge and presence of iron cause inhibition of the blood-brain barrier and pro-inflammatory changes, oedema and ferroptosis. The combined effect of these is the fibrotic changes and adhesions in the meningeal pia mater and arachnoid granules (**Figure 2**). Subependymal gliosis causes increased secretion of CSF, and all these combinations can cause communicating or non-obstructive hydrocephalus in aSAH patients (**Figure 2**) [8, 9].

6. Diagnosis

Acute hydrocephalus after aSAH has no specific clinical signs or symptoms. Often, patients show a sudden decrease in the level of consciousness or Glasgow comma score (GCS). Other typical manifestations are vomiting, papilloedema, increasing headache and dizziness. Imaging studies are essential to diagnose hydrocephalus after aneurysmal and non-aneurysmal subarachnoid haemorrhage. The imaging studies are ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI). CT is the preferred method for diagnosis as it is quick and widely available and has a short examination time. Current guidelines and protocols recommend the Evan's index (IE)

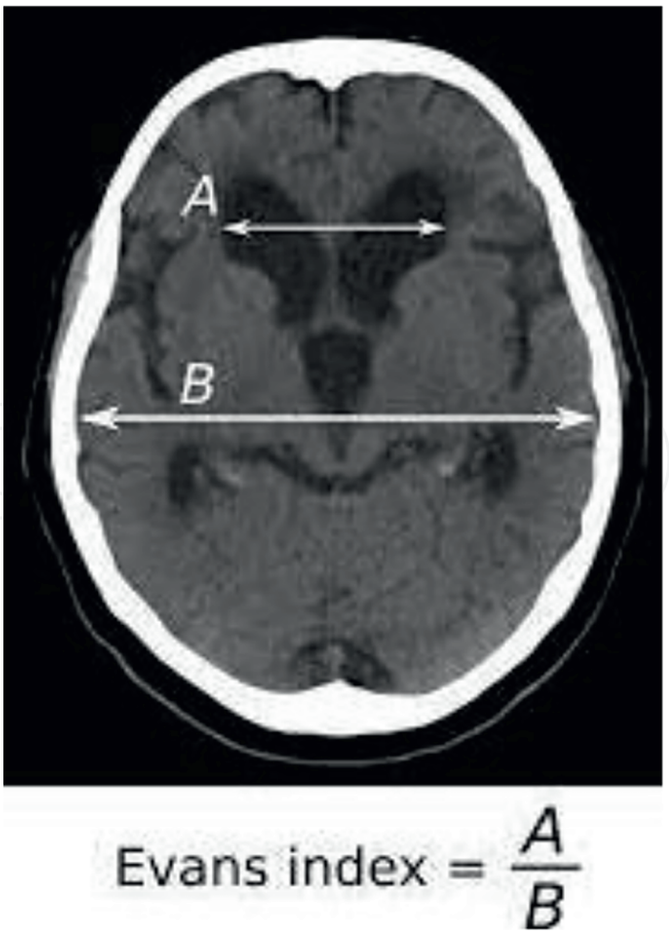

Figure 3.
Evan index.

as the gold standard for hydrocephalus. Evan's index can be used as a marker for ventricular enlargement or ventriculomegaly and ventricular volume. Evan's index gives a rough idea of ventricular dilatation, and one must be careful as it varies with location and slice angle. IE is defined or proposed by William Evan in 1942, as the ratio of the maximum width of the frontal horn of the lateral ventricle and the maximum internal diameter of the skull, as the same CT or MRI axial level (**Figure 3**). A normal Evans index is between 0.20 and 0.25, an index between 0.25 and 0.30 indicates possible or early ventriculomegaly, while a ratio of >0.30 indicates definite ventriculomegaly [10].

7. Management

Acute hydrocephalus is managed by the insertion of EVD (external ventricular drain). Bhattacharjee et al. described in their experience that 15% of their SAH patients required EVD insertion and only 9% required VP shunt [3].

EVD is required for a few days; as the CSF secretion and hemodynamics stabilise, it can be weaned and removed. The issue with EVD is that it can slowly remove blood and blood products and have a risk of blockage. The role of continuous drainage of CSF through EVD is to relieve the symptoms, clear the blood, reduce shunt occlusion and improve cerebral perfusion [3]. Olson et al. demonstrated that the intermittent opening of the EVD is safe and has lower malfunctioning [11].

There are two schools about removing the EVD. One school suggests removing EVD as early as possible and early, as it helps in opening the clogged CSF pathway, hence acquiring early normal flow of CSF, reducing the risk of ventriculitis and ultimately decreasing the length of intensive care stay. In contrast, the other group of physicians favours gradual weaning and removal of EVD. Here, the EVD is raised gradually keeping it open for a more extended period and over days followed by clamping and removal of the EVD [12].

Al-Tamimi et al., in LUMAS trial, which was a single-centre, controlled randomised trial, recruited around 200 patients with aneurysmal subarachnoid haemorrhage and concluded that lumbar drainage reduces the delayed cerebral ischemia but failed to improve the outcome [13].

In many retrospective studies, lumbar CSF drainage has shown to be a safe, feasible measure to remove blood from CSF. The reason to use lumbar CSF drainage in patients with aSAH is to promote CSF circulation from the ventricle and the subarachnoid space. It also removes the CSF blood from the spinal cistern and improves the CSF flow from the ventricles, thus not only decreasing the incidence of shunt-dependent hydrocephalus and reducing cerebral vasospasm [14]. But one must be careful about lumbar CSF drainage to avoid brain hernia due to excessive and frequent CSF drainage and needs strict aseptic precautions to prevent infection. Acute hydrocephalus is self-limiting, but fewer patients may require a permanent shunt and commonly ventriculoperitoneal shunt, but if the abdomen is infected or frozen then a ventriculopleural shunt cannot be performed. Approximately 1 to 45% of aSAH reported to require permanent shunt [15].

Chronic hydrocephalus and shunt requirements in aSAH had guarded prognosis and cognitive deficits with readmissions. Short- or long-term shunting has a risk of obstruction and infection, which can be life-threatening. Factors found to increase shunt requirements were increasing age, ruptured posterior circulation aneurysm, poor-grade aSAH, presence of IVH, higher modified Fisher grade, mode of treatment and female gender [11]. As for as lumbar drain in aSAH-induced hydrocephalus, we preferred intermittent lumbar drainage

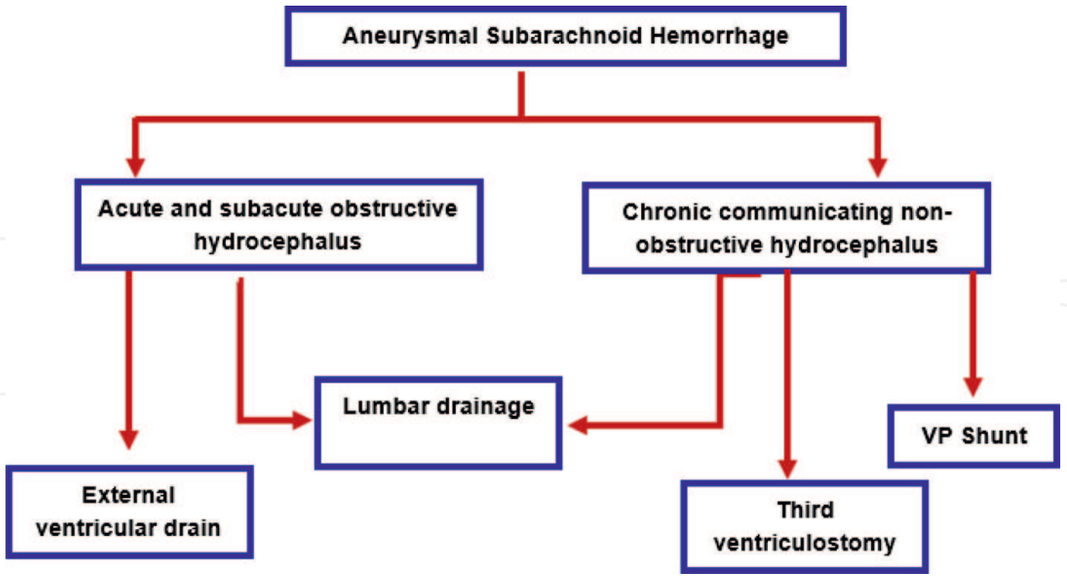

Figure 4.
Management of hydrocephalus after aSAH.

to the continuous one, as continuous drainage with lumbar catheter drain has higher risk for over drainage and leads to brain tonsillar herniation, hence unsafe.

Endoscopic third ventriculostomy (EVT) is a minimally invasive endoscopic procedure that helps to avoid VP shunt and has fewer chances of over-drainage complications and is a durable surgical option. Rarely it is infective; in general, the procedure is well tolerated. The known complications include infection and bleeding. This procedure was initially described in 1900 by *Dandy* with open and primitive endoscopy choroid plexectomy. William performed the first EVT in 1923 [16]. ETV is preferred for acute obstructive HCP, especially with mesencephalic aqueduct obstruction, as the fenestration made with ETV will clear the obstruction for CSF flow. Although Hailong et al [17] mentioned used of ETV in the communicating HCP due to other causes than aSAH, in aSAH communicating HCP main problem is oversecretion and at the same time impaired absorption; hence, ETV may not be useful. ETV also does not terminate or delay the fibrotic process of leptomeninges and subarachnoid granules, and there for not improving the CSF hemodynamics (**Figure 4**) [5].

8. Conclusion

Cerebral aneurysm rupture is a common cause of subarachnoid haemorrhage. One of the frequently occurring complications in these patients is hydrocephalus. Hydrocephalus can be acute, subacute or chronic. Acute hydrocephalus is either obstructive or non-communicating, while chronic hydrocephalus is communicating or non-obstructive. Acute hydrocephalus is commonly managed with the insertion of EVD, and fewer of these patients progress further into chronic hydrocephalus requiring shunt or long-term shunting of CSF.

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