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Review Article

SUBARACHNOID HEMORRHAGE: CURRENT OVERVIEW

***V Sardana[1], D Maheshwari[1] and R K Aseri[2]**
[1]Department of Neurology, Govt. Med. College, Kota
[2]Department of Anaesthesia, Govt. Med. College, Kota
**Author for Correspondence*

ABSTRACT
Subarachnoid hemorrhage is a devastating condition due to many etiologies, accounting for 5% of all stroke patients. Rupture of an intracranial aneurysms is most frequent causative factor. The diagnosis is usually easy as clinical symptomatology is quite classical, but diagnosis of warning leaks requires increased degree of suspicion. Non contrast computerised scan (CT) of head is initial investigation of choice, followed by cerebrospinal fluid (CSF) examination if required. Conventional cerebral angiography or 4 vessel intra arterial digital subtraction angiography (DSA) is performed in all cases for aneurysm and cerebral vasculature details. Magnetic resonance angiography (MRA) and CT angiography are increasingly available and popular modes of evaluating aneurysmal bleed but lack the sensitivity of DSA, and are frequently insufficient for surgical planning. The current trend is towards early clipping or coiling of an aneurism, choice depending on aneurysm characteristics, associated features, and centre's experience. This review discusses current knowledge about subarachnoid hemorrhage due to aneurysmal bleed

Key Words: *Subarachnoid Hemorrhage, Sah, Intracranial Aneuryms, Aneurismal Clipping, Aneurismal Coiling*

INTRODUCTION
The subarachnoid space, contained within arachnoid cistern, is a well formed fluid compartment in cranial and spinal cavity which contains and circulates cerebrospinal fluid (CSF) (Liliequist, 1995). Subarachnoid hemorrhage is a devastating condition, not a disease, caused by many etiologies.

A Subarachnoid hemorrhage (SAH) accounts for 5% of all strokes but its burden is relevant due to high mortality, high disability and a remarkable incidence in the young (Feiginet *et al.*, 2005). The annual incidence of aneurysmal subarachnoid hemorrhage (SAH) is approximately 20 per 100,000/ year in Japan, and it is suggested that approximately 40% of SAH patients have poor outcome (Fujinaka *et al.*, 2012). The incidence of SAH in the United States of America (USA) on an average has remained constant at approximately 11 per 100,000 populations, annually, while the deaths from SAH account for about 4.3 per 100,000 populations (Ingall *et al.*, 1989). There is one chance of good recovery out of five and one chance of getting crippled, three out of five die sooner or later (Ask-Upmarks *et al.*, 1989).There are very few studies in India to determine that whether incidence is similar to the developed nations or different, due to lack of exact epidemiological data and properly designed studies (Bhagwati, 1998). In a study of stroke in young from India (Chopra and Prabhakar., 1979) 7.6% of all stroke patients below 40 years of age had SAH. Regardless of the aetiology, SAH most frequently occurs between ages 40 and 60 years, with the peak frequency between 55 and 60 years of age (Locksley *et al.*, 1966).

Intracranial aneurysms are the most frequent causative factor of spontaneous SAH. The rupture of an intracranial aneurysm is responsible for about 85% of SAHs; 10% are represented by non-aneurysmal conditions; 5% are represented by other medical conditions such as inflammatory or non-inflammatory lesions of cerebral artery, coagulopathy, neoplasms or drug abuse (Venti, 2012).Other causes include arteriovenous malformations (AVM), trauma, collagen vascular disease and sickle cell anemia. We shall discuss aneurysmal SAH (aSAH), as this is the most common cause of spontaneous subarachnoid hemorrhage and make a passing reference to nonaneurysmal SAH at the end.

Aneurysmal SAH

The prevalence of incidental intracranial aneurysms at post-mortem examination is 1-6% among adults in large autopsy series (Mc Corrmick and Nofzinger., 1965; Inagawa and Hirame (1990).whereas its prevalence among adults undergoing cerebral angiography is between 0.5-1 percent (Winn *et al.*,1983; Atkins *et al.*, 1989). Asymptomatic aneurysms of various sizes, types, and locations are detected in approximately 1% to 6% of the healthy subjects who undergo brain examination (Uchino *et al.*, 1995). Prevalence of intracranial saccular aneurysms in a Japanese community based on a recent consecutive autopsy series during a 30-Year observation period was 4.6%, which is the mean level among previous many studies (Iwamoto *et al.*, 1999).

Approximately 80-85% of aneurysms are located in anterior circulation, most commonly at the junction of internal carotid artery and posterior communicating artery, anterior cerebral artery complex or bifurcation of middle cerebral artery (Fox, 1983). Table 1 shows location of single aneurisms (Yasargil, 1984). Aneurysms of posterior circulations are most commonly located at the basilar artery or the junction of a vertebral artery and the ipsilateral posterior inferior cerebellar artery (Fox, 1983; Kassell *et al.*, 1990). The Hisayama study revealed that the most frequent site of aneurysm was the middle cerebral artery, where 23 aneurysms (31.5%) occurred, followed by 22 (30.1%) at the anterior communicating artery, 11 (15.1%) at the anterior cerebral artery, 9 (12.3%) at the vertebrobasilar artery, and 8 (11.0%) at the intracranial internal carotid artery and 12 (21.3%) had multiple aneurysms, although as many as 13 aneurysms have been reported (Cedzich *et al.*, 1990).

Aneurysmal subarachnoid hemorrhage is a form of hemorrhagic stroke that affects up to 30,000 individuals per year in the United States (Zacharia *et al.*, 2010), higher than incidence of primary brain tumour and multiple sclerosis. However, little is known about the true prevalence of ruptured and unruptured aneurysms of the brain in the general population because of technical difficulties in detecting the presence of aneurysms. Only 2% of aneurysms under 5mm diameter rupture in contrast to 40% of these between 6-10 mm.

Pathogenesis

Aneurysms arising from intracranial arteries are much more common than extracranial arteries of similar size possibly because former do not have external elastic lamina and have an attenuated tunica media (Schievink, 1997). The aneurysmal wall is composed of only intima and adventitia with variable amount of fibrohyaline tissue interposed.

There are various theories of pathogenesis of aneurysms. One proposes that a congenital layer weakness in muscular layer of cerebral arteries is responsible for herniation of internal layer which destroys the elastic membrane, distending and outpouching as an aneurysmal sac. Other favour postnatal degeneration within the vessel wall leading to deterioration of internal elastic lamina and ultimately aneurysm formation. Still other postulate combination of congenital and degenerative effects (Sahs *et al.*, 1969).

Saccular intracranial aneurysms (sIA) are pouch-like pathological dilatations of intracranial arteries that develop when the cerebral artery wall becomes too weak to resist hemodynamic pressure and distends. Some sIAs remain stable over time, but in others, mural cells die, the matrix degenerates, and eventually the wall ruptures, causing life-threatening hemorrhage (Frösen *et al.*, 2012).. Current data suggest that the loss of mural cells and wall degeneration are related to impaired endothelial function and high oxidative stress, caused in part by luminal thrombosis. The aberrant flow conditions caused by sIA geometry are the likely cause of the endothelial dysfunction, which results in accumulation of cytotoxic and pro-inflammatory substances into the sIA wall, as well as thrombus formation. This may start the processes that eventually can lead to the decellularized and degenerated sIA wall that is prone to rupture (Frösen *et al.*, 2012).

Genetic and environmental factors also merit discussion. Many heritable connective tissue diseases like autosomal dominant polycystic kidney disease (Xu *et al.*, 2011). Ehlers Danlos syndrome type IV and neurofibromatosis-I are associated with intracranial aneurysms (Dohle *et al.*, 2012; Becker *et al.*, 2010). Familial aggregation of intracranial aneurysms is known and 7-20% of patients with aneurysmal SAH

have a first or second degree relative with a confirmed intracranial aneurysm. Familial aneurysms, as compared to sporadic ones, rupture at an earlier age and one more often followed by the formation of new aneurysm. The inheritance pattern is still unclear, although autosomal transmission seems likely (Goksu *et al.*, 2012; Ronkainen *et al.*, 1995).

Among environmental factors, cigarette smoking is associated with 3-10 times higher risk of aneurysmal SAH as compared to non-smokers (Longstreth *et al.*, 1992). Cigarette smoking decreases the effectiveness of Alpha-1 antitrypsin, the main inhibitor of proteolytic enzymes (proteases) such as elastase, and the imbalance between proteases and antiproteases may result in the degradation of a variety of connective tissue including the arterial wall (Schievink *et al.*, 1994). In recent studies it is found that joint effect of current smoking and hypertension on the risk of aneurysmal SAH was stronger than was the sum of the independent effects of each factor suggesting that combining smoking cessation and blood pressure lowering may have an extra risk reduction effect in preventing aneurysmal SAH (Lindekleiv *et al*., 2012).

Hypertension, the most frequently studied risk factor is associated with an increased risk of aneurysmal SAH as well as unruptured intracranial aneurysm. In one hospital-based case-control and cohort study it was seen that patients with hypertension had a nearly seven-fold higher risk of aneurysmal subarachnoid hemorrhage and substantiating the hypothesis that aneurysm is an acquired and hemodynamically induced chronic disease (Kleinpeter and Lehar 2012; Qureshi *et al*., 2011).

In recent population-based case-control study in Australasia, it was found that alcohol consumption is not a significant risk factor for causing aneurysmal SAH (Shiue *et al.*, 2012; Inagawa, 2010)but recent heavy alcohol consumption, particularly 'binge alcohol' may increase the risk of SAH (Juvela *et al.*, 1993).

Clinical Features

Most aneurysm may remain asymptomatic and some may present by mass effect. Although rupture of an aneurysm leading to SAH can occur anytime, it is most common during times of exertion or stress (Schievink *et al.*, 1989). There are anecdotal reports and reviews available suggesting that sexual activity may be important cause for rupture of intracranial aneurysm (Portunato *et al.*, 2012; Reynolds *et al.*, 2011; Bhat *et al.*, 2011). Uniquely severe headache is most common symptom in 80-90%, often accompanied by photophobia, nausea or vomiting.

The meningeal irritation is the most common sign, found in 50 % of aneurysmal SAH, but may not develop until several hours as it is caused by blood breakdown products. Neck pain, lower back pain and sometimes bilateral radicular leg pain may occur as bloody CSF flows down the spinal canal. The fundal abnormalities are also commonly seen in SAH. The 3rd nerve palsy is seen in some of the patients. The sine qua non of SAH in an awake patient is the complaint of "the worst headache of my life (Bhat *et al.*, 2011; Bassi *et al.*, 1991; Tandon, 1988; Fontanarosa, 1989). A systematic review of case series and randomized trials revealed that patient may or may not lose consciousness and seizures may occur in up to 20% of patients after SAH, most commonly in the first 24 hours and in SAH associated with intracerebral hemorrhage, hypertension, and middle cerebral and anterior communicating artery aneurysms (Raper *et al.*, 2012; Lanzino *et al.*, 2011; Claassen *et al.*, 2003).

Intracerebral, intraventricular or subdural hematoma may occur after aneurysmal rupture but rarely (1.6%) without any evidence of SAH (Thai *et al.*, 2005; Prasad *et al.*, 2009; Biesbroek *et al.*, 2012). One third to one half of patients may have prodromal or sentinel headache which is generally unusual and acute preceding the hemorrhage by several days or weeks and is considered to be a warning symptom of impending aneurysmal rupture. This warning leak is often misdiagnosed as migraine, sinusitis, influenzae or malignancy, causing crucial delay in diagnosis (Regli *et al.*, 1997; Hauerberg *et al.*, 1991).

Examination may reveal meningeal signs, unilateral or bilateral gravity dependent flat superior and convex inferior subhyaloid/vitreous hemorrhage also known as Terson syndrome on ophthalmological examination and/or focal neurological deficit. Terson syndrome is likely to occur in severe aneurysmal SAH with poor admission scores and has been suggested to indicate a worse functional outcome so an ophthalmological examination is strongly recommended in aneurysmal SAH patients with poor admission

scores (Stienen *et al.*, 2012). Oculomotor nerve palsy most commonly occurs with posterior communicating artery aneurysms but may also be seen with aneurysms of carotid bifurcation, the posterior cerebral artery, the basilar bifurcation, and the superior cerebellar artery (Güresir *et al.*, 2011). Trigeminal nerve distribution pain, though uncommon, may be seen with aneurysms within cavernous sinus (Stiebel-Kalish *et al.*, 2005). Abducent nerve palsy is usually related to increased intracranial tension but an aneurysm arising from the cavernous segment to the petrous segment of the internal carotid artery may present with it (Ikeda *et al.*, 2012).

Patients with SAH are usually assigned clinical grades. Worldwide, different scales are used to assess the clinical condition on admission after aneurysmal subarachnoid hemorrhage and two of the most universally applied scale systems are that of Hunt and Hess and one developed by the world Federation of neurological surgeons based in part of Glasgow coma scale. Table 2(Degen *et al.*, 2011)shows grading according to different rating scales. Associated severe systemic disease like diabetes, pulmonary disease and hypertension may place patient in less favourable grade.

Investigations

Noncontrast computerized tomographic (CT) scan of head should be first investigation to be performed in a suspected case. It can demonstrate magnitude and location of the hemorrhage, presence of intracerebral hematoma, can assess ventricular size and can also suggest the probable location of an aneurysm. Older studies have shown that CT scan can demonstrate the presence of a SAH in 90-95% of patients within 24 hours after the hemorrhage, after which its sensitivity decreases to 80% at 3 days, 70% at 5 days, 50% at one week and 30% at two weeks and almost nil after 3 weeks (van Gijn and van Dongen, 1982)**.** The CT scan should be carefully scrutinized because small amounts of subarachnoid blood may easily be missed. Even if CT is performed within 12 h after the hemorrhage and with a modern CT machine, studies are negative in about 2% of patients with SAH (van der *et al.*, 1995). A false-positive diagnosis of SAH on CT is possible in the presence of generalized brain oedema and cerebral venous sinus thrombosis, which causes venous congestion in the subarachnoid space and in this way may mimic SAH (Avrahami *et al.*, 1998; Kato *et al.*, 2010).

The pattern of hemorrhage often suggests the location of any underlying aneurysm like presence of blood within the supratentorial ventricular system may be due to suspected anterior communicating artery aneurysm, intracerebral hematomas one most frequently seen with suspected middle cerebral artery or distal anterior cerebral artery aneurysm, inferior frontal hematomas occur commonly with ruptured anterior communicating artery aneurysms (van Gijn and van Dongen, 1980)**.**

Lumbar puncture should only be performed when clinical suspicion is strong and diagnosis remains in question following C T scan or when the latter is not available. Although lumbar puncture carries a risk of brain herniation and aneurysmal rebleed, it can improve the diagnosis and is also helpful in ruling out meningitis. As xanthochromia is caused by breakdown of blood products which takes several hours, a lumber puncture performed very soon after SAH may fail to show the same (Vermeulen *et al.*, 1989). Xanthochromia can usually be detected 4 hours after the hemorrhage, becomes maximum one week later, and at 3 weeks mostly it is undetected. Spectrophotometry can detect xanthochromia in CSF in all patients between 12 hours and 2 weeks after the hemorrhage, in 70% after 3 weeks and in 40% after 4 weeks (Vermeulen *et al.*, 1989).

The diagnostic investigation of CT-negative subarachnoid hemorrhage is a particular challenge in clinical neurology. The diagnosis of SAH in CSF is based on a bloody or xanthochromic discoloration of the CSF as well as on findings in non-automated CSF cytology including the detection of erythrophages and siderophages. The automated determination of CSF ferritin concentrations or spectrophotometric detection of xanthochromia may contribute to the diagnosis (Tumani *et al.*, 2010).

Conventional four vessels angiography remains the 'gold standard' but 4 vessel intra arterial digital substraction angiography (DSA)or selective catheter cerebral angiography is method of choice for detecting an intracranial aneurysm and its anatomical details as the cause of SAH. The mortality associated with the procedure in experienced hands is low. In a recent review of prospective studies of

1,000 or more procedures revealed a combined transient and reversible neurologic complication rate from 0.34% to 2.3%, a permanent neurologic complication rate from 0.1% to 0.5% , and a mean overall rate of 1.6% only (Willinsky *et al.*, 2003; Dawkins *et al.*, 2007). Angiography can delineate the vessels arising adjacent to the aneurysm, can detect multiple aneurysm if present, and can assess presence and degree of vasospasm. The resolution of 4 vessels intraarterial DSA is somewhat inferior to conventional angiography but it has got an advantage of using less amount of dye. If initial angiography is normal, then it should be repeated in 5-7 days especially when it shows focal vasospasm or when whole of the cerebral vasculature is not adequately visualized. Repeat angiography after one week may show a previously unrecognized aneurysm in an additional 1% to 2% of cases (Forster *et al.*, 1978).

Helical CT angiography is now increasingly recognised as an effective diagnostic tool in evaluation of SAH patients. It can help in finding relationship of an aneurysm to bony structures and can be done in patients who already have ferromagnetic clips for earlier treated aneurysms where MRA cannot be used (Schievink, 1997).In one recent Indian study conducted at Kashmir proved that non-invasive tool like CT-angiography (CTA) detected aneurysms in around 83.42% patients and was negative in only 16.58 percent (Bhat *et al.*, 2011). In another recent well designed study of 513 patients, 106 (20.7%) had no aneurysms, while 407 patients (79.3%) had 459 aneurysms at 3 dimentional (3D) DSA. Digital subtraction CT angiography correctly depicted 456 (99.3%) of the 459 aneurysms. By using 3D DSA as the standard of reference, the sensitivity and specificity of depicting intracranial aneurysms were 97.8% and 88.7% , respectively, on a per-patient basis, and 96.5% (443 of 459) and 87.8% (94 of 107), respectively, on a per-aneurysm basis. Digital subtraction CT angiography had sensitivities of 91.3%, 94.0% ,98.4% , and 100% in depicting aneurysms of less than 3 mm, between 3 mm but less than 5 mm, between 5 mm but less than 10 mm, and 10 mm or greater, respectively, and of 95.8% and 97.7% in depicting anterior circulation and posterior circulation aneurysms, respectively (Lu *et al.*, 2012). CT Angiography is highly sensitive, specific, and accurate in detecting no spasm or severe cerebral vasospasm in proximal arterial locations but it is less accurate for detecting mild and moderate spasm in distal locations (Anderson *et al.*, 2000).

Magnetic resonance angiography (MRA), although most convenient and without risk, can miss aneurysm below 5mm in diameter. Nowadays there are claims that MRA can detect aneurysm as small as 2 or 3mm but it is definitely not sufficient for surgical planning. In one recent study patients who underwent CTA or MRA, the treatment plan was changed in 18.9% and 30.1%, respectively, based on subsequent information gleaned from DSA (Luke *et al.*, 2011). Reasons for the change in the treatment plan included size and location discrepancies, detection of a benign vascular variant rather than a true aneurysm, inadequate feeders and other vascular details. It was suggested that in "real-world" analysis of intracranial aneurysms, DSA continues to play an important role in determining the optimal management strategy (Luke *et al.*, 2011).

Management

All patients should be transferred to neurosurgical centre as soon as possible. The aim of treatment is to exclude the aneurysm sac from intracranial circulation while preserving the parent artery, minimal brain tissue dysfunction, and to remove as much subarachnoid clot as safely possible.

Preoperative management includes anticonvulsants, steroids, analgesics, antihypertensive like calcium channel blockers and supportive therapy. Treatment of hypertension, seen in approximately 10% of patients in post rupture period, is essential to reduce the risk of rebleeding because systolic arterial pressure >160 mm Hg has been shown to be a possible risk factor of rebleeding (Ohkuma *et al.*, 2001). At the same time hypertension may be reactionary due to reduction in cerebral perfusion pressure and even mild hypotension in the presence of disordered autoregulation may result in clinical worsening or infarction. One should thus, be thoughtful in treating hypertension. The response to sedation and analgesics should be assessed first and if it is decided to intervene, it is better to use short acting antihypertensive agents whose effect can be rapidly terminated.

The prophylactic anticonvulsants therapy may be considered in the immediate posthemorrhagic period but the routine long-term use of anticonvulsants is not recommended except for the patients with known risk factors for delayed seizure disorder, such as prior seizure, intracerebral hematoma, intractable hypertension, infarction, or aneurysm at the middle cerebral artery [Ukkola *et al.*, 1990 ;Cabral *et al.*, 2009).Use of antifibrinolytic agents is controversial. They have been shown to reduce the incidence of aneurysmal rebleeding when there is an unavoidable delay in aneurysm obliteration, specially a short-term use of Tranexamic acid or Aminocaproic acid during patient transfer. But there is an increased risk of deep venous thrombosis (Starke *et al.*, 2008). and these drugs are not approved by the US Food and Drug administration.

Several studies have shown benefit of Nimodipine, a calcium channel blocker in minimising or averting vasospasm (Petruk *et al.*, 1988; Seileret *et al.*, 1987; Schmid-Elsaesser *et al.*, 2006). In our opinion it should be used routinely in every patient of SAH in dosage of 60mg three times a day if there is no hypotension.

Clipping the neck of ruptured aneurysm as well as endovascular intervention by neuroradiologist are the most definitive treatments available. In last few years results of aneurysmal surgery have improved remarkably after improved introduction of operating microscope, microsurgical techniques, advanced aneurysm clips and bipolar coagulation. Surgical clipping or endovascular coiling of the ruptured aneurysm should be performed as early as feasible in the majority of patients to reduce the rate of rebleeding after aSAH and complete obliteration of the aneurysm should be done whenever possible.

For ruptured aneurysms judged to be technically amenable to both endovascular coiling and neurosurgical clipping, endovascular coiling should be considered if facility and expertise is available (Molyneux *et al.*, 2005 ; Bakker *et al.*, 2010). If possible, all the patients who undergo coiling or clipping of a ruptured aneurysm should have follow-up vascular imaging, and should be re-treated either by repeat coiling or microsurgical clipping, if there is a clinically significant residual aneurysm (Piotin *et al.*, 2010). Decision regarding nature of aneurysm treatment should be made by both experienced cerebrovascular surgeon and endovascular specialist, based on characteristics of the patient and the aneurysm (Piotin *et al.*, 2010). Microsurgical clipping is more favourable in patients with large (>50 ml) intraparenchymal hematomas and middle cerebral artery aneurysms (Rinne *et al.*, 1996). Endovascular coiling is favoured for the elderly (Karamanakos *et al.*, 2010), those presenting with poor-grade aSAH (Bracard *et al.*, 2002), and those with aneurysms of the basilar apex (Lusseveld *et al.*, 2002).

Timing of surgery has been controversial. Early surgery has an advantage of preventing much dreaded rebleeding but at the same time is technically slightly more difficult due to brain oedema and clot around the aneurysm. Most neurosurgeons now believe that early surgery is not significantly difficult technically (Kopitnic *et al.*, 1993). The incidence of vasospasm is not significantly different in early v/s late surgery but with former, aggressive treatment of vasospasm can be given (Weir *et al.*, 1982) Earlier trend was to delay definitive surgery but nowadays more and more neurosurgeons are opting for early surgery especially in grade 1-3 patients.

Complications

Rebleeding is most important complication of untreated ruptured aneurysm. The risk of rebleeding is between 4% and 13.6% within the first 24 hours and is associated with worse outcome (Kassell and Torner., 1983; Ohkuma *et al.*, 2001; Cha *et al.*, 2010). It reduces to 1-2% per day for first 2 weeks (Kassel *et al.*, 1983). Factors associated with aneurysm rebleeding include longer time to aneurysm treatment, poor Hunt-Hess grading at admission, early loss of consciousness, larger aneurysm size, and possibly high systolic blood pressure >160 mm Hg (Naidech *et al.*, 2005; Yundt *et al.*, 1998). Early surgery is best way to prevent rebleeding.

Significant vasospasm occurs in approximately 30% of patients between 3-15 days after SAH with peak incidence around 7-10 days, it may last for days to weeks and spontaneously resolve in 21 days but there is gradual and progressive decline in neurological status once it develops (Yundt *et al.*, 1998 ; Takeuchi et al., 1991).Its occurrence correlates well with amount and distribution of blood on CT scan. Diffuse

cerebral ischemia especially that associated with arterial vasospasm remains a major cause of mortality and morbidity in patients with aSAH. Hemodynamic augmentation therapy (triple-H therapy) in which hemodilution, hypervolaemia and systemic hypertension is induced, is the main stay of management (Lazaridis et al., 2010) The use of endovascular therapy with vasodilators and balloon angioplasty for large intracranial vessel vasospasm needs more evidence for efficacy and safety, can be used in patients who are not responding to hypertensive therapy (Kimball et al., 2011). Transcranial Doppler is useful, though sparingly used to monitor the development of arterial vasospasm especially for middle cerebral artery and its territories (Suarez et al., 2002).

Communicating hydrocephalus can develop in 15% to 87%, both before and after surgery or endovascular coiling, and should be managed appropriately (Little et al., 2008; Hoh et al., 2011). Acute hydrocephalus associated with poor grade aSAH is usually managed by external ventricular drainage while chronic hydrocephalus associated with aSAH is usually treated with ventricular shunt placement (Ransom et al., 2007; Jartti et al., 2008).

Both hypernatremia and hyponatremia are frequently observed in the acute phase after aSAH, the reported incidence of later ranges from 10% to 30 percent (Qureshi et al., 2002; Nakagawa et al., 2010). Hyponatremia commonly attributed either to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting syndrome (Kao et al., 2009).

Table 1: Location of single aneurysm (Yasargil, 1984)

Location	Percentage
Antrior communicating artery	37.1
Internal Carotid Artery	31.5
Middle Cerebral Artery	18.2
Basilar apex	5.0
Pericallosal	2.0
Posterior Inferior cerebellar artery	1.0

Table 2: Summary of the hunt and hess scale, the world federation of neurological surgeons scale, and the prognosis on admission of aneurysmal subarachnoid hemorrhage scale (Degen *et al.*, 2011).

Grade	Hunt and Hess Scale	WFNS Scale	PAASH Scale
I	Asymptomatic, or minimal headache and slight nuchal rigidity	GCS score 15	GCS score 15
II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy	GCS score 13–14 without focal deficit	GCS score 11–14
III	Drowsiness, confusion, or mild focal deficit	GCS score 13–14 with focal deficit	GCS score 8–10
IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity, and vegetative disturbances	GCS score 7–12	GCS score 4–7
V	Deep coma, decerebrate rigidity, moribund appearance	GCS score 3–5	GCS score 3

- *GCS: Glasgow Coma Scale*
- *PAASH: Prognosis on Admission of Aneurysmal Subarachnoid Hemorrhage*
- *WFNS: World Federation of Neurological Surgeons.*

Nonaneurysmal SAH

Approximately three fourth of patients have aneurysm responsible for spontaneous SAH. Out of remaining one fourth, 5% have arterio-venous malformations (AVMs) (Gross and Du., 2012). In 20% various other etiologies like coagulopathy, tumours, spinal AVMs, angiopathy etc. are responsible. Non aneurysmal SAH patients fare better initially and subsequently as compared to patients with ruptured aneurysms. The incidence of rebleeding is only 4% at 6 months and 0.2-0.86% per year thereafter (Nishioka et al., 1984). Eighty percent patients with negative angiography and SAH of uncertain etiology have good outcome as compared to 50% of patients with aneurysmal SAH (Brismar and Sundbärg., 1985; Fujii et al., 2006). The management is conservative and same as outlined in aneurysmal SAH.

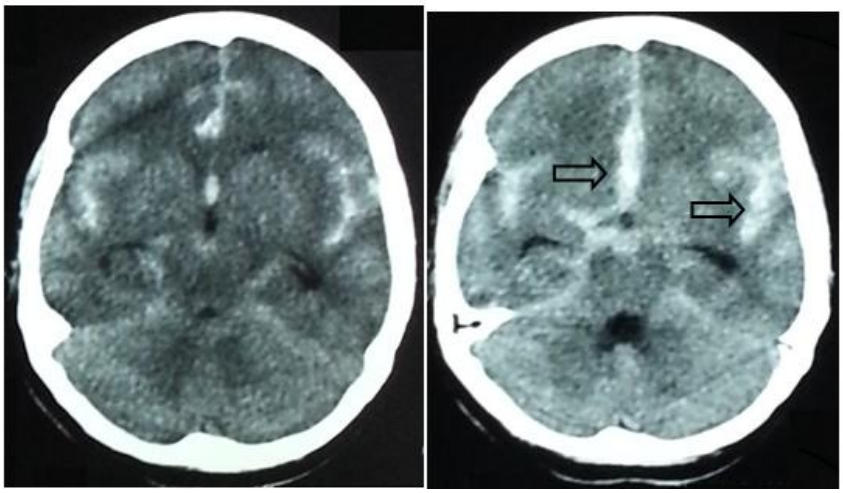

Figure 1: A 39 years old woman had severe bursting headache 'never before in her life' and followed by loss of consciousness. Her axial CT scan showing extensive subarachnoid hemorrhage involving all cisterns, bilateral sylvian fissure and frontal interhemispheric fissure.

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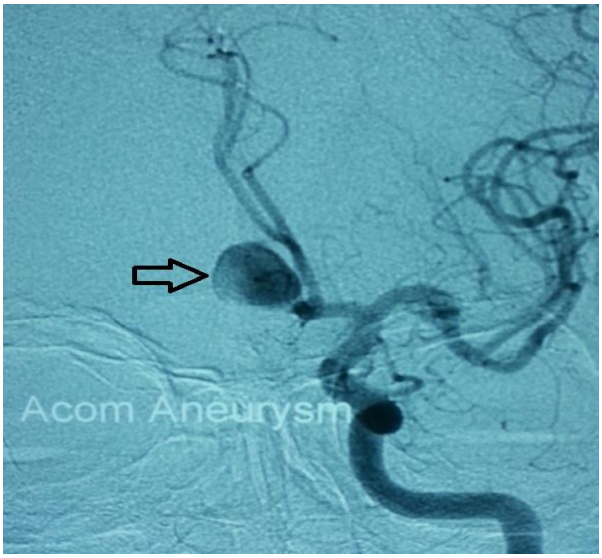

Figure 2: DSA of same patient figure1 showing large anterior communicating artery aneurysm

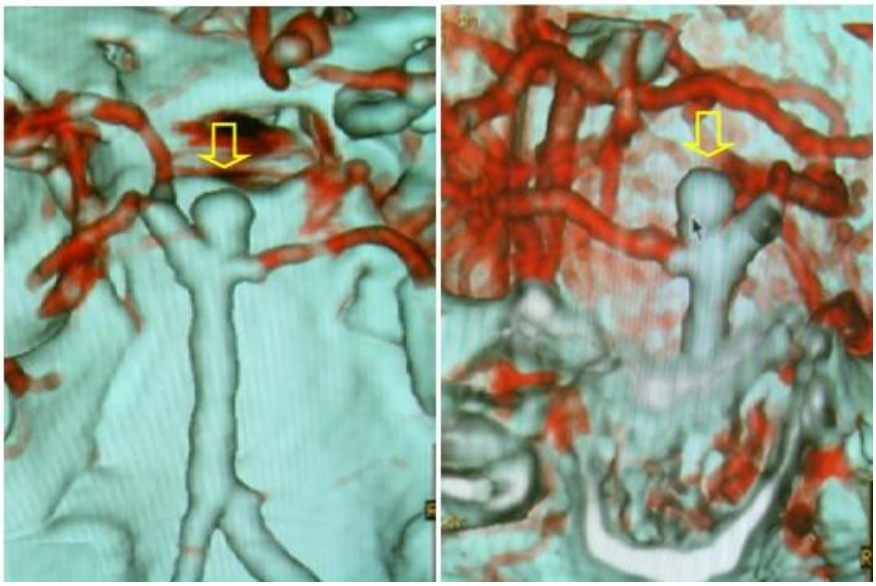

Figure 2: Another 27 years old gentleman saying "the worst headache of my life" associated with diplopia and ataxia. The 3D reconstructed images of DSA showing basilar top aneurysm.

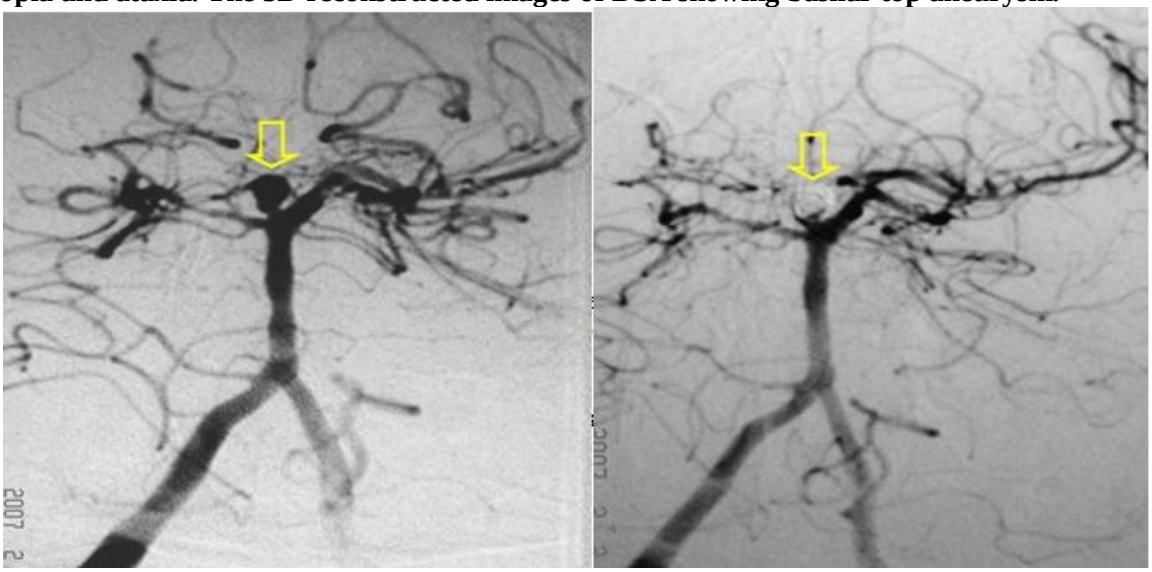

Figure 3: Before and after Embolization, DSA of the same patient showing well occluded sac of aneyrysm and patient improved remarkably without recurrence on follow up.

SUMMARY

Rupture of an intracranial aneurysm is the most common cause of sudden, unexpected and devastating SAH. The rupture usually poses no difficulty in diagnosis but for recognising preceding headaches due to warning leak, one requires increased index of suspicion. After initial CT scan, 4 vessels intraarterial digital substraction angiography is used to delineate the vessels arising adjacent to the aneurysm, can detect multiple aneurysms if present, and can assess presence and degree of vasospasm. Magnetic resonance angiography, though non-invasive and easy technique, but can miss aneurysms smaller than 5mm and may not be ideal for surgical planning. Another non-invasive tool, CT-angiography is also useful in poor grade SAH patients which can detect around 85% of aneurysms. Trans cranial Doppler is useful to monitor the arterial vasospasm especially for middle cerebral artery and its territories.

The current trend is towards early intervention. More and more centres are opting for endovascular coiling over aneurysmal clipping which is the most definitive treatment. Vasospasm following the rupture is still managed conservatively but in refractory cases endovascular therapy with vasodilators and balloon angioplasty can reasonably be considered at higher neurosurgical and neuro-intervention centres though more proof is required for its wider use.

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