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Original Research Article.

Role of Microneurosurgery for Aneurysms of Brain: A Retrospective Study

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ABSTRACT

Background: Subarachnoid hemorrhage (SAH) from ruptured aneurysms carries a poor prognosis; half the patients die within 1 month of the hemorrhage; of the patients who survive longer than 1 month,40% remain dependent. Approximately 15% of patients with aneurismal SAH die before reaching the hospital. The initial and definitive management of ruptured cerebral aneurysm is evolving, with clipping and coiling being alternative treatments.

Aim and Objective: To study the outcome of patients with aneurysmal subarachnoid hemorrhage who underwent surgery for clipping of aneurysm.

Methods and Materials: This is the retrospective study conducted from January 2009 to December 2017, 105 patients were diagnosed as spontaneous subarachnoid hemorrhage resulting from ruptured cerebral aneurysms at Dept. of Neurosurgery, Tertiary Care Hospital, Ahmedabad. We have included the patients with ruptured cerebral aneurysms only, and excluded the patients without any treatment for aneurysms or incidentally found asymptomatic unruptured aneurysms. Neurological outcome was assessed using modified Rankin's scale (mRS), and post-operative CTA. For use as the dichotomous variables, unfavorable and favorable outcomes were defined by mRS of 1-3 and 4-5, respectively.

Results: In our study, most common age group affected is 41-60 years. Aneurysms are common in female sex as male to female ratio is 1:1.76. According to the criteria of size, 84.76% patients had aneurysm size of less than 10 mm. Anterior circulation aneurysms were present in 93.33% patients and posterior circulation aneurysms were present in 6.67% patients. Vasospasm was present in 76.19% patients detected radiologically. Multiple aneurysms were present in 7.6% patients. Intraoperative rupture was happened in 10 patients (9.52%), and 5 patients had expired. Post-operative complications were faced like rebleeding (2 patients), wound infection (5 patients), CSF leaf (5 patients), and diabetes insipidus in 12 patients, infarction in 13 patients and hydrocephalus in 22 patients. According to modified Rankins Scale, 75 patients (71.42%) were under Good category (0-2 mRS), 20 patients (19.04%) were under severely disabled category (3-5 mRS) and 10 patients (9.54%) were dead (mRS 6). Out of 105 patients, 10 patients had died in post-operative period.

Keywords: Subarachnoid Haemorrhage; Microneurosurgery; Aneurysm; Outcome.

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INTRODUCTION

Subarachnoid hemorrhage(SAH) from ruptured aneurysms carries a poor prognosis; half the patients die within 1 month of the hemorrhage; of the patients who survive longer than 1 month,40% remain dependent.[1] Approximately 15% of patients with aneurismal SAH die before reaching the hospital.[2]

The initial and definitive management of ruptured cerebral aneurysm is evolving, with clipping and coiling being alternative treatments. The outcomes of these treatment modalities are changing rapidly because of the improved quality of microsurgery and the development of new endovascular techniques as well as aggressive detection and management of vasospasm and other complications related to SAH itself. When more than one procedure is performed for the same condition, then it is unlikely that one procedure is clearly the superior one. Intracranial aneurysms can be occluded using direct surgical techniques, endovascular approaches, combined surgical and endovascular procedures or indirect techniques such as revascularization procedures or parent vessel occlusion. There are two ideal and equal goals in the treatment of patients with intracranial aneurysms. The first is complete, permanent occlusion of the aneurysm sac. The second goal is optimal preservation or restoration of the patient's neurological function.[3]

The optimal treatment strategy is best handled by a team of neurovascular and endovascular surgeons to select an occlusion strategy that is suited to the patient and the aneurysm. The present study is retrospective analysis of patients with ruptured

intracranial aneurysms who were treated at Tertiary Care Hospital. Clinical outcome and associated complications were analyzed using the Modified Rankins scale to determine the need and efficacy of each procedure in these patients.

AIMS AND OBJECTIVES

To study the outcome of patients with aneurysmal subarachnoid hemorrhage who underwent surgery for clipping of aneurysm.

METHODS AND MATERIALS

This is the retrospective study conducted from January 2009 to December 2017, 105 patients were diagnosed as spontaneous subarachnoid hemorrhage resulting from ruptured cerebral aneurysms at Dept. of Neurosurgery, Tertiary Care Hospital, Ahmedabad. We have included the patients with ruptured cerebral aneurysms only, and excluded the patients without any treatment for aneurysms or incidentally found asymptomatic unruptured aneurysms.

Data Collection

The characteristics of the patients were documented, including age, sex, associated comorbidity, symptoms, Glasgow coma scale (GCS) score, Hunt and Hess Grade, features of CT scan (SAH/IVH/ICH/HCP). All patients were initially admitted to the intensive care unit. The diagnosis of cerebral aneurysms was confirmed by computed tomography angiography (CTA).Surgical treatment & approaches decided on the basis of aneurysm size, shape, location, presence or absence of mass effect due to hematoma and grading of SAH in the patients.

Surgical Technique

Surgical treatment was undertaken following the diagnosis of cerebral aneurysm, and no later than 24 h following bleeding or following admission in patients with grade 1. Different surgical approaches were used according to aneurysm location. Following the intraoperatively identification of the aneurysm, accordingly, aneurysm was isolated and clipped using titanium clips as per size of aneurysm. Temporary clips were used in some cases to prevent aneurysm rupture. At the end of the procedure, patients were monitored in the Neuro Intensive Care

Unit and nimodipine was administered for cerebral vasospasm. Patients returned to neurosurgical unit and discharged to home or rehabilitation or physiotherapy unit according to their functional disabilities.

Statistical Analysis

Morbidity and mortality related to clipping were defined as intra-procedural and/or postoperative ischemic or bleeding events. New or worsening of neurological deficits without other identifiable cause on neuroradiological exams in between days 4 to 14 post SAH were considered affected by cerebral vasospasm. Bleeding complications were defined as a new postoperative hematoma in the aneurysmal vascular area or an increasing volume of more than one-third of an intra-parenchymal hematoma in the next 12 h after surgery.

Neurological outcome was assessed using modified Rankin's scale (mRS), and post-operative CTA. For use as the dichotomous variables, unfavorable and favorable outcomes were defined by mRS of 1-3 and 4-5, respectively.

Table 1: Biographic Data

Parameters	No. of Patients	Percentage
Age Incidence		
<30 Years	08	7.6
31-40 Years	18	17.2
41-50 Years	34	32.4
51-60 Years	33	31.4
61-70 Years	11	10.5
>70 Years	01	0.9
Total	105	100.0
Sex Incidence		
Male	38	36.2
Female	67	63.8
Total	105	100.0

Table 2: Clinical Data

Parameters	No. Of Patients	Percentage
Location Of Aneuysm		
Anterior Circulation	98	93.3
Posterior Circulation	07	6.7
Total	105	100
Location Of Anterior Circulation Aneurysm		
Distal Anterior Cerebral Artery	02	2.1
Anterior Communicating Artery	29	29.6
Anterior Cerebral Artery	12	12.2
Middle Cerebral Artery	20	20.4
Internal Carotid Artery	35	35.7
Total	98	100.0

Location Of Posterior Circulation Aneurysm		
Posterior Cerebral Artery	02	28.6
Vertebral Artery	04	57.1
Basilar Artery	01	14.3
Total	07	100.0
Size Of Aneurysm		
<3 mm	04	3.8
3 – 6 mm	38	36.2
7 – 10 mm	45	42.9
11 – 25 mm	16	15.2
>25 mm	02	1.9
Total	105	100.0
Clinical Presentation (Hunt And Hess Grading Of Subarachanoid Haemorrhage)		
I	15	14.2
II	48	45.7
III	32	30.5
IV	05	4.8
V	05	4.8
Total	105	100.0
Multiplicity Of Aneurysm		
Multiple Aneurysm	08	7.6
Intra Operative Rupture Of Aneurysm		
Rupture Of Aneurysm	10	9.5
Vasospasm		
Radiological Evidence	80	76.2
Symptomatic Vasospasm	64	61.0
Complications		
Rebleeding	02	1.9
Wound Infection	05	4.8
CSF Leak / Meningitis	05	4.8
Diabetes Insipidus	12	11.2
Infarction	13	12.4
Hydrocephalus	22	21.0

DISCUSSION

The dawn of modern aneurysm surgery came in 1933 when Egas Moniz demonstrated an aneurysm by the technique of cerebral angiography, which he had discovered. The first planned intracranial operation for a saccular aneurysm was conducted by Norman Dott in 1933.He stuffed a muscle graft against an aneurysm that had ruptured intraoperatively and succeeded in stopping the bleeding and obtaining a good long term result.

Credit for the first definitive treatment of a preoperatively diagnosed intracranial aneurysm goes to Walter Dandy, who in 1937 clipped the neck of an aneurysm with a metal clip and shriveled the sac with electrocautery. Aided by the technical advances and general progress in the field of radiology, anaesthesia and intensive care, many neurosurgeons achieved progressively lower postoperative mortality rates. Preeminent among these workers are Gazi Yasargil of Zurich and Charles Drake of London, Ontario.[4] Fedor Serbinenko, a Russian neurosurgeon, first described endovascular treatment of intracranial aneurysms in the early 1970s.He used detachable latex balloon directly into the aneurysm lumen or by occluding the artery from which the aneurysm was arising; he was later followed by Romodanov and Shcheglov, Debrun, Berenstein, Higashida and Halbach et al.[5]

In 1991, Guido Guglielmi was the first to describe the technique of embolising aneurysms using an endovascular approach with electrolytically detachable platinum coils, termed Guglielmi detachable coils (GDC).

Recanalisation of aneurysms after coiling is still an issue, despite the low risk of rebleeding. The disadvantage of coiling in our country is the high cost of the material.

The majority of intracranial aneurysms are true aneurysms containing all layers or components of the normal vessel wall. In contrast, in false aneurysms or pseudoaneurysms the vascular lumen does not enlarge, although the external diameter of the abnormal segment may be increased. These aneurysms are within the skull.

They can arise as solitary (70%-75%) or multiple (25%-30%) vascular lesions, usually located at the circle of Willis.

While traumatic or infectious or tumor associated aneurysms are rare, most of them develop spontaneously. However, the pathogenesis criteria for the development of spontaneous aneurysms are only partially understood. Endogenous factors like elevated arterial blood pressure, special anatomical relationships given by the Circle of Willis, altered flow conditions, and exogenous factors like cigarette smoking, heavy alcohol consumption and anticoagulant or contraceptive medications have

been found to be associated with the occurrence of cerebral aneurysms.[6]

The most common causes for the development of an aneurysm are hemodynamically induced vascular injuries, atherosclerosis, underlying vasculopathy and high flow states. More uncommon etiologies are trauma, infection, drug abuse and neoplasms

There are three aspects of anatomy of saccular aneurysms which should be considered when planning the operative approach.[7] First, these aneurysms arise at a branching site on the parent artery. Second, saccular aneurysms arise at a turn or curve in the artery. Third, saccular aneurysms point in the direction that the blood would have gone if the curve at the aneurysm site were not present.[8]

Aneurysms are infrequently encountered on a straight non branching segment of an intracranial artery. The aneurysms occurring on straight, non-branching segments are more often found to have sacs that point longitudinally along the wall of the artery in the direction of blood flow and project only minimally above the adventitial surface. Aneurysms having these characteristics are of a dissecting type rather than of the congenital saccular type, and their development is heralded by the onset of ischemic neurological deficit than by the subarachnoid hemorrhage associated with congenital saccular aneurysms.[8]

Fusiform aneurysms are dilated, tortuous and elongated arterial segments. The term dolichoectasia describes a giant ectatic vessel of this type of aneurysms. Fusiform aneurysms are characterized by absence of a defined neck, circumferential involvement of the parent artery.[8]

(A) Internal carotid artery aneurysms (ICA)

These aneurysms arise at five sites:

1) Upper surface of the internal carotid artery at the origin of the ophthalmic artery;

2) Medial wall at the origin of the superior hupophyseal artery;

3) Posterior wall near the orogin of the posterior communicating artery;

4) Posterior wall near the origin of the anterior choroidal artery;

5) Apex of the carotic artery bifurcation into the anterior and middle cerebral arteries.[5]

(B) Middle cerebral artery aneurysms (MCA)

They arise at the level of the bifurcation or trifurcation of the artery. These aneurysms point laterally in the direction of the long axis of the prebifurcation segment of the main trunk.[8]

(C) Anterior cerebral artery aneurysms (ACA)

The most common aneurysm site on the anterior cerebral artery is at the level of the anterior communicating artery.The aneurysms arise usually at the point where the dominant proximal or precommunicating segment bifurcates at the level of anterior communicating artery to give rise to both distal segments.The direction in which the fundus points is determined by the course of the anterior cerebral arteries proximal to their junction with the anterior communicating artery.

The next most common aneurysm site on the anterior cerebral artery is at the level of origin of the callosomarginal artery from the pericallosal artery.[8]

(D) Vertebral and Basilar artery aneurysms

The basilar apex aneurysms arise at the branching of the posterior cerebral arteries from the basilar artery directing upwards. The aneurysm located at the origin of the anterior inferior cerebellar artery commonly arises from the convex side of the curve in the basilar artery and points in the direction of the long axis of the basilar segment immediately proximal to the aneurysm.

The most common aneurysm site on the vertebral artery is at the level of origin of the posterior inferior cerebellar artery. Aneurysms arising infrequently at the junction of the two vertebral arteries with the basilar artery arise on the convex side of a tortuous curve.[8]

TYPES OF INTRACRANIAL ANEURYSMS

1) Congenital

These type of aneurysms estimate as low as 8 % and as high as 20%. In the opinion of Taveras and Wood,middle cerebral artery aneurysms are most common,with posterior communicating artery aneurysms second in frequency and anterior communicating artery third. Upto 20 % of the aneurysms are multiple.[9]

2) Atherosclerotic

These aneurysms occur at the base of the brain and are most common on the basilar artery,where they may block the outlet of the third ventricle and produce hydrocephalus.They can occur on the internal carotid artery as well.The vessel involved with the atherosclerotic aneurysm is usually uniformly enlarged,somewhat irregular,tortuous and elongated.[10] These aneurysms seldom produce subarachnoid haemorrhage but more often present as masses,producing cranial nerve deficits and in some instances even mimicking extra axial brain tumours.[9]

3) Infectious

The first infectious intracranial aneurysm was described by Church in 1869 when he established a relationship between an intracranial aneurysm and infectious endocarditis.Infectious (mycotic) aneurysms represent approximately 6 % of all intracranial aneurysms.They usually inviolve peripheral branches.They may produce infectious hematoma by bleeding into the brain or subdural space. The underlying condition is often a craniofacial infection with aspergillus,phycomycetes or candida endocarditis.They may produce an infected hematoma by bleeding into either the brain or the subdural space.[11]

4) Traumatic

Traumatic aneurysms are usually secondary to blunt trauma to the skull and result when a spicule of bone in a depressed skull fracture lacerates an adjacent cortical branch of the middle cerebral artery.They may occur on the pericallosal artery where it lies adjacent to the rigid falx.[12] They may thrombose,regress,enlarge or rupture.

5) Inflammatory

Inflammatory transmural angitis in systemic lupus erythematosus, polyarteritis nodosa or giant cell arteritis cause focal fibrinoid necrosis and elastic tissue disruption. Subacute

or chronic changes usually produce ectasia and may facilitate aneurysm formation. Aneurysms in acute arteritis tend to be multiple, peripheral.

6) Neoplastic and Radaition induced Aneurysms

Neoplastic aneurysms have been reported in patients with metastatic atrial myxoma and choriocarcinoma.They usually lie peripherally in the distribution of the middle cerebral artery.

Formation of fusiform aneurysms following radiation and radioactive intrathecal gold therapy has been reported after treatment of germinoma and medulloblastoma.These aneurysms are located in the midline or parasellar region.

7) Aneurysms associated with the AV Malformations

The incidence of these aneurysms in arteriovenous malformation

is up to 25%. Approximately 50% of these aneurysms are located on a feeding artery, 25% within the nidus.[13] In general, the symptomatic lesion should be treated first, but aneurysms on feeding vessels may be at increased risk of rupture if they are not clipped before or when the arteriovenous malformation is obliterated.[14]

CHARACTERISTICS OF ANEURYSMS

1) Location of Intracranial Aneurysms

Aneurysms commonly arise at the bifurcation of major arteries and from branching sites. Most saccular aneurysms arise in the circle of Willis or the middle cerebral artery bifurcation. Approximately 86.5% of all intracranial aneurysms arise in the anterior circulation and about 10% arise in the vertebrobasilar junction. About 3.5% of aneurysms arise at miscellaneous locations like the superior cerebellar artery and the anterior inferior cerebellar artery.

Common locations include the anterior communicating artery (30%),the junction of the internal carotid artery and the posterior communicating artery (25%) and the middle cerebral artery bifurcation (20%).The internal carotid artery bifurcation (7.5%) and the pericallosal/ callosomarginal artery junction account for the remainder(4%). Around 7 % arise from basilar artery bifurcation and the remaining 3% arise at the origin of the posterior inferior cerebellar artery where it comes off the vertebral artery.

Aneurysms that arise at distal sites in the intracranial circulation are caused by trauma or infection. Non traumatic distal aneurysms, particularly along the anterior cerebral artery, have a high frequency of multiplicity and spontaneous haemorrhage.[15]

In this study, anterior circulation aneurysms were seen in 98 patients (93.33%) and posterior circulation aneurysms in 7 patients (6.67%). Among the anterior circulation aneurysms, most common location is internal carotid artery, while in posterior circulation, most common location was vertebral artery. In Shekhar LN et al study[16], 85% patients had anterior circulation aneurysms and 15% patients had posterior circulation aneurysms. In our study, one patient had duplication of anterior communicating artery with aneurysm over proximal artery.

2) FAMILIAL OCCURRENCE

The prevalence of intracranial aneurysms among first degree relatives of patients with cerebral aneurysms is higher than in the general population. The risk for a first degree relative harboring an aneurysm about three to four times higher than for someone from the general population.

Various hereditary connective tissue disorders have been associated with formation of aneurysms, most likely as a result of the weakening of the vessel wall. Intracranial aneurysms may develop in 10%-15% patients with polycystic kidney disease, an autosomal dominant disorder. Coarctation of the aorta, fibromuscular dysplasia and pheochromocytoma have been associated with intracranial aneurysms, most likely because of the elevated blood pressure.[17]

3) INCIDENCE

Aneurysmal SAH commonly occurs in the age group of 40-60 years with a peak incidence in the fifties. This is two decades older when compared to SAH due to arteriovenous malformations and one decade younger when compared with hypertensive intracranial hematoma. The average prevalence of intracranial aneurysms is around 2%.Aneurysms are approximately 1.6 times more common in women and are associated with a number of genetic conditions. Above age 40, there was an increasingly strong predominance of females, with female/male ratios of 2.74:1 and 4.16:1 for 60-69 years and 70-87 years old, respectively. The incidence of subarachnoid haemorrhage has been reported to be higher in Japan and Finland than in the United states.[17]

In this study, maximum numbers of patients were between the ages of 41 to 60 years, which is compared to Shekhar LN et al. study[16] showing increased incidence in the same age group. In our study, out of 105 patients, 38 were male and 67 were female patients with M: F ratio of 1:1.76; this is compared to Shekhar LN et al study[16] which is showing M: F ratio of 1:1.95.

4) MULTIPLE ANEURYSMS

Multiple aneurysms are relatively more common in females (74%) than in males. When two carotid aneurysms coexist, the chance of their being mirror aneurysms is three times greater than that of their both being on the same side. When an aneurysm on the anterior circulation is found, the chance of a second aneurysm existing on the posterior circulation is between 3 and 5 percents.[17]

In our study, out of 105 patients, 8 patients (7.6%) had double aneurysms detected on CT angiography but were operated for symptomatic aneurysm. Out of 8 patients, 1 patient had bilateral anterior cerebral artery (ACA) aneurysms, 2 patients had bilateral internal carotid artery (ICA) aneurysms, 3 patients had bilateral middle cerebral artery aneurysms, 1 patient had each aneurysm on basilar artery and anterior communicating artery, 1 patient had each aneurysm on internal carotid artery and anterior communicating artery. In patient with bilateral ACA aneurysms and bilateral ICA aneurysms, both aneurysms were clipped. Other patients were operated for symptomatic aneurysm. In Shekhar LN et al study[16], 3% patients had multiple aneurysms on CT angiography.

5) SIZE OF ANEURYSMS

Ruptured aneurysms tend to be larger than unruptured aneurysms and symptomatic aneurysms are larger than symptomatic aneurysms. The size at which anerysms usually begin to rupture is about 3 mm in maximum diameter and the size at which they begin to produce symptoms by means other than rupture is around 7 mm.[18]

In this study, maximum number of patients had aneurysm of size between 7-10 mm. Aneurysms with less than 3 mm diameter size were diagnosed by digital subtraction angiography. In our study 84.76% patients had aneurysm size of <10 mm which is compared to Shekhar LN et al[16] in which 82% patients had aneurysms <10mm.

CLINICAL PRESENTATION

Clinical features of Ruptured Aneurysms

The hallmark of subarachnoid haemorrhage is sudden, severe headache in about 80% cases and 45% complain of brief loss of consciousness. About 35% have nuchal rigidity. Aneurysmal rupture can lead to sudden death in about 12- 15%. Other symptoms are hamiparesis, dysphasia, extraocular muscle involvement, visual loss, visual field defect, localised haedache and seizures and sometimes giant thrombosed aneurysms may present as a mass lesions. Prior to frank bleed, many patients experience warning symptoms, the so-called “sentinel symptoms”.

These are reported to occur in as high as 60%-70%. These consist of headache, retrobulbar pain and nuchal pain.

Ruptured aneurysms at specific sites may produce distinct clinical features. Anterior cerebral artery aneurysm rupture may produce brief weakness of the lower limbs bilaterally. Middle cerebral artery aneurysm may produce hemiparesis, paraesthesias, hemianopsia and dysphasia. Seizures occur more commonly with anterior circulation aneurysms and more so with the middle cerebral artery aneurysms. Thirs nerve palsy or unilateral retro orbital pain suggests an aneurysm arising at the internal carotid artery-posterior communicating artery junction. Third nerve involvement can occur also with superior cerebellar artery aneurysms. Carotico-ophthalmic aneurysms and superior hypophyseal aneurysms may produce unilateral vision loss or even bilateral visual defects. In about 25% of patients with SAH there may be vitreous haemorrhage.[15]

In our study, 48 patients (45.70%) were presented with grade II Hess and Hunt scale and 32 patients (30.48%) were presented with grade III Hess and Hunt scale. These patients were operated within 72 hours of admission and had good outcome compared to the patients with grade IV and V scale. The patients with grade IV and V were operated within one week of admission and out of 10 patients, 4 patients were died after operation.

PATHOPHYSIOLOGY OF ANEURYSM RUPTURE

There may be a small number of SAH presenting as 'warning leak" or sentinel haemorrhage, usually only associated with a sudden severe headache. In general, there is a correlation between the extent of SAH and the clinical grade, incidence of vasospasm and other complications such as cerebral ischemia, increased intracranial pressure and hydrocephalus. With increased severity of SAH there are increasing changes in physiologic parameters such as reduced cerebral blood flow, hypovolemia, hypermetabolism and cardiac arrhythmia.

PATHOLOGIC SEQUELAE OF ANEURYSMAL RUPTURE

A. Immediate

1. Haemorrhage
    a. Subarachnoid
    b. Intracerebral
    c. Subdural
    d. Intraventricular
    e. Intra aneurysmal
2. Acute brain swelling
3. Acute ventricular dilatation
4. Brain shifts
    a. Tentorial herniation
    b. Tonsilar herniation

B. Delayed

1. Haemorrhage
    a. Repeat aneurysmal rupture
    b. Secondary midbrain haemorrhages
2. Chronic hydrocephalus
3. Arterial vasospasm
4. Brain infarction
    a. Direct pressure from space occupying lesion
    b. Vessel compression from brain shift
    c. Severe diffuse vasospasm
    d. Systemic hypotension, decreased cardiac output, decreased red cell mass and blood volume, hypoxia, acidosis, other fluid and electrolyte abnormalities.[17]

In this study, intraoperative rupture of aneurysm was found in 10 cases (9.52%).The patients with intraoperative rupture of aneurysm were managed with temporary occlusion of internal carotid artery and post-operative ventilatory support. In 3 patients with ICA aneurysm,ligation of internal carotid artery in neck was done out of which 1 patient had mortality.In other patients, clipping was done successfully after temporary occlusion of ICA and out of 7 patients, 4 patients had mortality. In Shekhar LN et al study[16], intraoperative rupture of aneurysm was found in 3% patients.

Complications of subarachnoid haemorrhage

1. Hydrocephalus
2. Rebleeding from aneurysm rupture
3. Cerebral vasospasm with ischemia

1) Hydrocephalus

Acute hydrocephalus within the first 24 hours of haemorrhage may develop due to blood within the basal cisterns or in the ventricular system causing CSF obstruction. If confirmed by CT, early ventricular drainage is indicated and can dramatically improve the clinical status of the patient. However, caution during placement of a ventricular drain is important, since sudden drainage may precede aneurysm re-rupture, mainly because the transmural pressure along the aneurysm wall may exceed the intraventricular pressure. Hydrocephalus may develop over days or weeks following SAH, clinically often presenting with gait disturbance, impaired intellectual function and progressive lethargy. The possibility to eliminate major parts of the subarachnoid blood by intraoperative lavage and thereby decreasing the incidence of vasospasm and hydrocephalus is widely considered as an advantage of the neurosurgical approach compared to the endovascular route.

2) Rebleeding

Rebleeding is a frequent and sometimes devastating neurologic complication of SAH and is postulated to be due to breakdown of perianeurysmal clot. Early bleeding in the first hours after admission for the initial haemorrhage with clinical deterioration occurs in up to 18% of patients.[19] Since these early rebleedings commonly occur before the first CT scan is obtained, the true frequency of early rebleeding definitely underestimated. Clinically, recurrent haemorrhage may present with new neurologic deficits, increasing headache, vomiting and a depressed level of consciousness. Seizures might occue as a result, but not as the cause of bleeding. Clot formation and tissue damage stimulate fibrinolytic activity in the CSF, increasing the potential risk of rebleeding. Intracerebral hematoma occurs in up to 30% of patients with aneurysmal rupture.

3) Vasospasm

This is a major cause of morbidity and mortality in patients after SAH and is often associated with delayed cerebral ischemia. It generally involves medium and large size cerebral arteries and usually occurs between the 4th day and 14th day after SAH. It is maximal during day 6-8th and spontaneously resolves by the 12-14th day and rarely may be sustained over 2 weeks. Angiographic spasm has a typical temporal course, with onset at 3 to 5 days after the haemorrhage, maximal narrowing at 5 to 14 days, and gradual resolution over 2 to 4 weeks.[20] Three phases in the evolution of chronic vasospasm were proposed by Kapp et al.[1] an initial muscular contraction,[2] secondary injury to the arterial wall due to injury to internal elastic lamina,[3] cascade of repair.

Vasospasm according to its severity:

Grade I: The vessel still has 50% of luminal flow.

Grade II: There is more than 50% of reduction of the lumen.

Grade III: The vessels are barely visible on angiogram.[20]

The delayed ischemic neurological deficit associated with symptomatic vasospasm usually presents shortly after the onset of angiographic vasospasm with the acute or subacute development of focal or generalized signs and symptoms.

For lesions affecting the anterior circulation, these signs include hemiparesis, hemisensory deficits, visual disturbance or dysphasia or change in level of consciousness.

For vasospasm of the posterior circulation, manifestations may include dysarthria, diplopia, vertigo, ataxia or altered sensorium.

Progression to permanent cerebral infarction occurs in approximately 50 percent of symptomatic cases; recovery without deficit in the remaining individuals may occur despite the persistence of angiographic vasospasm.

The Cooperative aneurysm study in 1987 reported the incidence of angiographic vasospasm at over 50 percent, with symptomatic vasospasm in 32 percent of patients. Recent uncontrolled retrospective studies utilizing intravenous calcium-channel antagonists have reported the incidence of symptomatic vasospasm lower than 10 percent.

Predisposing factors are increased volume of SAH on CT scan, worse clinical grade, intraventricular blood and hydrocephalus, fever, hyponatremia and antifibrinolytic agents.

Therapies to treat vasospasm are vasodilating agents, hypertension, hypervolemia, hemodilution, calcium channel antagonists or transluminal angioplasty.[21]

In our study, vasospasm was found radiologically in 80 patients (76.19%) out of 105 patients. Out of 64 patients with symptomatic vasospasm, 13 patients had developed infarct in the territory of involved vessel during postoperative period and operated for decompression craniectomy. In Shekhar LN et al study[16], vasospasm was seen in 65.7% cases.

TREATMENT OF RUPTURED INTRACRANIAL ANEURYSMS

Intracranial aneurysms can be occluded using direct surgical techniques, endovascular approaches, combined surgical and endovascular procedures or indirect techniques such as revascularization procedures or parent vessel occlusion. Microsurgical techniques focus on exclusion of the aneurysm from the cerebral circulation and reduction of mass effects on adjacent structures. Various approaches have been developed and tailored to the anatomy and location of the aneurysm. A surgical clip usually is placed across the aneurysm neck with preservation of the parent vessel, eliminating any aneurysmal rests that may redevelop subsequently. Alternative surgical techniques involve proximal or Hunterian ligation, wrapping of the aneurysm or trapping (a combination of proximal and distal vessel occlusion). Adjunctive measures have been developed to reduce operative morbidity and to provide cerebral protection. Aneurysmal rupture, the principal surgical complication, may be avoided with induced hypotension, CSF drainage, diuretics, hyperventilation and use of minimal brain retraction.

CONSIDERATIONS IN TIMING OF ANEURYSM SURGERY

1. Propensity for rehaemorrhage
2. Influence of surgery on vasospasm
3. Likelihood of patient developing vasospasm
4. Effect of surgery on hemodynamically compromised brain
5. Patient's clinical grade
6. Amount of subarachnoid blood on CT scan
7. Patient's age
8. Concurrent medical problems
9. "Tightness" of brain
10. Planned operative approach
11. Anatomy of aneurysm
12. Presence of intraparenchymal haemorrhage
13. Expertise of operating team
14. Timing of patient referral[22]

THEORETICAL ADVANTAGES OF EARLY SURGERY

- Eliminates chance of rehaemorrhage
- Removes clots, possibly decreasing incidence of vasospasm
- Allows hypertensive and endovascular treatment of vasospasm
- Prevents complications of bed rest
- Soft clot easier to dissect
- Shortens hospital stay[22]

THEORETICAL ADVANTAGES OF DELAYED SURGERY

- Improved hemodynamic status of brain
- Brain slack
- Proven excellent surgical results[22]

ANATOMIC PRINCIPLES DIRECTING SURGERY

1. The parent artery should be exposed proximal to the aneurysm. This allows control of the flow to the aneurysm if it ruptures during dissection.
2. If possible, the side of the parent vessel opposite the side on which the aneurysm arises should be exposed before dissecting the neck of the aneurysm. The dissection then be carried around the side of the parent vessel to the origin of the aneurysm.
3. The aneurysmal neck should be dissected before the fundus. The neck is the area that can tolerate the greatest manipulation, has the least tendency to rupture,and is to be clipped. The dissection should not be started at the dome because this is the area most likely to rupture before or during surgery.
4. All perforating arterial branches should be separated from the aneurysmal neck prior to passing the clip around the aneurysm. The use of magnification has permitted increased accuracy of dissection of the aneurysmal neck and more frequent preservation of the recurrent and other perforating arteries.[23-26]
5. If rupture occurs during microdissection, bleeding should be controlled by applying small cotton pledge to the bleeding point and concomitantly reducing mean arterial pressure. If this technique does not stop the haemorrhage, a temporary clip can be applied to the proximal blood supply, but only for a brief time.
6. The bone flap should be placed as low as possible to minimize the retraction of the brain. Most aneurysms are located on or near the circle of willis under the central portion of the brain. A low flap minimizes the amount of brain retraction to reach these areas.
7. A clip with a spring mechanism that allows it to be removed, repositioned and reapplied should be used.

8. After the clip is applied, the area should be inspected to make certain the clip does not kink or obstruct a major vessel and that no perforating branches are included in it.
9. If an aneurysm has broad based neck that will not accept the clip easily,the neck may be reduced by bipolar coagulation.[8]

OPERATIVE APPROACHES

The frontotemporal craniotomy with slight modification is suitable for approaching all aneurysms from the anterior circle of Willis and for some originating from upper part of the basilar artery.

Most basilar artery aneurysms are approached through a subtemporal craniotomy. If basilar artery aneurysms are approached through frontotemporal craniotomy there are 4 routes: basilar apex exposure through the optico carotid triangle, separation of frontal and temporal lobes through the sylvian fissure, interval between the carotid bifurcation and the optic tract, behind the carotid artery and above the posterior cerebral artery through the interval between the carotid artery and the oculomotor nerve.

The subtemporal approach when combined with sectioning of the tentorium cerebelli posterior to the trochlear nerve, can be used to reach the aneurysms arising from the junction of the basilar artery and the superior cerebellar artery.

Aneurysms arising from the vertebrobasilar junction are approached through a subtemporal exposure if the junction is high, through a combined supra-and infratentorial presigmoid exposure if the junction is deep in the mid part of the posterior fossa ,or through a suboccipital craniectomy if the vertebrobasilar junction is low.[8]

SURGICAL TECHNIQUE OF MICROSURGICAL CLIPPING

After induction of anaesthesia, blood pressure is maintained between 100 to 110 mmhg. After aneurysm clipping, blood pressure should be maintained at the higher end of the patient's normal range to maximize cerebral perfusion. An infusion of 20% mannitol is begun at the time of the skin incision. This results in a slack brain even before the dura is opened. The final step to achieve a slack brain is to maintain a PCO_2 in the range of 25 to 30 mmhg before the dura is opened; thereafter pCo_2 is kept between 30 and 35 mmhg. When the dura is opened,it is easier to open the leaves of the arachnoid in the sylvian fissure if there is some CSF present. The first step is to identify the optic nerve. Then the exposure is maintained with self-retaining retractor. Then the microscope is taken with 300 mm objective lens. This is particularly important when the arachnoid is being dissected from the neck of aneurysm.

The first step of microdissection is to divide various arachnoid connections. This frees the aneurysm from any undue traction and increases the room in which to operate within the subarachnoid space. The arachnoid between the frontal lobe and optic nerve is divided first. This plane is extended laterally into the medial portion of the sylvian fissure. The next step is to obtain control of the carotid artery, this is particularly important for carotid artery aneurysms. The best place to begin this portion of dissection is between the optic nerve and the artery. Once this arachnoid is opened, the dissection can proceed along the carotid to the point of origin of the aneurysm. With aneurysms of the carotid artery at the posterior cerebral artery and anterior choroidal artery, one can usually obtain proximal control of intracranial carotid arterty. In some situations the PCA aneurysm may be quite proximal or the aneurysm may be partially covered by the anterior clinoid process, in these situations control of carotid artery can only be obtained in the neck.[27]

ANEURYSM CLIPS

In 1911, Cushing described the use of a malleable silver vascular clip. In

1950, spring clips were developed by Schwartz and Mayfield. In 1980s MRI compatible clips were developed. Since the early 1990s, four major brands of aneurysm clips are being used:

The McFadden Vari-angle clips, Sundt slim-line clips, Sugita clips, Yasargil clips.

CLASSIFICATION OF ANEURYSM CLIPS

Dujovny and Kossowsky pioneered the classification. Clips are divided into three classes based on mechanical principles: Alpha clips, Pivot clips and Mobile fulcrum clips.

It is important to remember that the length of the clip blade must exceed the diameter of the aneurysm neck because the collapsed neck will be longer than the distended diameter.[28]

TECHNIQUES FOR LARGE ANEURYSMS

Several problems that occur with aneurysms of 2 cm diameter or larger must be recognized before the clip application is attempted. Special attention must be given to the selection and application of clip. Sir Yasargil has advocated using bipolar coagulation of the aneurysm neck to reduce its size and permit safer clipping.In these cases large clip can be applied or more than one clip ,usually from different directions, to obliterate some of the larger aneurysms can be applied.

Another technique is suction decompression. A 21 gauge scalp vein needle with the flanges removed is connected to suction machine. By puncturing the aneurysm, blood can be suctioned through the aneurysm and the intraluminal tension reduced.

Another method is suction through the carotid artery in the neck with the use of a flexible catheter inserted into the internal carotid artery.It is important to place a temporary clip distal to the aneurysm to avoid suctioning blood away from the brain.[27]

PROBLEMS WHILE CLIPPING LARGE ANEURYSMS

1. Wall of the neck in large aneurysm is thick which may cause the clip to slip proximally and compromise the parent vessel.
2. Tension within the aneurysm prevents the clip from closing completely and increases chances of rupture. Temporary occlusion of internal carotid artery in the neck dramatically reduces the pressure in the supraclinoid carotid artery and within the aneurysm.[27]

INTRAOPERATIVE ANEURYSM RUPTURE

One of the most serious intraoperative complications of all aneurysm surgery is intraoperative rupture. This is particularly difficult to manage when it occurs during the initial exposure. The surgeon should be alert to this possibility in those patients with aneurysms of the posterior cerebral artery who do not have a third nerve palsy. This may indicate that the aneurysm is adherent to the temporal lobe rather than having the usual close relationship with the third nerve. This should also be suspected when the angiogram shows the aneurysm projecting lateral to the carotid artery. In these situations special attention to the placement of the temporal retraction is necessary. The first step in the case of a rupture is to maintain visualization of the operative field. This can

often be accomplished placement of suction over the dome. A temporary clip may be helpful if additional time is needed to complete the final delineation of the neck of the aneurysm as well as the branches of the carotid artery.[27]

POSTOPERATIVE MANAGEMENT

The goals of postoperative care:

- To maintain adequate cerebral perfusion
- To reduce any postoperative cerebral swelling and increase in intracranial pressure
- To prevent the occurrence of the seizures.

In the post-operative period, vasospasm and development of hydrocephalus are the main causes of neurological worsening in addition to other causes such as electrolyte imbalance. Post-operatively patients are assessed neurologically, and with CT scan. Patients at risk of developing vasospasm are monitored closely in the intensive care unit. All patients are given nimodipine, starting within 4 days of SAH and continued for 21 days regardless of admission grade. There is controversy regarding this and many surgeons do not give nimodipine. The usual dose for an adult is 60 mg 4th hourly.[29,30]

Neurological status is watched carefully. The main stay of medical treatment is triple H therapy where volume expansion is achieved with packed blood cells, and colloids or hypertonic saline solution.[31] As per the literature and the author's experience starch solutions should be avoided due to risk of coagulopathy. A central venous line is inserted for aggressive invasive monitoring. A central venous pressure of greater than 8 mmHg or pulmonary wedge pressure greater than 14 mmHg is usually enough to dilute the haematocrit to less than 35%. An adequate level of haemoglobin should also be maintained. This volume expansion maintains the blood pressure at the desired level.In case the situation demands it; the systolic blood pressure can be raised to 180-200 mmhg with the help of vasopressors.[32]

Hydrocephalus is a common complication with SAH due to A.Com aneurysms, especially when there is a significant intraventricular bleed. More than half of the patients may require either external ventricular drainage or a permanent ventriculoperitoneal shunt.[33] Fenestration of the lamina terminalis at the time of surgery appears to reduce the need for ventriculoperitoneal shunting.

Patients with anterior communicating artery aneurysms are susceptible to electrolyte abnormalities of which hyponatraemia is frequently seen (incidence 18%) lasting 1–5 days after surgery.[34] This is treated with normal saline infusion, supplemental salt intake and if need be with hypertonic saline infusion.[35]

In our study, the most common complication was hydrocephalus seen in 22 patients and managed with external ventricular drainage or frequent therapeutic lumbar puncture or ventriculoperitoneal shunt. Other complications were infarction followed by diabetes insipidus, CSF leak and wound infection. Patients with CSF leak were managed with therapeutic lumbar puncture for 3 days and higher antibiotics according to culture and sensitivity report. Rebleeding was observed in 2 patients.

In our study, outcome was measured by modified Rankins Scale (mRS). Out of 105 patients, 75 patients (71.42%) were under Good category (0-2 mRS), 20 patients (19.04%) were under severely disabled category (3-5 mRS) and 10 patients (9.54%) were dead (mRS 6). In Shekhar LN et al study[16], 69 % cases were under good category, 22.35% cases were under severely disabled category and 8.65% cases were dead.

Table 3: Comparison of Our Study to Standard Study

Criteria	Our study	Shekhar LN et al (AJNR 2008)
Age	41-60 years	41-60 years
Sex	M:F 1:1.76	M:F 1:1.95
Size(<10 mm)	84.76%	82%
Location (Ant. VS Post.)	93.33% VS 6.67%	85% VS 15%
Vasospasm	76.19%	65.7%
Multiplicity	7.6%	3%
Intraoperative rupture	9.52%	3%
Mortality	9.54%	8.6%

CONCLUSION

- This is the retrospective study conducted from January 2009 to December 2017, 105 patients were diagnosed as spontaneous subarachnoid hemorrhage resulting from ruptured cerebral aneurysms.
- Most common age group affected is 41-60 years.
- Aneurysms are common in female sex as male to female ratio is 1:1.76.
- 84.76% patients had aneurysm size of less than 10 mm.
- Anterior circulation aneurysms were present in 93.33% patients and posterior circulation aneurysms were present in 6.67% patients.
- Vasospasm was present in 76.19% patients detected radiologically.
- Multiple aneurysms were present in 7.6% patients.
- Intraoperative rupture was happened in 10 patients (9.52%), and 5 patients had expired.
- Post-operative complications were faced like rebleeding (2 patients), wound infection (5 patients), CSF leaf (5 patients), and diabetes insipidus in 12 patients, infarction in 13 patients and hydrocephalus in 22 patients.
- According to modified Rankins Scale, 75 patients (71.42%) were under Good category (0-2 mRS), 20 patients (19.04%) were under severely disabled category (3-5 mRS) and 10 patients (9.54%) were dead (mRS 6).
- Out of 105 patients, 10 patients had died in post-operative period.

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