A RETROSPECTIVE STUDY OF POSTERIOR FOSSA ARTERIOVENOUS MALFORMATIONS: MANAGEMENT AND OUTCOME

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Certificate

This is to certify that the study related to the thesis entitled **"A RETROSPECTIVE STUDY OF POSTERIOR FOSSA ARTERIOVENOUS MALFORMATIONS - MANAGEMENT AND OUTCOME"** is a bonafide work of ***Dr. Amitabh Gupta***, conducted in Department of Neurosurgery , Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram, under our supervision and guidance.

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DR. AMITABH GUPTA

CONTENTS

Introduction

INTRODUCTION

Arteriovenous malformations (AVMs) of the central nervous system are uncommon lesions with an estimated incidence of approximately 3 per 100,000 population. The prevalence of these lesions is difficult to estimate because many are asymptomatic for long periods of time before they become symptomatic[1] Vascular malformations including arteriovenous, cavernous, venous, and dural malformations of the posterior fossa are rare, and limited experience exists regarding their presentation, evaluation, and treatment.[2]

In the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage, only 38 of 545 patients (7%) had arteriovenous malformations (AVMs) that localized to the posterior fossa.[3] Other studies involving fewer patients, the reported frequencies range from 10% to 20%.[3–6] Due to the rare nature of these abnormal vascular structures, not only is there a lack of understanding of their natural history but surgical as well as interventional experience is generally limited. Results of treatment in the published series indicate a rate of death and disability about twice that for the general series of supratentorial AVMs.[3,4] Approximately half of all vascular malformations found within the cerebellum are AVMs. Cerebellar AVMs have a distinctly different natural history and clinical presentation compared with similarly sized AVMs involving the brainstem. AVMs within the posterior fossa typically involve either the cerebellum or the brainstem, but involvement of both is extremely rare

Despite technical breakthroughs in microneurosurgery and despite the development of radiological diagnosis in the more than 100 years since the first reported surgical procedure for treatment of infratentorial arteriovenous malformations of the brain, the therapy for these lesions, perhaps more than that for any other neurosurgical disease, demands the synthesis of all of the neurosurgeon's skills. In fact, treatment today is often rendered as a combination of the three principal therapeutic modalities: microsurgery, endovascular embolization, and radiosurgery.[3,7] The difficulty these lesions pose from an operative standpoint relates to the necessity for meticulous preservation of the innumerable critical neurovascular structures housed within and around the brainstem and cerebellum, the cranial nerves, and the deep nuclei.[8,9,10] Astute judgment, microsurgical proficiency, and clinical expertise are required of any surgeon contemplating excision of a given arteriovenous malformation. The modern neurosurgical literature is rife with reports of surgical misadventures and poor outcomes in the treatment of arteriovenous malformations.

Aim

AIM

To define the natural history, clinical features, treatment, and outcome of arteriovenous malformations of the posterior fossa.

Materials and Methods

MATERIALS AND METHODS

We retrospectively reviewed the charts of all patients admitted to Sree Chitra Tirunal Institute for Medical Sciences and Technology with a radiologically verified diagnosis of posterior circulation vascular malformation from 1990 through February 2006. The Digital Subtraction Angiography (DSA) of all these patients with AVM's were analyzed in depth with help of neuroradiologist and graded at admission according to the Spetzler-Martin scale[11]. In addition other data on the size, location, arterial supply, and venous drainage of the lesions, were determined from the available radiological studies. Surgical outcome was assessed by the Glasgow Outcome Scale (GOS)[12] at the time of hospital discharge and during the follow-up.

Glasgow Outcome Scale (GOS)[12]

	GRADE
Good recovery (GR - normal or minor deficits)	5
Moderate disability (MD - independent)	4
Severe disability (SD - dependent)	3
Vegetative state (VS)	2
Death (D)	1

These vascular malformations were accessed by routine microsurgical techniques and were approached either by retromastoid suboccipital or midline suboccipital craniectomy with patient in lateral or supine position. In one patient combined approach was used. Other standard perioperative measures included cerebral protection utilizing hypothermia and the avoidance of hypotension were used frequently.

A total of 40 patients were found to have DSA proven posterior fossa AVM and out of these 17 patients underwent treatment either in the form of surgery or embolization or radiosurgery or combination of above mentioned procedures and were included for the detailed analysis. Though the rest of the 24 patients were also advised treatment, no specific treatment was offered to them due to financial or socioeconomic constraints and were lost to follow-up, hence analysis could be made only of there demographics, presenting features, location, Spetzler Martin Grading[11] of AVM.

We further analyzed our angiographic data utilizing Chi-square test and multivariate logistic regression model for testing significance of various parameters of AVMs including size, number of feeders, venous drainage and SM grade etc. to predict for patients who were at higher risk of hemorrhage.

Review of literature

REVIEW OF LITERATURE

Cerebrovascular malformations can be divided into five distinct categories on the basis of pathological studies: Arteriovenous malformations (AVMs); capillary telengectasias; cavernous malformations; venous angiomas; and cerebral varices. Arteriovenous malformations are vascular abnormalities consisting of fistulous connections of arteries and veins without normal intervening capillary beds. Typically they are triangular, with base towards the meninges and apex towards ventricular system.[13] Arteriovenous malformations were only recognized as a distinct clinical entity in 1906, and only in 1932 a report of successful resection by Olivecrona and Rives appeared in literature.[1]

Posterior fossa AVMs supplied by the vertebrobasilar system have traditionally been regarded as rare lesions, originally thought to comprise 5 to 7% of all intracranial AVMs.[14,15] However, advances in neuroimaging and improvements in the understanding of natural history have led to an increase in the diagnosis of such lesions, with more recent clinical series reporting an incidence of approximately 10 to 18%.[16,17,18,19] This higher figure is corroborated by a 25% incidence found in the autopsy literature.[20] Posterior fossa AVMs can primarily involve the cerebellum (hemisphere and vermis), the brainstem (midbrain, pons, and medulla), or involve both structures in a combined manner (including the cerebellopontine angle) The cerebellum seems to be the most frequently involved structure, with several series reporting 60 to 75% of posterior fossa AVMs occurring in this location.[14,17,19] The remainder of posterior fossa AVMs can be evenly divided between primary involvement of the brainstem or involvement of the brainstem in combination with the cerebellum.[11,17] Unlike supratentorial AVMs, 72 to 92% of patients harboring infratentorial AVMs present with at least one episode of bleeding.[14,16,17,19]

EPIDEMIOLOGY[22]

Exact incidence of AVMs is unknown. Large autopsy series estimate the frequency of AVM detection to be 1.4% to 4.3 %.[23]In the only population based study, the sex- and age- adjusted incidence rate was 1.11 per 100,000 persons.[24]AVMs are most frequently detected vascular fornmation,[25] accounting for 2% of all strokes[26,27] and 38% of all intracranial hemorrhages in patients between 15 and 45 years.[28] AVMs are one seventh as common as aneurysms and the prevalence has been estimated as 0.2% to 0.8% of general population.[24,29,30,31] The prevalence may be slightly higher in the Asian population.[32] Patients typically present between 20 and 40 years of age.[33-51] Most studies report an equal gender predilection[27,40] or a slight male predominance among patients presenting with AVMs.[33-36,44] Although AVMs are typically solitary, multiple AVMs have rarely been described.[48,49] A cooperative study of intracranial aneurysms and subarachnoid hemorrhage (SAH) reported a less than 1% incidence of multiple AVMs.[27] Willinsky and coworkers,[49] however, reported an incidence of 9% in 203 consecutive patients. The majority of AVMs are located supratentorially.[27] Less common sites include the cerebellum, the brainstem and within the ventricle.

In the posterior fossa cerebellum is the most common site.[47] Infratentorial AVMs were first reported as a clinical entity by Clingenstein[52] in 1908, although the first removal of a cerebellar AVM was not accomplished successfully until 1932 by Olivecrona.[53] Posterior fossa AVMs account for 10% to 20% of all intracranial intraparenchymal

AVMs.[3,4,52,53] In the Cooperative study of Intracranial aneurysms and SAH, 32(7%) of 453 AVMs were located in the posterior fossa, and in Drake's series of 600 AVMs 116 (20%) were located in posterior fossa.[4,27] Stoodly et.al[54] treated 53 patients of posterior fossa AVMs at Stanford Medical Center from 1985 to 1998. Patients ranged in age from 5 to 64 years (29±14).

CLINICAL PRESENTATION

The majority of patients with AVMs present with intracranial hemorrhage and symptoms related to elevated intracranial pressure. Among these patients, headache, nausea, vomiting, and dizziness were the most common symptoms. While few of them having no evidence of hemorrhage on computed tomography (CT) scans present with neurologic deficits.

Asymptomatic: In one large autopsy series, only 12% of patients harboring an AVM had symptoms related to it.[23] Although exact number of asymptomatic persons are unknown, clinical studies reported that 2% to 4% of detected AVMs are incidental findings.[38,55,56] In a population-based study of patients with intracranial vascular malformations, 40% were asymptomatic.[24] Given that up to 50% of cerebellar and 80% of brainstem lesions can be asymptomatic,[57] however, it is unlikely that many of the relatively common and nonspecific symptoms are caused by posterior fossa AVMs.

Hemorrhage: AVMs most commonly present with hemorrhage. In a population based study, 65% of patients with AVMs presented with hemorrhage, with peak occurrence in the fifth decade.[24] Since the advent of computed tomography (CT), the distinction among hemorrhage types has become easier. Intraparenchymal hemorrhage is most common, followed by intraventricular haemorrhages and SAH.[16,55,56] SAH is more common when the AVM is located cortically and is rarely associated with vasospasm, depending on location and thickness of blood.[40.57,58] Among patients presenting with primary SAH, in 0.6% the hemorrhage was attributable to an AVM.[59] Higher reported rate of hemorrhage in posterior fossa AVMs compared with supratentorial AVMs is possibly explained by their small size relative to supratentorial AVMs and propensity for smaller AVMs to hemorrhage.[15-18,21,60]

Guidetti and Delitala[61] reported that 89% of patients with small AVMs presented with hemorrhage, compared with 74% of patients with medium sized AVMs and 58% of large sized AVMs. Fults and Kelly[19] found that hemorrhage from posterior fossa AVMs is more frequently and more often fatal than hemorrhage from supratentorial AVMs. In series by Lesnaik et.al,[2] majority of patients (63%) with posterior fossa AVMs presented with intracranial hemorrhage. Similar results reported by Drake et al[4] who reported a hemorrhage rate of 84% in their series, whereas Matsumura et al[6] documented hemorrhage in 83% of patients. Similarly, in the Cooperative study of Subarachnoid Hemorrhage and Intracranial Aneurysms, intracranial hemorrhage was the presenting sign in 72% of infratentorial AVMs.[27] In the same study, however, 66% of supratentorial AVMs were also associated with hemorrhage. Others have reported the rate of hemorrhage among patients with infratentorial AVMs between 60–80%[3,4,27,62,63] and is twice as high as among patients with supratentorial AVMs[62]. The rate of hemorrhage in patients with brainstem AVMs is 70 to 90%[10,64,65]. However, the annual risk of hemorrhage has not been reported. In Japanese patients with 212 brainstem AVMs, the probability of a later hemorrhage was estimated to be 60% during a 10-year follow-up period[66].

Headache: Headaches are a common compliant in the patients with AVMs, even in the absence of hemorrhage. Approximately 15% of unruptured AVMs present with headache.[46] The pathological cause of the headache is hypothesized to relate to long-standing meningeal artery involvement and recruitment of blood supply by the AVM. The relationship between these malformations and chronic headaches in the absence of hemorrhage is controversial.[68–70]

Neurological deficit: Less than 10% of patients present with transient, permanent, or progressive neurological deficits not ascribed to hemorrhage or seizure.[33,36,46,71] progression of neurological dysfunction maybe the result of the long-term effects of recurrent small hemorrhages, mass effect of the AVM, hydrocephalus, or ischemic complication and steal. Steal is the term used to describe blood flow away from a region of the brain in order to flow towards AVM shunt. This flow may cause hypoperfusion, ischemia and symptoms in the region where the blood was "stolen". Risk factors for progressive neurological deficits include size[72] and shunt characterstics[73].

Seizures: Approximately 15% to 35% of patients with AVMs present with seizures.[27,30,31,36,37,39,46] Seizures may be the result of mass effect with cortical irritation; flow characteristics leading to steal, ischemia, and neuronal damage; or hemorrhage and gliosis.[31,67] Seizures are most commonly focal (simple or partial complex) but may also be generalized. Ninety percent of patients presenting with seizures had supratentorial AVMs.

Unlike supratentorial vascular lesion, seizure as the presenting symptom of a cerebellar AVM is rare and is usually related to global nervous system dysfunction from secondary effects of hemorrhage or cerebrospinal fluid (CSF) circulation abnormalities. Of 68 patients with posterior fossa AVMs, Yasargil[52] described 2 patients who presented with history of seizures. Both patients also had evidence of massive hydrocephalus that he felt accounted for their epileptic syndrome.

Other presentations: Rarely, posterior fossa AVMs may present with progressive neurologic dysfunction or cranial neuropathies similar to that seen with demyelinating process. Patient can present with deficits related to the cerebellum, brainstem and, cranial nerves including symptoms of trigeminal neuralgia and hemifacial spasm. These deficits results from the tortuous feeding arteries and draining veins exerting a mass effect on the cranial nerves or nerve root entry zone on the surface of the brainstem. In addition to hemorrhage, AVMs of the posterior fossa may also present with other symptoms. Neurologic deficits most often presenting as a mild cerebellar syndrome have been shown in up to 30% of the patients.[3,4,6,75] Lesniak et.al[2] found that 37% of the patients with posterior fossa AVMs had neurologic signs related to brainstem and/or cerebellar dysfunction at initial presentation. Chronic headache, vertigo, and ataxia were also reported by a significant number of their patients. As pointed out by Batjer and Samson[3] these deficits may be attributed not only to compressive effects but to the large ischemic territories surrounding even small malformations. Stoodley et.al[54] study of fifty-three patients with posterior fossa AVMs, presentation was hemorrhage in 35 patients (66%), progressive neurological deficit in 10 (19%), and headache in 4 (8%); 2 patients were asymptomatic.

Risk of Hemorrhage: The risk of bleeding from an AVM has been calculated in several studies. The risk of hemorrhage has been estimated at 2 to 4% per year.[33,36,37,44,46,76] Brown et al studied 168 patients without prior history of hemorrhage over 8.2 years.[46] Eighteen percent of the patients experienced symptomatic hemorrhage during the follow-up period, yielding a crude risk of 2.2% per year. Ondra et al studied 160 patients with AVMs, representing 90% of such lesions in Finland.[44] These were followed over a 24-year period. This study found 147 new hemorrhages events in 64 patients during the follow-up period, for an overall hemorrhage risk of 4% per year that was constant over time. Graf and Pollock found the initial hemorrhage risk to be approximately 2% per year.[37,76]

Using the multiplicative law of probability, the life-time risk of AVM rupture can be assessed using the formula: [1-(risk of no. of hemorrhages)^n], where n is the number of expected years of life remaining obtained from life tables.[77] However, there is a simpler way of approximating this life time risk of hemorrhages for persons with intracranial AVMs. The following formula, based on a 3% annual risk of hemorrhage, closely approximates the multiplicative formula: 105 – patient's age in years = lifetime risk of hemorrhage.[78] For eg. a 45yr old presenting with an AVM, the risk of hemorrhage is 105 – 45 = 60%.

Risk factor for Hemorrhagic presentation: Many studies have attempted to elucidate clinical and angiographic predictors for presentation with AVM hemorrhage to delineate which patients maybe at higher risk. Several studies indicate that the location influences hemorrhagic risk.

◊ Location: The presence of an AVM in a deep location[41,79] such as the basal ganglia,[17,38] posterior fossa[38] or intraventricular and periventricular areas,[80,81] may predispose to hemorrhagic presentation. Some studies also report an increased risk of hemorrhage in cerebellar lesions.[3,4,80-82]The higher risk in one study was attributed to the relatively higher incidence of associated aneurysms.[4] In contrast few others found location to be inconsequential in predicting hemorrhagic risk.[42,44,46] Comparing the incidental autopsy incidence of posterior fossa AVMs with that reported in clinical series, Monaco et al.[83] demonstrated that a significant proportion of posterior fossa AVMs remain asymptomatic. As a result, they concluded that there may be minimal or no difference in rates of hemorrhage between posterior fossa AVMs and their supratentorial counterparts.

◊ Size is a controversial factor. Several large studies, including one of unruptured AVMs at the start of follow-up,[46] found no difference in hemorrhage risk based on the size of AVM.[17,35,42,44,46,80] Others found that small AVMs(<3cm) pose a higher risk of hemorrhagic presentation.[37-39,43,45,72,84-86]Spetzler and coworkers found higher intra-arterial pressure in smaller AVMs, suggesting a potential role in hemorrhage.[87] In addition transnidal pressure gradient is higher in smaller AVMs.[87]

◊ Feeding arteries have also been assessed. Norris and colleagues evaluated number of angiographic features in 31 patients, including size and several arterial and venous parameters.[88] The only difference in those presenting with hemorrhage was slower arterial filling with contrast, suggesting high feeding arterial pressure. Mean feeding arterial pressure was confirmed to be an important factor in the pathophysiology of AVM hemorrhage by the Columbia AVM study group[38],independent of size and location. Patient may also be predisposed to hemorrhagic presentation depending on which artery feeds the nidus. Various arteries have been implicated, including perforating arteries[17,38,79] and the vertebrobasilar trunk.[17]

◊ Venous drainage: Many AVMs that rupture do so from the venous drainage system. Contributing features include deep venous drainage, often with a accompanying stenosis and occlusion; the number of draining veins; and turbulent venous flow, perhaps leading to enhanced platelet aggregation and thrombosis.[38] Deep venous drainage have frequently shown to increase the risk of hemorrhagic presentation.[17,38,79,80,84,88,89] The Columbia AVM study group examined a large number of physiologic indices in 449 patients to determine the relationship of AVM hemorrhage and venous drainage, as well as other parameters.[90] A multivariate analysis revealed that size and deep venous drainage, were independent risk factors for bleeding. From this study four groups of patient emerged, based on model prediction of probability of intracerebral hemorrhage using size and venous drainage:

1. small AVM size and the presence of deep venous drainage only, probability = 96%;
2. medium or large AVM and deep venous drainage only, probability = 80%;
3. small AVM and superficial venous drainage only, probability = 69%;
4. medium or large AVM and superficial venous drainage only, probability = 29%;

The presence, but not the size, of fragile venous aneurysm was significantly associated with risk of hemorrhage in two studies,[79.91] although this, too, is controversial.[17,46,89]

Khaw.et.al[92] suggested that infratentorial AVM location is independently associated with hemorrhagic presentation. In the multivariate model, an independent effect of infratentorial AVM location on presentation with a hemorrhage was found (Table1). In the same model, AVM size, deep venous drainage, and the presence of AVM-associated arterial aneurysms were significantly associated with hemorrhagic AVM presentation.

Table 1. Multivariate Logistic Regression Model Testing the Effect of Infratentorial AVM Location, Age, Sex, AVM Size, Deep Venous Drainage, and Associated Aneurysms on Hemorrhagic Presentation in 623 AVM Patients

	OR	95% CI	P
Infratentorial location	1.99	1.07–3.69	0.03
Patient age	1.00	0.99–1.01	0.65
Female sex	0.73	0.51–1.06	0.10
AVM size*	0.95	0.94–0.96	<0.001
Deep venous drainage	3.09	1.87–5.12	<0.001
Associated aneurysm	2.78	1.76–4.40	<0.001

*Maximum diameter in 1-mm increments.

The attributable risk (etiologic fraction) of infratentorial AVMs on presentation with intracranial hemorrhage was 7.7% (95% confidence interval ([CI], 4.3 to 11.0).

AVM and Aneurysm

The relationship between AVMs and aneurysms has been established [27,37,59,79,93-100] and numerous studies have attempted to classify the types of aneurysms associated with AVMs. In general, each classification system takes in to account the distance and flow relationship to the AVM. Aneurysms may be flow related, intranidal or unrelated.[79] The pathogenesis of aneurysms associated with AVMs is unknown. Most favored theory is that aneurysms result from hemodynamic factors as a result of the increased flow through the AVM.[96] Flow related aneurysms are saccular aneurysms arising along the course of arteries that eventually supply the AVM. A proximal flow-related aneurysm is one located on the supraclinoid internal carotid artery (ICA), the circle of Willis, the middle cerebral artery upto the anterior communicating artery, or the vertebrobasilar trunk. All flow related aneurysms beyond these locations are distal flow-related aneurysms.[79] These distal aneurysms are generally on the main feeding artery of the AVM and are also known as pedicle artery aneurysms. Intranidal aneurysms lie within the AVM nidus. Unrelated or dysplastic aneurysms are remote to the AVM. Depending on the series, aneurysms present in 2.7% to 23% of patients with AVMs.[27,37,59,93,100] The average 8% to 10% exceeds the 0.5% to 2% prevalence of aneurysms in the general population.[98] Miyasaka and coworkers found that the mean age of presentation was 41years in those with aneurysms, versus 31 in those without.[94] The male to female ratio is similar to that of entire AVM population.[98] Males maybe more likely to harbor flow-related and intranidal aneurysm, whereas females maybe more likely to have associated dysplastic or remote aneurysms.[95] As with AVMs in isolation , AVMs with associated aneurysms typically presents with hemorrhage.[3,97] Among 39 patients with 64 aneurysms, Cunha and colleagues found that intracerebral hemorrhage was the presentation in 63%.[97] Forty six percent of these bleeds were secondary to aneurysm, 33% were related to the AVM, and in 21% the site of hemorrhage was unclear. Redekop and colleagues reported that 36% of 632 patients with AVMs presented with hemorrhage.[79]Among those with intranidal aneurysms, 72% presented with hemorrhage. Similar to overall group, 40% of the patients with flow-related aneurysms presented with hemorrhage.

Aneurysms maybe multiple in 30% to 50% of patients with AVMs and aneurysms.[3,94,97] The size of aneurysm ranges from 3mm to 2.5cm and averages approximately 7.2mm to 8mm.[79] Majority of aneurysms are flow related.[27,59,79,94,95,97] The frequency of distal, feeding artery aneurysms ranges from 37% to 69%.[27,59,79,94,101] Remote aneurysms are less common, found in 1.6% to 43%.[27,79,94,95,97] Intranidal aneruysms represents approximately 20% of aneurysms,[79] but this is variable, based on type of study performed.

Several studies reported an increased risk of hemorrhage when AVM is associated with an aneurysm.[79,99] Brown and coworkers studied 16 patients with 26 aneurysms associated with unruptured AVMs. The risk of hemorrhage in a patient with a coexisting aneurysm was 7% per year at 5 years. In those without aneurysm, rate was 3% per year at 1year and declined to 1.7% per year at 5 years. Pereta and coworkers[62] demonstrated the significance of pedicle artery aneurysms in four cases. They hypothesized that short perforators (thalamoperforate and lenticulostriate) are exposed to high pressure and flow rates are more likely to undergo aneurysmal formation and rupture. Batjer and colleagues confirmed the danger of pedicle or feeding artery aneurysms.[3]

Aneurysms may increase in size, remain the same, or regress over time. With definitive AVM treatment, distal flow-related aneurysms are most likely to regress.[79] Proximal aneurysms on the circle of Willis or remote aneurysms are unlikely to change.[68,96] Samson and colleagues observed that posterior fossa AVMs were frequently associated with aneurysms on the feeding arterial pedicle to the nidus and were often the cause of hemorrhage.[7] Between 5% to 8% of all intracranial AVMs are found to have coexisting aneurysms, whereas the incidence of aneurysms associated with infratentorial AVMs maybe as high as 25%.[3,7,79,102-106]

AVM in Pregnancy

Controversy exists whether pregnancy increases the risk of bleeding from an AVM.[35,107,108-113] Robinson and associates reported a 10% risk of hemorrhage from AVM in non pregnant women of child bearing age and an 87% risk in association with pregnancy.[107] Dias and Sekhar reviewed 154 cases of hemorrhage during pregnancy due to AVM or aneurysm.[108] They found 77% of hemorrhage were due to aneurysmal rupture and 23% due to AVMs. Recurrent hemorrhage is not uncommon and increases the rate of mortality.[110]Subsequent pregnancies also carry increase risk of rebleeding.[107,109]

AVM in Children

In the large clinical series, children (younger than 20 years) accounted for 15% to 33% of all patients presenting with AVM.[27,37,114] Symptomatic AVM hemorrhage during childhood is rare, although most common cause of intracerebral hemorrhage in children is AVM.[115,116] Hemorrhage is by far most common initial manifestation of the AVM (50% to 79%),[117-122] followed by seizures(8% to 25%) and congestive heart failure (18%)[118,123,124] Hemorrhage and seizures are more common in children older than 2 years of age.[27,120,121] D'Aliberti and colleagues compared the clinical and angiographic features of 19 children (mean age ,11 years) and 120 adult with AVM.[125]The main difference between two groups were sex distribution and the size, depth, location, and complexity of the AVM. In pediatric age group AVM were commoner in males, and tended to be smaller and located superficially. Although 68% of pediatric patients presented with hemorrhage, only 6% had deep venous drainage. AVM can grow in size, and this is usually more common in the pediatric age group. The mortality associated with AVM hemorrhage in children remains high. Clinical studies of pediatric patients presenting with hemorrhage report a 6.5% to 35% mortality rate.[118,119,121,126-128]Hemorrhage location and volume predict mortality.[121,127] The mortality associated with posterior fossa hemorrhage was far greater than that associate with supratentorial lesions. The risk of rebleeding has been reported to be between 22% to 29%[121,128]and can increase mortality.[128]Despite high mortality rates; children are more likely than adults to improve after intracerebral hemorrhage due to AVM.[125]

OUTCOME

Morbidity and Mortality: The mortality associated with the initial symptomatic hemorrhage is approximately 6% to 29%.[24,30,37,42,44,46,114,129,130] Brown and coworkers reported a 29% 30-day mortality with initial hemorrhage and a long-term disability of 23%.[46] The annual risk of major morbidity or death was calculated at 2.7% per year.[14] Many studies report complete recovery or mild disability in more than 50% of patients after initial hemorrhage.[37,114,129,131] Outcome is dependent on location and type of hemorrhage, but not on size the of AVM.[36,37] Patients were more likely to have neurological deficits if the hemorrhage was parenchymal rather than SAH or intraventricular.[37,129,130] In addition hemorrhages in posterior fossa were associated with a higher mortality rate. The mortality was as high as 66.7% in patients with posterior fossa hemorrhage in one study.[36]

Recurrent hemorrhage: Recurrent hemorrhage occurs in 23% to 44% of patients, and the risk may be higher in first year after initial hemorrhage.[36,37] Of 315 patients in one study, 196 presented with an initial hemorrhage.[76] Recurrent hemorrhages was found in 44% patients with 591 patient-years of follow-up, for an annual risk of recurrence of 7.45% per year. Four AVM groups were constructed to predict hemorrhage risk on the basis of three significant variables of the multivariate analysis.

- ◊ The low risk group had no prior history of hemorrhage, more than 1 draining vein, and compact nidus
- ◊ The intermediate low risk group had no prior history of hemorrhage, more than 1 draining vein, and diffuse nidus
- ◊ The intermediate high-risk group had a history of prior hemorrhage, more than 1 draining vein, and diffuse nidus
- ◊ The high risk AVM group had history of prior hemorrhage, one draining vein and diffuses nidus.

The annual rates of hemorrhage were 1.31% for the low-risk group, 2.4% for both intermediate groups, and 8.99% for the high risk group. Other studies confirm that prior hemorrhage is a risk factor for subsequent hemorrhage.[27,33,36,37,80,88,114,132,133] The Columbia AVM study group found an 18% per year risk of hemorrhage in patients with prior hemorrhage, compared with 2% per for all others.[90] Mortality does not increase with subsequent hemorrhagic episodes.[36] The mortality associated with subsequent hemorrhage has been estimated as 12% to 15%.[27,33,42,114]

PREOPERATIVE IMAGING

Neuroimaging evaluation should begin with a non-contrasted CT scan to determine whether the AVM has ruptured and, if so, the location of the hemorrhage. The distribution of intracranial blood can be critical in the therapeutic decision-making process, particularly in cases in which the hemorrhage is predominantly subarachnoid and is therefore likely due to the rupture of an associated cerebral aneurysm. Use of MR imaging, and in particular T2-weighted imaging, will reveal regions of hypointense flow voids, representing dilated and tortuous arterialized veins as well as enlarged feeding arteries. All patients must undergo formal six-vessel catheter angiography for accurate characterization of the anatomy and hemodynamics of the AVM. In particular, all feeding arteries and draining veins must be diligently identified preoperatively in preparation for a complete resection. High-resolution magnification studies are required of both Vertebral arteries (VAs), both internal carotid arteries (ICAs), and both external carotid arteries (ECAs), because approximately 10% of infratentorial AVMs are fed by one or both external carotid arteries.[1] Because flow-related aneurysms are not infrequently the origin of hemorrhages in the case of posterior fossa AVMs, angiographic identification of all such lesions is of paramount consideration. Of additional importance to note on angiographic studies is the presence of en passage feeding vessels of the AVM, namely vessels that do not directly communicate with veins through the nidus, but rather travel through the AVM, often yielding branch vessels along the way. The presence of an en passage artery that subsequently subserves eloquent cortex after passing through the AVM immediately renders the AVM **"eloquent,"** even if the lesion does not reside in eloquent issue per se. Superselective angiography is often required to define this complex vascular anatomy accurately.

CLASSIFICATION SCHEMES[134]

The first grading system of AVMs was introduced in 1977 by Luessenhop and Gennarelli.[135] Since then many grading systems have been introduced.[11,136-139] However the most widely used is the system proposed by Spetzler and Martin.[11] This classification is based on summing the points assigned to three variables: size, venous drainage, and eloquence of adjacent brain. Eloquence was defined as the primary sensory, motor, language, and visual cortices; thalamus; hypothalamus; brainstem; the deep cerebellar nuclei and cerebellar peduncle. The Spetzler-Martin grading system has become adopted widely by surgical and focused irradiation series.[139-148] Criticisms of the Spetzler-Martin grading system have included the inclusion of deep venous drainage, the definition of eloquence, and lack of consideration of nidus compactness.[149] Independent researchers established, however, that the Spetzler-Martin grading variables are significant in influencing surgical outcome.[150,151]The advantages of this grading system are that it allows for interseries comparison along with easy applicability.

Spetzler Martin Grading of AVMs[11]

(With reference to posterior fossa AVMS)

Characteristic	Grade@
Size	
Small (<3 cm)	1
Medium (3–6 cm)	2
Large (>6 cm)	3

Eloquent area*	
No	0
Yes	1
Venous drainage	
Superficial	0
Deep#	1

@ **Grade** = [size]+[eloquence]+[venous drainage]

***Eloquent area** in posterior fossa: the brain stem; the deep cerebellar nuclei; and cerebellar peduncle. The cerebellar cortex is considered non-eloquent

#Deep veins: internal cerebral veins, basal veins or precentral cerebellar vein; only cerebellar hemispheric veins that drain directly into the straight sinus are considered to be superficial.

Posterior fossa AVMs have been further classified to facilitate developing an operative approach, to predict blood supply to the AVM, and aid in determining the need for preoperative adjunctive embolization.[72]

Kopitnik and colleagues have categorized AVMs in posterior fossa as located in the cerebellar vermis, cerebellar hemisphere, cerebellar tonsil, superficial pial brain stem, or deep parenchymal brainstem.[4,7,52] AVMs involving the cerebellar vermis and cerebellar hemisphere are the most commonly encountered AVMs within the posterior fossa because of the large geographical area represented by the cerebellar hemisphere and vermis compared with the size of the cerebellar tonsils and brainstem which comprises only 5% of posterior fossa AVM locations.[3,4,7,52,152]

In total of 72 patients included in study by Khaw et.al[62] harboring an infratentorial AVM: 42 involved the cerebellar hemisphere only; 8 affecting the cerebellar hemisphere and either brain stem, vermis, or peduncle; 6 affecting solely the vermis; 7 in the brain stem only; 7 involving the deep cerebellar nuclei in various combinations; and 1 in the brain stem and peduncles; 1 patient had a large, complex AVM extending from the brain stem through the peduncle and the deep nuclei to the hemispheric surface. Lesniak et .al[2] breakdown of the cases by location versus specific AVM characteristics is shown in Table 2.

Table 2. Posterior fossa arteriovenous malformations

Location	Vermis (n=8)	Hemisphere (n=15)	Brainstem (n=9)	Tonsil (n=2)	CPA (n=4)
S M GRADE					
V	0	0	0	0	0
IV	7	4	1	0	0
III	1	9	8	2	3
II	0	2	0	0	1
I	0	0	0	0	0
Nidus size (cm)					
>6	0	2	0	0	0
3-6	6	9	2	2	2
<3	2	5	7	0	2
Arterial supply					
SCA	8	10	7	0	2
AICA	3	4	3	0	2
PICA	1	3	0	2	2
VERT	0	0	2	0	0
ECA	1	1	0	0	0
Venous drainage					
Galenic	6	11	7	2	2
Transverse	2	4	0	1	1
Straight	3	2	3	0	0
Petrosal	0	0	0	0	1

(AICA, anterior inferior cerebellar artery, CPA, cerebellopontine angle; ECA, external carotid artery; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery.)

On the Spetzler-Martin scale, 7 of 8 patients with vermian AVMs presented with grade IV lesions. In the remaining locations, the most common was grade III. Medium- sized AVMs (3–6 cm) were present in 21 of the patients (55%). In the brainstem, however, the majority (75%) of AVMs were small (<3 cm). The arterial supply was almost invariably from bilateral superior cerebellar arteries (SCAs), with occasional contributions from the anterior inferior cerebellar artery (AICA) and rare contributions from the posterior inferior cerebellar artery (PICA). The most common venous drainage occurred directly into the galenic system, with few veins emptying into the straight or transverse.

PATIENT SELECTION AND RATIONALE

Most patients with posterior fossa AVMs present with an acute hemorrhagic event at which time emergent evacuation of the associated intraparenchymal hematoma is often required. The goal of surgery in this situation is to reduce intracranial pressure and to decompress the brainstem via subtotal hematoma removal. The AVM and immediately adherent hematoma are left in situ, with definitive management of the underlying AVM deferred until associated cerebral / cerebellar edema has resolved (usually 4–6 wk after the last hemorrhagic episode). However aneurysms associated with AVM are treated by endovascular intervention in the acute setting if they are considered the likely source of the AVM hemorrhage. Concurrent obstructive hydrocephalus, when present, usually requires external ventricular drainage, with a minority of patients eventually requiring permanent ventriculoperitoneal diversion. Rarely, it may be necessary to resect the AVM during the initial hematoma evacuation because of persisting bleeding. Clearly, this is not an optimal approach given incomplete preoperative imaging, the presence of edematous brain and blood products that impair identification of AVM and normal anatomy, and loss of physiological brain autoregulation after hemorrhage.

Patients presenting with nonhemorrhagic symptoms or presenting in a delayed fashion after previous hematoma evacuation can be evaluated in a more elective manner. After a complete history and physical examination, a preoperative MRI scan is obtained with axial, coronal, and sagittal images to identify the location of the AVM in relation to the eloquent structures of the posterior fossa. A standard six vessel cerebral angiogram is also obtained to determine arterial supply, nidus architecture, and venous drainage patterns. Occasionally, patients with asymptomatic AVMs may not be treated but followed clinically on an annual or semiannual basis. However, surgical removal of symptomatic posterior fossa AVMs should be considered the treatment of choice because it immediately removes the risk of future hemorrhage and alleviates symptoms related to mass effect and vascular steal. AVMs involving the cerebellum, subarachnoid cisterns, and pial surfaces of the brainstem can be surgically resected with minimal morbidity to the patient. In certain situations, resection carries an unacceptable risk of injury to the patient, including surgical removal for brainstem AVMs that do not present to the pial surface, cerebellar AVMs that involve the deep cerebellar nuclei, poor neurological or medical condition of the patient, and advancing age. In these situations nonoperative management using stereotactic radiosurgery or staged endovascular embolization is considered along with components of combined AVMs that are not amenable to surgical resection (e.g., the brainstem parenchymal component of a cerebellar hemisphere-brainstem parenchymal AVM).

After it has been decided that the AVM is amenable to resection, surgery is planned in a single or staged setting after recovery from initial hemorrhagic sequelae (when present), treatment of associated aneurysms, and completion of preoperative adjuvant treatments. Many posterior fossa AVMs can be treated with preoperative endovascular embolization to decrease the size and morphology of the AVM. Embolization is often completed in several stages to minimize the risk of breakthrough bleeding that can occur when a large volume of an AVM is embolized in a single session. Radiosurgery can also be used preoperatively to decrease AVM size and obliterate AVM components located in high-risk eloquent regions of the brainstem and cerebellum. Timing of surgery can be further modified after careful evaluation of hemorrhagic risk.

MANAGEMENT

A multidisciplinary decision-making process is the best way to offer patients better results with less morbidity. Treatment should be initiated only after a complete evaluation has been performed and taking into consideration such factors as the patient's occupation, age, and clinical status in the context of the results that may be achieved from all available therapeutic modalities. Current treatment modalities for AVMs include endovascular intervention, radiosurgery, and microsurgery. Embolization is often beneficial in reducing the vascular supply of an AVM, and it often used preoperatively to minimize the risk of intraoperative bleeding.

Lesnaik.et.al[2] treated all the patients with uniformly large cerebellar AVMs (>5 cm) and multiple arterial feeders with preoperative embolization. The combination of intraoperative embolization with surgical resection has been successful in the treatment of large cerebral AVMs. For instance, Zhao et al[153] treated 50 patients with giant AVMs by means of preoperative embolization and surgical resection and found excellent results in 88% of the patients. Similarly, Hongo et al[154] treated 27 cases of giant AVMs with preoperative embolization and found good results in 70% of the patients. Lesnaik et.al has divided the benefits of preoperative embolization into early and late advantages:

1. Early benefits have been shown to result in less intraoperative bleeding and, therefore, easier surgical resection.[155]
2. Late benefits include decreased postoperative bleeds, and there is an increasing amount of data indicating that reducing the shunt flow through the nidus in a stepwise fashion is helpful in preventing postoperative hemodynamic overload to the surrounding brain.[154,156,157]

When used alone, however, embolization has shown limited success mainly in the treatment of small AVMs.[158-161] Moreover, incomplete embolization is associated with worse clinical outcome as well as the potential for increased neovascularization.[162,163] The results of Lesnaik. et.al further corroborate the experience of others and help to reinforce the role of embolization in the management of patients with posterior circulation AVMs.

Radiosurgery represents a viable treatment modality for patients with inaccessible or small AVMs. Lesnaik et.al treated 16 patients with radiosurgery. Of these, 9 had brainstem AVMs. Overall, they observed a good neurologic outcome in 81% of the patients at follow-up. The outcome was directly related to initial presentation, because 2 patients who presented with hemorrhage remained severely disabled despite successful obliteration of the AVM. One other patient who rebled during the course of therapy recovered, although with considerable neurologic deficits. They observed no permanent neurologic deficits caused by radiosurgery. Although the results with respect to the obliteration rate and rebleeding rate are better than those published in the literature (50%–70% at 3 years),[64,164] this discrepancy can be explained by the relatively small number of patients treated with radiosurgery in this series as well as the extended follow-up time. Nevertheless, when chosen appropriately, radiosurgery can provide a useful adjunct in the management of posterior fossa AVMs.

As in the case of supratentorial AVMs, surgery remains a safe treatment of choice for select lesions within the posterior fossa. Indeed, it may represent the most effective treatment for large AVMs that present with or without hemorrhage and for AVMs that lie outside the brainstem and are easily accessible via surgical exploration. Lesnaik.et.al[2] showed good results in 72% of the patients. They undertook large surgical exposure, microsurgical dissection, and preservation of main drainage until total control of the nidus was achieved and this all contributed to achieving complete and safe resection of AVM. Only few reports have been available in which more than 10 brainstem AVMs were surgically treated.[4,52] Solomon and Stein[10] reported nine surgically-treated cases out of 12 patients with brainstem AVMs, including four dorsal midbrain, five pons, and three cerebellopontine angle AVMs. In this series, total resection was accomplished in two dorsal midbrain, four pons, and three cerebellopontine angle AVMs with a 22% morbidity rate. Drake et al[4] described total resection in one dorsal midbrain AVM, two pontine hemorrhage with cryptic vascular malformations, and seven cerebellopontine angle AVMs, with partial resection in one primarily extrapial ventral midbrain AVM extending into posterior perforated substance, in a total of 15 brainstem and seven cerebellopontine angle AVMs. Five patients died related to the surgery. In their series, most of brainstem AVMs received only exploration and surgical excision was abandoned when the lesions were buried within the brainstem. Yasargil[52] successfully resected six out of 14 mesencephalic AVMs (three were small-sized and located in the dorsal and three were medium-sized and in the dorsolateral mesencephalon). He also described four surgical cases of pontine AVMs. He described that most of safely resected cases were located epipially. Batjer and Samson[1] described six surgically resected cases (four pons and two cerebellopontine angle AVMs) with one mortality. Considering the reported cases and series by Kazuhiko Nozaki et.al[165], AVMs involving the brainstem are candidates for surgical extirpation when the nidus locates mainly sub or extrapially, such as dorsal midbrain AVMs and cerebellopontine angle AVMs. However, when most part of the nidus locates within the brainstem, considerable morbidity should be taken into account particularly in brainstem AVMs without intraparenchymal hemorrhage. Recently, Lawton et al.[166] reported that eight brainstem AVMs were successfully resected with no serious neurological deterioration. They stated that brainstem AVMs typically reached a subarachnoid surface that could be accessed through several surgical approaches, but the location of the nidus was not described.

Stereotactic radiosurgery has been widely applied to cerebral AVMs with a diameter of 3 cm or less and an obliteration rate of 65 to 85% is expected with no definite effect on bleeding rates during latency periods[164,167-170,171] Obliteration rates depend on dose and nidus volume, and increase in obliteration rates may be achieved at the price of higher risks of complications.[174] Recently, the role of stereotactic radiosurgery in the treatment of brainstem AVMs has been reported, and relatively lower obliteration rates (28–73% at 3 yr and 66% at 6 yr) as compared with AVMs in other locations, probably because of the reduced dose to avoid serious adverse radiation effects.[64,65,164,173] Post-radiosurgical hemorrhagic rates in brainstem AVMs have been reported to remain unchanged[64] or decrease[65] until complete obliteration. Because endovascular reduction of nidus size is not yet proven to be effective[171], preradiosurgical deliberate planning should be mandatory considering the size, location of the nidus, patient's age, and neurological deficits.

Kazuhiko et.al[165]undertook microsurgical total resection in 56% of all patients (14 out of 25 patients; 0 out of 3 in patients with ventral midbrain, 6 out of 10 in patients with dorsal midbrain, 2 out of 5 in patients with pons, 6 out of 6 in patients with cerebellopontine angle, 0 out of 1 in patients with and medulla oblongata, AVMs respectively), and surgical resectability was largely influenced by the location of the nidus. Dorsal midbrain and cerebellopontine angle AVMs located mainly sub- or epipially and surgical resection could be performed with acceptable morbidity. Hematoma cavity between the nidus and brainstem facilitated microsurgical resection. However, even in these regions, no patients with nonruptured AVMs received surgical resection. Surgical application seemed to be considerably limited in intrinsic brainstem AVMs in this series, even in hemorrhagic cases. Total resection was achieved in 14 of 19 patients (74%). Stereotactic radiosurgery was applied as a main treatment modality in two patients with ventral midbrain AVMs and one patient with a pons AVM, and as a post microsurgical treatment in one patient with a medulla oblongata AVM. Complete obliteration was observed in all four (100%) of these patients. One patient with a ventral midbrain AVM suffered from hemiparesis owing to radiation necrosis. They performed embolization in nine patients (six preoperative, one preradiosurgical, one feeder occlusion, and one failed) and embolization related complication was not observed. The morbidity and mortality rates in this series were 25 and 0%, respectively, in patients with brainstem AVMs, which was about four times higher than that in patients with supratentorial AVMs. Microsurgery related permanent neurological complications were observed in five patients (postoperative nonfatal bleeding in one patient and hemiparesis in one patient with dorsal midbrain AVMs, deterioration of hearing acuity in two patients, and abducens paresis and hearing deterioration in one patient with a cerebellopontine angle AVM). During the follow-up period of 7.8 years, one patient with a nonruptured pontine AVM died owing to fatal hemorrhage and one patient with a subtotally resected dorsal midbrain AVM experienced nonfatal hemorrhage 2 years after the surgery and died 4 years after the surgery of unknown etiology. However, because of the small numbers of patients, we cannot conclude the definite treatment strategies for these lesions.

General Principles of Surgical Management[175]

Sinclair et.al follows similar surgical strategies as those used in the resection of supratentorial malformations for infratentorial AVMs. After routine preoperative anesthesia consultation, patients do not require specific medical therapy before surgery. Resection is usually planned 1 week after the last endovascular embolization when feasible to permit resolution of associated edema, re-equilibration of AVM hemodynamics, and minimize recruitment of new arterial supply. Surgery is performed under general anesthesia with mild hypothermia (33.0°C) and intraoperative electrophysiologic monitoring using somatosensory evoked potentials, motor evoked potentials, and brainstem auditory evoked potentials. A radiolucent head clamp and frame are required for intraoperative angiography to confirm complete AVM resection. Patients receive preoperative antibiotics and steroids in the operating room after induction of anesthesia but rarely require osmotic diuretic therapy. Blood pressure is maintained initially in the normotensive to slightly hypotensive range and lowered accordingly (occasionally to a mean arterial pressure of 60–65 mm Hg) during AVM dissection and removal. Use of the sitting or slouch positions for surgery mandates use of precordial ultrasonography and a central venous access at the level of the superior vena cava/right atrium for detection and treatment of air embolism. After selection of surgical approach, the patient is positioned appropriately to ensure that the head is maintained above the heart and that jugular venous drainage is not impaired. A craniotomy is fashioned using intraoperative frameless stereotaxy to facilitate wide exposure of the underlying vascular malformation. Similar to supratentorial AVMs, the underlying parenchyma is first inspected to identify the superficial arterial inflow and venous outflow, which are correlated with the preoperative MRI and angiogram. If the AVM does not present to the pial surface, sometimes a draining vein can be followed retrograde to the AVM nidus, with care taken not to compromise flow in the vein. Intraoperative frameless stereotaxy is also used after opening the dura to confirm the location of the AVM nidus and its relationship to surrounding brain, cranium, and other normal vascular structures. The dissection then proceeds around the margins of the AVM with coagulation and division of arterial feeders and small veins circumferentially. An attempt is made to expose and occlude larger arterial feeders to the AVM first. Dissection is facilitated by the gliotic plane, which usually exits between the AVM and normal parenchyma[3,4,176] as well as by previous hemorrhage cavities.[4,176] Care is taken to spare large veins draining the vascular malformation, with dissection often continuing under or around such vessels. Occasionally, it is difficult to distinguish arterialized veins from arterial feeders, and in these instances, determining the direction of flow in the vessel with a flow meter can be useful. The surgeon must ensure that arteries supply only the vascular malformation before division because sacrifice of an en passage artery may lead to significant postoperative neurological deficit. This can usually achieved by dividing arteries as close as possible to the margins of the AVM[4,176] and, occasionally, using temporary clip occlusion with observation for changes in electrophysiologic monitoring. After observation for 10 to 15 minutes of temporary occlusion, the vessel is usually safe to divide if the electrophysiological monitoring is normal. The dissection is gradually deepened circumferentially until the majority of the AVM is mobilized on its venous pedicle (s). The deepest aspect of the AVM usually arises from an ependymal surface and is often supplied by very fragile arteries in close proximity to associated veins.[4] Such vessels often create problematic bleeding as a result of retraction into the adjacent white matter. As a result, bipolar coagulation is less effective, and the surgeon often must use Sundt AVM microclips to achieve hemostasis of such vessels[3,9] Sinclair et.al have found that the no. 3, 4, and 5 microclips were most valuable. After complete dissection of the AVM and interruption of arterial inflow, the AVM usually reduces in caliber and size and darkens in color. The preserved major draining vein (s) can then be safely coagulated or clipped and divided. If there is any uncertainty regarding patency of the AVM, the vein can first be temporarily occluded with a clip or bayonet forceps to ensure that the AVM does not enlarge as a result of persisting arterial inflow. Should this occur, the vein is unoccluded, and the surgeon must explore the dissection bed to verify that the dissection has continued in the correct plane and that all arterial inflow has been completely interrupted from the nidus. Once this has been confirmed, the venous drainage can then be safely divided and the AVM removed.

After AVM removal, attention is directed to the walls of the resection bed, with the surgeon carefully inspecting for evidence of residual malformation. This is often apparent to the surgeon as persisting bleeding from a region of white matter. After complete removal of the AVM, the patient's blood pressure is slowly normalized with careful observation for bleeding. After achievement of hemostasis, the resection bed is lined with a hemostatic agent, and the patient's blood pressure is again reduced to the slightly hypotensive range. An intraoperative cerebral angiogram is subsequently obtained to verify complete AVM removal before standard closure of the craniotomy.[3,176]

SURGICAL APPROACHES TO POSTERIOR FOSSA

As previously described, posterior fossa AVMs can be classified into three groups on the basis of location of the nidus, principal arterial supply, and venous drainage. Primary involvement of the cerebellum, brainstem, or a combination of both structures dictates selection of surgical approach. Within each group, the AVM location can be further described as ventral or dorsal, rostral or caudal, and midline, paramedian, or lateral. The surgeon must select the surgical approach based not only on the specific nidus location but also with consideration to which approach will best provide initial access to the arterial supply of the AVM and later the venous drainage.[176,177]The surgeon must also be aware of the small confines of the posterior fossa, as well as its eloquent anatomy including cranial and spinal nerves, vertebral and basilar arteries, jugular veins, dural sinuses, vestibular and auditory apparatus, and craniovertebral junction.[176,178]

Suboccipital and Retrosigmoid Approaches: AVMs involving the vermis, paramedian cerebellar hemispheres, fourth ventricle, and dorsal medulla are easily accessed via a standard suboccipital craniotomy.[176-78] Depending on rostral or caudal location of the AVM, the craniectomy/craniotomy can be extended to expose the torcula and transverse sinuses superiorly or the foramen magnum inferiorly. Lateral extension of this approach to the sigmoid sinus provides access to lesions of the lateral pons, middle cerebellar peduncle, superior lateral medulla, and cerebellopontine angle.

The standard midline or paramedian suboccipital approach is performed with the patient prone and the neck appropriately flexed to open the interval between the foramen magnum and C1. The lateral suboccipital (retrosigmoid) approach is usually performed with the patient supine, head flexed, and rotated to the opposite shoulder but also may be performed with the patient in a prone or lateral position.[176,178] Also one may use lateral or modified park-bench position.[176,178] The patient's neck is flexed in the anteroposterior plane, rotated 45 degrees to the contralateral shoulder, and laterally flexed 30 degrees to the floor[178] The patient's dependent arm is suspended between the edge of the table and Mayfield head holder with tape or a padded sling. The superior shoulder is then retracted inferiorly with tape to provide an unobstructed view of the cranial-cervical junction. Somatosensory evoked potential monitoring during positioning is very important to prevent stretch injury to the brachial plexus and peripheral nerves of the upper extremities. Care must be taken to minimize tension on the cervical spine and brachial plexus with use of shoulder or axillary rolls where applicable and continuous somatosensory evoked potential monitoring during positioning. In addition, excessive rotation or flexion of the cervical spine must also be avoided to prevent compromise of jugular venous outflow. The craniotomy is planned with or without intraoperative stereotaxy to provide a large bony exposure circumferentially around the AVM. This often requires exposure of the underlying dural sinuses and extension to the foramen magnum.[177] The dura is subsequently opened widely to the limits of the craniotomy within the margins of the dural sinuses. Intradurally, bridging veins that are not involved in the AVM are first coagulated and then divided to ensure they are not torn during placement of retractors and dissection. Landmarks including the tentorium, petrous bone, vertebrobasilar circulation, and cranial nerves are then identified.[178] Retractors are subsequently placed after localization of the AVM by inspection of the parenchyma and confirmation with intraoperative stereotaxy. AVMs involving the midline cerebellar structures including the fourth ventricle often require the vermis to be split before retractor placement. Lesions of the lateral pons, cerebellar peduncle, or cerebellopontine angle require retraction of the cerebellar hemisphere, often with coagulation and division of the petrosal vein. After resection of the AVM and achievement of meticulous hemostasis, the dura is closed in a watertight fashion, often with the aid of a dural patch graft. The bony margins of the craniotomy are waxed with bone wax, and any exposed mastoid air cells are exenterated of mucosa before packing with muscle and bone wax. The craniotomy and overlying soft tissues are then closed in standard fashion.

Far Lateral Approach: AVMs of the anterior and lateral cervicomedullary junction, medulla, lower pons, and lateral aspect of the inferior cerebellum are optimally accessed via the far lateral approach.

Supracerebellar Infratentorial: Exposure of the tectal plate of the mesencephalon and superior cerebellum is achieved with the supracerebellar infratentorial approach with the advantage of avoiding significant dissection of the galenic venous system.[176,178] AVMs situated in this region can be resected via this exposure, providing that the nidus does not extend above the incisura.[178]

Occipital Transtentorial Approach: AVMs of the superior cerebellar peduncle, anterior cerebellar vermis, superior medullary velum, and tectal plate of the mesencephalon can be resected via the occipital transtentorial approach. This approach provides a wide exposure in the region of the tentorial incisura in comparison with the supracerebellar-infratentorial approach but often requires significant occipital lobe retraction, with the possibility of resultant visual field deficits.[178] AVMs of the dorsal midbrain and pineal region can be accessed after further division of the overlying arachnoid and careful mobilization of the deep venous system accordingly.

Petrosal Approach: Subtotal removal of the petrous temporal bone offers visualization of the midpontine region of the brainstem and is often combined with a standard subtemporal or retrosigmoid approach depending on the rostral or caudal extent of the AVM.

Subtemporal Approach: Access to the lateral midbrain and pontomesencephalic region is achieved via the standard subtemporal approach. The tentorial edge often requires division posterior to the entry of the trochlear nerve into the tentorium and suturing of the respective flaps to the floor of the middle cranial fossa to visualize the pontomesencephalic junction and upper pons.

Transsylvian Approach: AVMs involving the anterolateral midbrain and interpeduncular cistern may be approached with a pterional craniotomy. AVMs located in the anterolateral upper brainstem can be removed via this approach with the advantage of early identification of arterial supply and visualization of upper cranial nerves.[178] This approach can be modified with an orbitozygomatic craniotomy to provide a more midline trajectory to the interpeduncular fossa. The standard pterional craniotomy can also be modified to gain more lateral exposure of the brainstem with an extended frontotemporal craniotomy. The modified pterional craniotomy or anterior temporal approach allows greater mobilization of the temporal lobe with the option to combine a transsylvian and subtemporal exposure if necessary.

Combined Approaches: Occasionally, it may be advantageous to perform two approaches in a combined fashion, offering two different avenues for AVM resection. This is particularly true for lesions located in the superior and anterolateral aspect of the posterior fossa where exposure from above and below the tentorium greatly facilitates removal. The most commonly used exposure involves the use of the subtemporal and the retrosigmoid approaches with division of the intervening tentorium. In this approach, the patient is positioned supine with the head turned contralateral to the lesion with use of a shoulder roll for protection of the cervical spine and brachial plexus. After division of the tentorium, the surgeon is able to access the extreme anterolateral brainstem in a combined supratentorial-infratentorial manner. Other combined approaches that will not be further detailed in this chapter include the far lateral-presigmoid or petrosal approach, the transsylvian-subtemporal or modified frontal-temporal approach, and the transsylvian-anterior petrosal approach.

POSTOPERATIVE CARE

After surgery, patients are managed in the intensive care unit for 24 to 48 hours. Similar to supratentorial AVM surgery, steroid therapy is continued and fluid status controlled to maintain euvolemia with aims to minimize both postoperative edema and venous stasis. In addition, blood pressure is maintained in the moderately hypotensive range (65–75 mm Hg) for the first 24 hours followed by normotension the next day. Immediately after surgery, a postoperative computed tomography scan is obtained as a baseline study while the patient is closely monitored for signs of neurological deterioration. Given the small size of the posterior fossa and its eloquent contents, the surgeon and intensive care unit team must remain diligent for evidence of postoperative swelling, hemorrhage, and hydrocephalus, which can lead to rapid clinical deterioration and death. The patient is subsequently transferred to a step-down observation unit and progressively ambulated before discharge. Steroid medication is gradually tapered over a 7 to 14 day period after surgery. Formal cerebral angiography is obtained postoperatively before hospital discharge to confirm complete AVM removal because occasionally a small residual AVM or arteriovenous shunt may not be visualized on the intraoperative study.

COMPLICATIONS

O'Shaughnessy et.al found that problems in the early postoperative period were hemorrhage, cerebral edema, hydrocephalus, and normal perfusion pressure breakthrough bleeding and in many cases, related to inadequate blood pressure control postoperatively. It is extremely important to recognize and treat each complication optimally even after an apparently complete resection. Clearly, the risk of postoperative hemorrhage is highest when there is significant fragility of vessels in the surrounding microcirculation. Perhaps more importantly, an increased risk of bleeding in the postoperative period occurs when the AVM and the volume of previously shunted blood is largest. Although surgical resection remains the treatment of choice for posterior fossa AVMs, certain risks remain inherent to this modality of therapy. The major complications associated with surgery include hemorrhage, edema, arterial infarction, venous infarction, hydrocephalus, and CSF leakage. Intraoperative bleeding occurs to some degree during resection of most AVMs. It is often a result of inadvertently entering the malformation nidus during dissection or as a consequence of subtotal removal. Gentle tamponade and coagulation usually will control the bleeding followed by reestablishment of the dissection plane around the gliotic periphery of the AVM. Significant hemorrhage may also occur during the final stages of deep dissection from small friable arterial feeders, which often respond poorly to coagulation. Control of bleeding from these vessels is best approached with the placement of Sundt AVM microclips. Catastrophic hemorrhage may occur from premature division of venous outflow during AVM dissection. Distension and rupture of the AVM ensues in addition to swelling and hemorrhage from the surrounding brain as a result of normal perfusion breakthrough bleeding (NPPB). Management of bleeding in this situation is difficult, but attention should be directed to further dissection and interruption of the remaining arterial inflow rather than attempts to coagulate the hemorrhagic portions of the AVM.

Postoperative hemorrhage must always be assumed to be a result of incomplete AVM resection.[9] Management, when feasible, includes obtaining an immediate angiogram before hematoma evacuation and removal of residual AVM. All patients should receive formal postoperative angiography despite the presence of normal intraoperative imaging because general anesthesia may significantly alter cerebral hemodynamics and subsequent image quality. A postoperative hemorrhage with angiographic confirmation of AVM resection may occur as a result of incomplete operative hemostasis or NPPB. NPPB is a life-threatening complication that usually occurs after removal of a large AVM. Reestablishment of normal perfusion to chronically ischemic areas of brain adjacent to the AVM can lead to progressive edema and bleeding because of impaired autoregulation. This phenomenon can be avoided by preoperative reduction in AVM volume using embolization or radiosurgery, staging surgical resection, and maintaining induced hypotension for 24 to 48 hours postoperatively. Management of NPPB involves induced hypotension and barbiturate coma to decrease cerebral metabolism and blood flow.[178]

Cerebral edema often occurs after AVM resection in the adjacent white matter as a result of retraction, dissection, and redirection of blood flow to non AVM brain. Control with steroids, induced hypotension, and hyperosmolar therapy is usually successful in preventing clinical deterioration in the patient. Occasionally, edema may become life threatening and refractory to medical management. An external ventricular drain should be placed to manage concurrent hydrocephalus and consideration given to other etiologies such as infarction and NPPB. Subtotal resection of the involved cerebellar hemisphere with preservation of the deep nuclei may be required in the setting of progressive mass effect but should be completed before significant neurological deterioration when possible.[9]

Perioperative infarction during AVM surgery may be arterial or venous in etiology. Interruption of en passage arterial feeders can lead to infarction in adjacent brain during AVM resection. This can be avoided by temporarily occluding vessels before division with temporary aneurysm clips while performing intraoperative electrophysiological monitoring. Venous infarction may occur after ligation of large veins or as a result of stasis in veins after removal of arteriovenous shunting. Retrograde thrombosis can lead to extension of infarct volume, particularly in the immediate postoperative period when blood pressure and volume are iatrogenically reduced to prevent hemorrhage.

Postoperative noncommunicating hydrocephalus may occur as a result of edema or hemorrhage, obstructing CSF outflow pathways in the posterior fossa. Occasionally, communicating hydrocephalus may also occur as a consequence of meningeal inflammation, particularly when significant spillage of blood occurs during surgery. Temporary external CSF drainage may be required in either situation along with steroid administration. Rarely, permanent CSF diversion with a ventriculoperitoneal shunt is required. Pseudomeningocele formation and CSF leakage are also potential complications of any intradural posterior fossa procedure. They are best avoided by ensuring a water-tight dural seal and multilayer soft tissue closure. CSF leakage can be usually be managed with a short course of bed rest and, occasionally, temporary lumbar drainage. Most CSF leaks respond to this regime with few patients requiring surgical re-exploration.

Although postoperative hemorrhage accounts for most of the operative morbidity in AVM surgery, experience of this complication was seen in only 1 patient (5.5%) in series by Lesnaik et al. This was within the range of 8% to 10% reported by Drake et al.[3] Lesnaik et al also showed excellent patient recovery following clot removal. A total of 2 patients (11%) died after the initial evacuation of the clot as a result of irreversible brainstem damage and cerebellar edema. The cumulative morbidity and mortality rates reflect the increased risks associated with neurovascular lesions of the posterior fossa. Early diagnosis combined with safe and secure obliteration of these lesions should result in even better results of treatment for posterior circulation AVMs.

NEUROLOGICAL OUTCOME

Neurological outcome of patients analyzed by Lesnaik et.al[2] for posterior fossa AVMs and is shown in Table 3

Table 3

	Total patients AVMs (n = 38)	**Surgical Group AVM (n=18)**
GOS*		
GR	21	9
MD	9	4
SD	5	2
VS	1	1
Dead	2	2

	Embolization group (n = 4)	Radiosurgical group (n = 16)
GOS*		
GR	4	8
MD	0	5
SD	0	3
VS	0	0
Dead	0	0

GOS-Glasgow Outcome Scale, Good recovery GR, Moderate disability MD
Severe disability SD, Vegetative state VS

Overall, 50 patients (66%) had a good recovery, 14 (18%) exhibited moderate disability, 7 showed (9.2%) severe disability, 1 (1.3%) was in a vegetative state, and 4 (5.3%) died. This yields an overall morbidity rate of 10.5% and a mortality rate of 5.3%. In the surgical group, 14 patients (43%) showed good recovery, 9 (28%) had moderate disability, 4 (12%) had severe disability, 1 (3%) was in a vegetative state, and 2 (6%) died. When analyzed by category, 13 patients (72%) with AVMs had moderate to good recovery, 2 (11%) remained in a state of severe disability, and 1 (5%) was in a vegetative state. Two patients (11%) died.

Observations and results

OBSERVATIONS & RESULTS

In our study, incidence of posterior fossa AVMs was found more commonly in males as compared to females. The mean age at presentation was 32 years in majority of the patients.

Table 4: Patient Demographics of Posterior fossa Arteriovenous Malformations

Total no. of posterior fossa AVMs –	40
M/F ratio	26:14 :: 1.85:1
Mean age in years (range) -	32.05 years
(Youngest being 3 years old and eldest 60 year old)	

CLINICAL PRESENTATION

In our series most of the patients presented with the features of raised intracranial pressure i.e. headache and vomiting, followed by decrease in the level of consciousness. Only few patients presented with symptoms relating to focal neurological deficits as shown in table 5.

Table 5: Presenting symptoms

Headache	30 (75%)
Vomiting	21 (52.5%)
LOC	18 (45%)
Altered sensorium	8 (20%)
Ataxia	9 (22.5%)
Weakness	5 (12.5%)

Dizziness	5 (12.5%)
Diplopia	4 (10%)
Dysarthria	4 (10%)
Facial numb/spasm	3 (7.5%)
Facial deviation	3 (7.5%)
Vertigo	3 (7.5%)
Seizure	3 (7.5%)
Dysphasia	2 (5%)
Hearing loss	1 (2.5%)

GCS AT PRESENTATION

Most of the patients at initial presentation to us were in GCS 15, only six patients had GCS score less than 15 as shown in the table 12.

Table 6: GCS on admission

15	34 (85%)
11-14	3 (7.5%)
<10	3 (7.5%)

NEUROLOGICAL DEFICIT

Out of 40 patients 13 presented with various combinations of III, V, VI, VII, VIII and lower cranial nerve deficits. Six patients presented with motor weakness, out of which one had bilateral quadriplegia and rest one side hemiplegia/ hemiparesis. Sixteen patients had cerebellar signs on presentation.

LOCATION

Majority of the patients in our study had AVMs located in cerebellar hemisphere followed by AVM in CPA and brainstem, as shown in the table 7. In three of our patients with posterior fossa AVM had an associated suprasellar AVM. One patient had AVM in brainstem and suprasellar location, while another had in cerebellar hemisphere along with another in parietal region. Another patient who underwent embolization of the occipital AVM on check DSA showed new AVM in cerebellar hemisphere. All three were lost to follow-up and none of them had any hereditary association.

Table 7: Following table shows location wise distribution of AVMs

Vermis	03 (7.5%)
Hemisphere	25 (62.5%)
Brainstem	05 (12.5%)
Tonsil	01 (2.5%)
CPA	05 (12.5%)
Hemisphere + tonsil	01 (2.5%)
Total	**40 (100%)**

CPA - cerebellopontine angle;

PRESENTATION

Table 8 shows that most of the patients with posterior fossa AVMs in this study presented with episode of hemorrhage and out of which only eight (20%) patients had evidence of SAH at the ictus in the computed tomography scan.

Table 8: Presentation on admission

Hemorrhage	31 (77.5%)
+ SAH	8 (5 with aneurysm)
+ aneurysm	13 (30%)

SAH - subarachnoid hemorrhage,

AVM CHARACTERISTICS

The breakdown of the cases in current study by location versus specific AVM characteristics is shown in Table 9. On the Spetzler-Martin scale, 2 each of 25 patients with cerebellar AVMs presented with grade IV & V lesions, though grade II was most common grade. In the remaining locations, the most common was grade III. Small sized AVMs (<3 cm) were present in 26(65%) of the patients. In the brainstem, however, all the AVMs were small(<3 cm) sized. The arterial supply was almost invariably bilateral in cases of vermian AVMs [either from superior cerebellar arteries (SCAs), or from the posterior inferior cerebellar artery (PICA)]. Major arterial feeders in posterior fossa AVMs were from superior cerebellar arteries (SCAs), the anterior inferior cerebellar artery (AICA) and the posterior inferior cerebellar artery (PICA). The most common venous drainage occurred directly into the deep venous system, with few veins emptying into the superficial system.

Table 9: **<u>A breakdown of all 40 cases by location versus specific AVM characteristics</u>**

Location	Vermis (n=3)	Hemisphere (n=25)	Brainstem (n=5)	Tonsil (n=2)	CPA (n=5)
SM grade					
V	0	2	0	0	0
IV	0	2	0	0	0
III	2	4	4	0	1
II	0	11	1	1	4
I	1	6	0	1	0
Nidus size (cm)					
>6	0	4	0	0	0
3-6	2	5	0	1	2
<3	1	16	5	1	3
Arterial supply					
SCA	1* + 1	17	4	0	2
AICA	1	14	1	1	4
PICA	2* + 1	18	1	2	2
BA	0	0	2	0	2
ECA	0	4	0	0	1
PCA	0	1	1	1	1
Venous drainage					
Deep	2	15	4	0	1
Superficial	1	9	1	2	4

(AICA, anterior inferior cerebellar artery, CPA, cerebellopontine angle; ECA, external carotid artery; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery. * bilateral supply)

We further analyzed our angiographic data for various parameters of AVMs in the posterior fossa which included size, number of feeders, venous drainage and SM grade etc. to predict and delineate patients who maybe at higher risk for hemorrhagic presentation in case of posterior fossa AVMs.

Size: The nidus size of posterior fossa AVMs in majority of the patients as shown in table 10 was small (< 3 cm), followed by medium size AVMs. Only 4 (10%) patients had large nidus. Though the most common mode of presentation with all the sizes of AVMs was hemorrhage, size itself was not predictive of the incidence of hemorrhage at the initial presentation.

Table 10: Bleed with relation to nidus size

Nidus size	Bled	Unbled	No. Of pts.
<3 cm	19(73%)	7(27%)	26 (65%)
>3 <6 cm	8(80%)	2(20%)	10 (25%)
>6 cm	4(100%)	0 (0%)	4 (10%)
Total	31 (78%)	9 (22%)	40 (100%)

(Chi-square test was applied and the size was found not significant in relation to bleed)

Venous Drainage: The deep venous system drainage was seen in 24 (60%) of the patients out of 40, but no statistical difference was seen with the type of drainage in relation to the hemorrhagic presentation of AVM as seen in table 11.

Table 11: Bleed with relation to venous drainage

Venous drainage	Bled	Unbled	Total
Deep	18(45%)	6(15%)	24(60%)
Superficial	13(32.5%)	3(7.5%)	16(40%)
Total	31(77.5%)	9(22.5%)	40(100%)

(Chi-square test was applied and the venous drainage was found not significant in relation to bleed)

Number of Feeders: Though majority (33 patients) of posterior fossa AVMs were fed by multiple feeders, it was not statistically significant to relate the incidence of hemorrhage in posterior fossa AVMs on Chi-square test as shown in table 12.

Table 12: Bleed with relation to number of feeder to AVM

Nos. feeder	Bled	Unbled	Total
Single	5(12.5%)	2(5%)	7
Multiple	26(65%)	7(17.5%)	33
Total	31(77.5%)	9(22.5%)	40

(Chi-square test was applied and the venous drainage was found not significant in relation to bleed).

SPETZLER MARTIN GRADE: Finally the posterior fossa AVMs were categorized with SM garding and Chi-square test was applied. It was found that the relation between SM grade and the probability of hemorrhage at presentation in posterior fossa AVMs was not statistically significant. This is shown in table 13.

Table 13: Bleed with relation to Spetzler Martin grade

Spetzler Martin grade	Bled	Unbled	Total
I	7(17.5%)	1(15%)	8(20%)
II	10(25%)	7(17.5%)	17(42.5%)
III	10(25%)	1(2.25%)	11(27.5%)
IV	2(5%)	0(0%)	2(5%)
V	2(5%)	0(0%)	2(5%)
Total	31(77.5%)	9(22.5%)	40(100%)

(Chi-square test was applied and the SM grade was found not significant in relation to bleed).

Finally, **Multivariate Logistic Regression Model Testing** was applied taking into consideration the various clinical and angiographic parameters like age, sex, AVM size, deep venous drainage, and associated aneurysms with relation to the hemorrhagic presentation in 40 posterior fossa AVMs patients. Out of all the parameters only the AVM size >3cm was found to be statistically significant in predicting higher risk of presentation of AVM with hemorrhage as shown in table 14.

Table 14: The effect of Age, Sex, AVM Size, Deep Venous Drainage, and Associated Aneurysms on Hemorrhagic Presentation

	OR	95% CI	P
Patient age	0.929	0.136–6.334	0.94
Female sex	0.878	0.143–5.337	0.88
AVM size	3.31	1.174–9.344	0.024
Deep venous drainage	3.09	0.335–8.131	1.65
Associated aneurysm	0.527	0.083–3.577	0.545

Seventeen patients out of forty having posterior fossa AVMs underwent single or combined modality of treatment in form of surgery /or embolization /or radiosurgery. Their clinical characteristics, presentation, location of AVM, mode of treatment and outcome are given in table no. 15.

Table 15: <u>Clinical characteristics of 17 patients with arteriovenous malformations managed with surgery / embolization / radiosurgery / combined Rx</u>

Pt. Age (yr)/ Sex	Location	SM Gr	Hemorrhage	Symptom	GCS	Feed-ing Artery	Treat-ment	Follow up GOS & deficits
13/m	Lt. Hemi-sp	3	Parenchymal	Headache, vomiting, LOC, diplopia	15	Sca, pica	Total exc.	GOS-5
19/f	Rt. CPA	3	Parenchymal, IVH	Headache, vomiting, altered sensorium, Dysarthria	10	Sca, pica, aica	Embolzation near total exc.	GOS-4, Rt. 6,7,8, LCN & Rt. Hemi-paresis
6/f	Rt. Tonsillar	1	Parenchymal, IVH	Headache, vomiting,	15	Pica, aica	Total exc.	GOS-5
38/f	Rt. Hemi-sp	2	Parenchymal	Headache, vomiting, LOC	15	Pica, aica	Total exc.	GOS-5
51/m	Vermian	1	Parenchymal, IVH	Headache, vomiting	15	b/l pica, pica aneury	Total exc.	GOS-4,B/L cerebellar signs, staccato speech

39/m	RT. CPA	2	SAH, Serpengin ous contrast enhancement	Headache,	15	Aica, sca, sca-pca aneury	Clip & total exc.	GOS-3 with Rt III B/L cerebellar
46/m	Lt. CPA	2	Parenchymal, IVH, SAH	Headache, Vomiting, LOC, gait unstead, dizziness	15	aica, Pica	Total exc.	GOS – 5
41/m	RT. CPA	2	Parenchymal, IVH	Headache, weaknees, gait ustead, facial numb & deviation	15	Aica, Ba, Ba aneury	Clip & Total exc.	GOS-3 with improved LCN
19/ m	Rt. Hemi -sp	2	Parenchymal, IVH	Headache, vomiting, alt. sensorium	12	Aica, Pica, b/l va	Embol+ Rt. VP shunt & Re-embol	GOS-5
52/m	Lt. Hemi -sp	2	Parenchymal	Lt. hemifacial spasm	15	Pica, sca, aica, pica aneury	Embol	GOS – 5
51/m	Rt. Hemi -sp	2	Parenchymal	RT. Facial numbness	15	Sca double artery.	Embol	GOS – 4
30/m	Rt. Hemi -sp + dural Com ponent	4	Parenchymal	LOC	15	Aica,pi ca, sca, eca	Hemat oma evacu + embol	GOS 4

45/m	Rt. Hemi –sp + dural Com pone nt	3	Parenchymal	Headache, vomiting, LOC, dizziness	15	Aica,pi ca, sca, eca, aica aneury	Embol	GOS – 5
34/m	Brain stem	2	Dorsal BS	Headache, diplopia, vertigo	15	Sca	Embol	GOS – 3
28/m	Lt. Hemi -sp	3	Parenchymal,	Headache, vomiting, LOC, speech difficulty, gait unstead.	15	Pica, aica	Embol + B/L EVD	Expired
30/m	Rt. Hemi -sp	2	Parenchymal	Headache, Vomiting, LOC	15	Pica	Embol + GK	GOS – 4
23/m	Lt. hemi- sp	5	Parenchymal	Headache, vomiting, Seizure, diplopia hearing loss, gait unstead, facial numb 7 deviation	15	Aica, sca, b/l pica	RT	GOS – 4

SM Gr – Spetzler Martin grade, Hge- Haemorrhage
GCS – Glasgow comma scale, GOS – Glasgow outcome scale,
Rt. – right, Lt. – left, CPA – cerebellopontine angle, LCN – lower cranial nerves
SAH – subarachnoid hemorrhage, IVH – intraventricular hemorrhage,
aneury. – aneurysm, LOC – loss of consciousness, unstead – unsteadiness,
exc. – excision, embol – embolization, RT – Radiotherapy, GK – Gamma knife
Rx – management, EVD – external ventriculostomy drainage,
aica - anterior inferior cerebellar artery, eca - external carotid artery; pica - posterior inferior cerebellar artery; sca - superior cerebellar artery, ba – basilar artery,
Va – vertebral artery, pca – posterior cerebellar artery, b/l - bilateral supply

RIGHT CPA AVM

Plate I

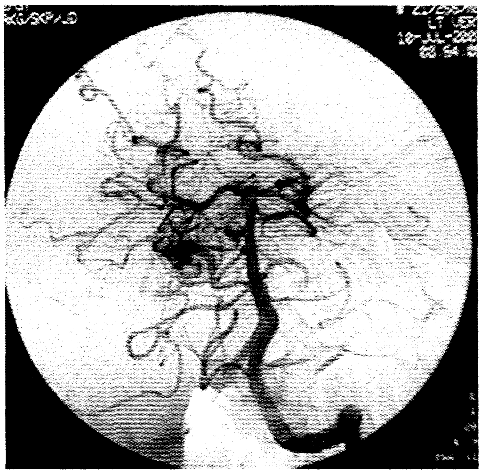
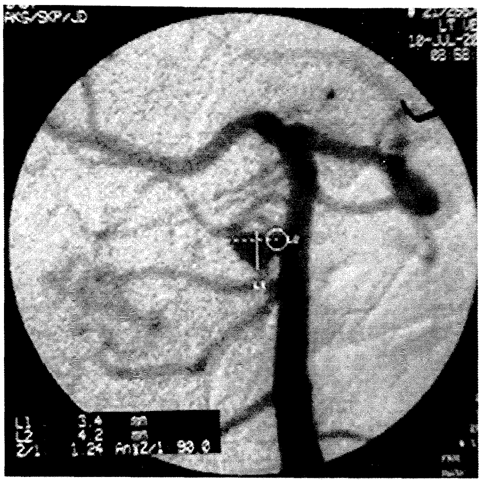


Plate II



<u>Plates I & II</u> : Preoperative DSA of 39 yr old male presenting with SAH, showing Rt. CPA AVM (SM Gr2) with feeders from Rt. SCA & AICA with SCA – BA junction aneurysm.

Plate III

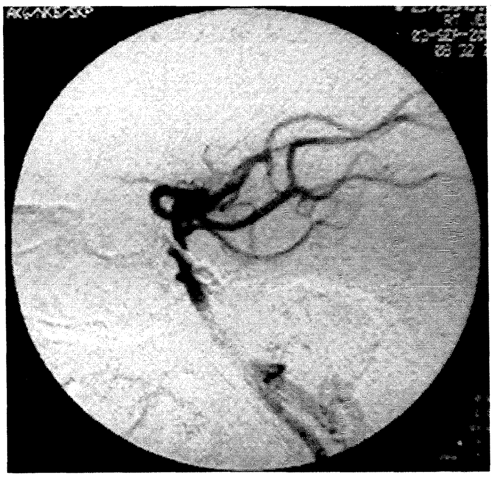
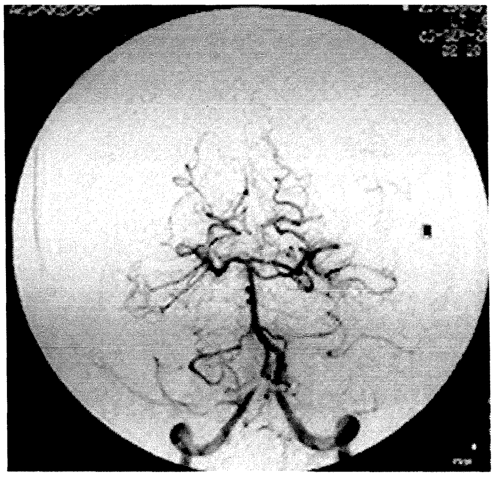


Plate IV



<u>Plates III & IV</u> : Postoperative DSA showing no residual AVM / aneurysm after clipping & total excision.

RIGHT HEMISPHERE AVM

Plate V

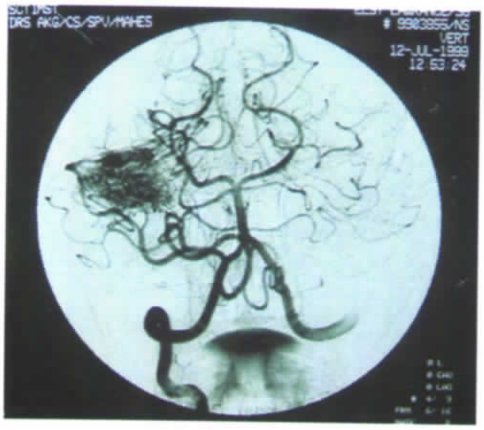
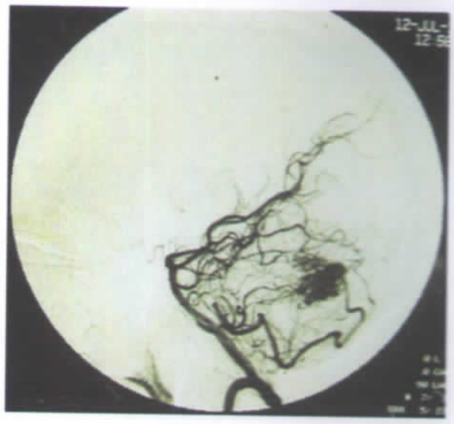


Plate VI



Plates V & VI : Preoperative DSA of 38 yr old female presenting with Rt. Cerebellar bleed, showing Rt. hemispheric AVM (SM Gr2) with feeders from Rt. PICA & AICA.

Plate VII

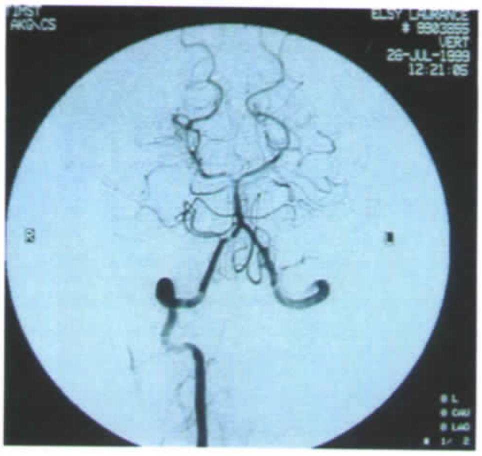


Plate VIII

Plates VII & VIII : Postoperative DSA showing no residual AVM after total excision of AVM.

RIGHT TONSILLAR AVM

Plate IX

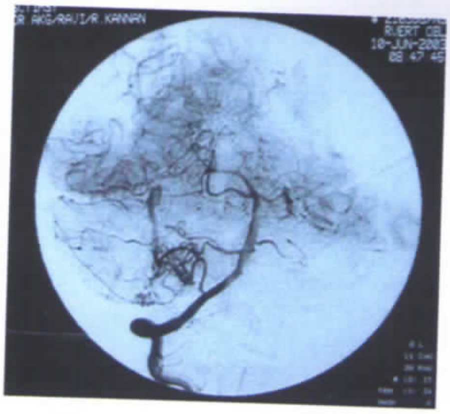


Plate X

Plates IX & X : Preoperative DSA of 6 yr old female presenting with IV ventricular bleed, showing Rt. Tonsillar AVM(SM Gr1) with feeders from Rt. PICA & AICA.

Plate XI

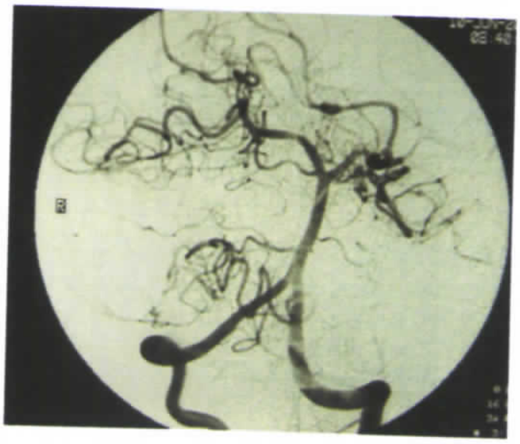
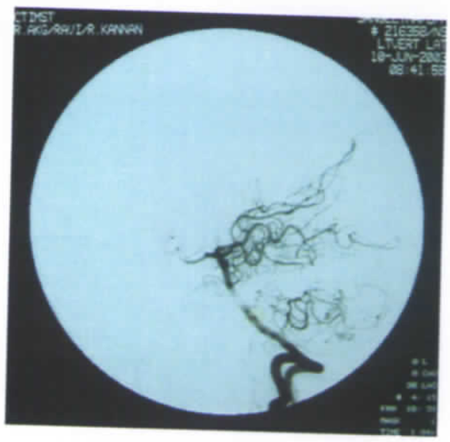


Plate XII



Plates XI & XII : Postoperative DSA showing no residual AVM after total excision of AVM.

LEFT CPA AVM

Plate XIII

Plate XIV

Plates XIII & XIV : Preoperative DSA of 46 yr old male presenting with Rt. Cerebellar bleed & IVH showing Lt. CPA AVM(SM Gr2) with feeders from Lt. PICA & AICA.

Plate XV

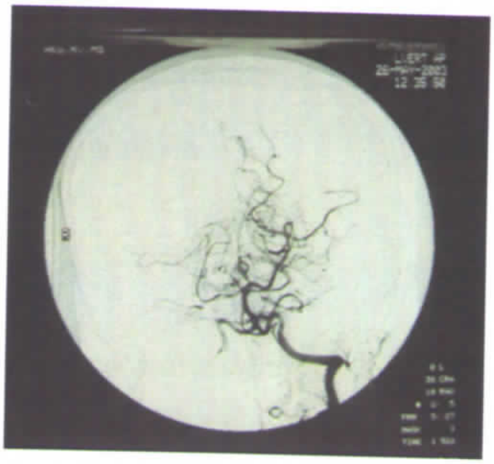
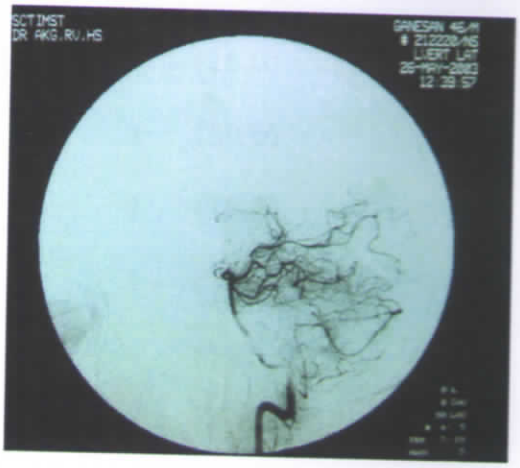


Plate XVI



Plates XV & XIV : Postoperative DSA showing no residual AVM after total excision of AVM.

SURGICALLY MANAGED PATIENTS

Out of the total 40 patients 8 patients had underwent definitive treatment in form of surgery and excision of posterior fossa AVM after determining the patient selection criteria as described above in review on basis of location of AVM and its characteristic along with clinical condition of the patient. These patients were further analyzed separately for their demographic and clinical characteristics as given in table no 16.

Table 16: Patients characteristics managed surgically

Characteristics	Total (n=8)	
Average age	31.6yr	
Sex:		%
Male	5	(62.2)
Female	3	(37.5)
Hemorrhage on presentation	8	(100)
Neurological signs	4	(50.0)
Eye movement disturbance	2	(25.0)
Cerebellar sign/ataxia	4	(50.0)
Paresis	1	(12.5)
Paresthesia	1	(12.5)
Lower cranial palsy/dysarthria	1	(12.5)
Vertigo/tinnitus	1	(12.5)
Facial palsy	1	(12.5)

AVM CHARACTERISTICS

The breakdown of the surgically managed cases by location versus specific AVM characteristics is shown in Table-17. Location wise four out of the eight had AVM in cerebellopontine location, two in cerebellar hemisphere and remaining one each in vermis & tonsil. On the Spetzler-Martin scale, one each of CPA & hemispheric AVMs were grade 3 and rest either grade 1 or 2. Almost all AVMs were small sized. The arterial supply was mostly from the posterior inferior cerebellar artery (PICA) , superior cerebellar arteries (SCAs), and anterior inferior cerebellar artery (AICA). The feeding artery aneurysm was present in 50% of surgically treated patients. Two of these present were associated with cerebellopontine angle AVM and another with vermian AVM. All aneurysms were clipped either along with excision of AVM or during initial surgery for aneurysmal bleed as in one case of cerebellopontine angle AVM. The venous drainage was into superficial system in five and to deep venous system in three patients.

Table 17: A breakdown of 8 cases managed surgically by location versus specific AVM characteristics

Location	Vermis (n=1)	Hemisphere (n=2)	Tonsil (n=1)	CPA (n=4)
SM grade				
V	0	0	0	0
IV	0	0	0	0
III	0	1	0	1
II	0	1	0	3
I	1	0	1	0
Nidus size(cm)				
>6	0	0	0	0
3-6	0	0	0	1
<3	1	2	1	3
Arterial feeders				
SCA	0	2	0	2
AICA	0	1	1	3
PICA	1*	2	1	2
BA	0	0	0	1
ECA	0	0	0	0
PCA	0	0	1	0
Feeding artery aneurysm				
Present	1	1	0	2
Clipped	1	0	0	2
Venous drainage				
Deep	0	2	0	1
Superficial	1	0	1	3

AICA, anterior inferior cerebellar artery, CPA, cerebellopontine angle; ECA, external carotid artery; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery. * bilateral supply

MANAGEMENT

As evident by table-18, majority of patients (57.5%) with posterior fossa AVMs who were initially advised to undergo embolization or in a selected few direct surgery did not turn up for treatment or refused treatment. Out of the remaining 17 patients, 8 underwent excision of AVM. Two patients were embolized before taking up for surgery, one of them located in cerebellar hemisphere with nidus size >3cm & SM grade III and another was located in vermis with bilateral supply from PICA where the dominant PICA feeder was embolized. The remaining 6 patients underwent direct surgery. Total excision was achieved in 7 out of 8 patients and remaining one had near total excision which was confirmed on DSA. Two patients with posterior fossa AVM; one with the cerebellar hematoma underwent evacuation of the hematoma and other who had presented with SAH underwent clipping for basilar artery feeder aneurysm. First was referred for stereotactic radiosurgery (SRT) and other refused surgery for AVM and was lost to follow-up. Another patient a 30yr old male with right cerebellar grade IV AVM had emergency evacuation of hematoma outside and then referred to us underwent embolization.

Total 7 patients underwent embolization and another two radiosurgery. Most of patient undergoing embolization was having AVM in hemispheric location and either SM grade I or II and in none cure was achieved. One patient with grade III hemispheric AVM had intranidal rupture of aneurysm during embolization and expired, while in another procedure had to be abandoned due to technical reasons. Both patients who had undergone radiosurgery had hemispheric AVMs. One had diffuse nidus and initially underwent embolization (95% obliteration of nidus) followed by Gamma knife and had complete obliteration of nidus after combined treatment.

Other had SM grade V AVM and had undergone primary radiosurgery with check DSA showing residual AVM.

Table 18: **<u>Modality of treatment used</u>**

Surgery	**No. of Patients (n=40)**
Primary resection	5
Staged resection	1
Embolization and resection	2
Embolization	7
Radiosurgery	2
No treatment &	
Lost to follow-up	23

<u>OUTCOME</u>

The patients eight operated had average follow-up of two and half years with minimum follow-up of 1year. As shown in table-19, four (50%) patients had a good outcome and two (25%) patients each had moderate to severe disability. Three of the patients showed improvement in their grade on follow-up compared to that at discharge. One patient on presentation had poor GCS and made good improvement after surgery and another who presented with hemiparesis did make recovery but still was dependent till the last follow-up.

Table 19: Outcome of patients who underwent excision of AVM

Pt. no	Site	GCS	SM Gr	Postop. Complications/ deficits	GOS at discharge	Follow-Up	GOS on follow-up & present status
1	Lt. Hemisp	15	2	Nil	5	8mths	5
2	Rt. CPA	10	3	Rt. VI, VII & LCN, Rt. hemiparesis	3	4yr	4
3	Rt. Tonsil	15	1	Nil	5	3yr	5
4	Rt. Hemisp	15	2	Nil	4	7yr	5
5	Vermian	15	1	b/l cerebellar sign, cerebellar dysarthria	3	2yr	4
6	Rt. CPA	15	2	Rt. III, LCN, b/l XII, developed haematoma – evacuated	3	1yr	3, Rt. III, LCN & dysarthria
7	Rt. CPA	15	2	Preop Rt. V, VII, LCN paresis & Lt. hemiparesis accentuated, had stridor, on tracheostomy	3	1yr	3, Rt. III, off tracheostomy
8	Lt. CPA	15	2	Lt. VII, VIII & LCN	5	3yr	5, Lt. V, VII & LCN function improved

SM Gr – Spetzler Martin grade, GCS – Glasgow comma scale, GOS – Glasgow outcome scale, Rt. – right, Lt. – left, CPA – cerebellopontine angle, LCN – lower cranial nerves, exc. – excision.

NEUROLOGICAL OUTCOME BY GOS

Finally, from the outcome of total 17 managed cases there was only single mortality in embolized group (table 20). Rest of the patients in embolized group had outcome comparable to surgically managed patients, though none achieved cure in the embolized group. Patients in radiosurgical group had moderate disability and in a single patient who had undergone embolization followed by Gamma knife cure was achieved.

Table 20: Neurologic Outcome of seventeen cases managed

GOS*	Surgical Grp (n = 08)	Embolization Grp (n = 07)	Radiosurgical Grp (n = 2)
GR	4(50%)	3(43%)	0
MD	2(25%)	2(28.5%)	2(100%)
SD	2(25%)	1(14.25%)	0
VS	0	0	0
DEAD	0	1(14.25%)	0

Discussion

DISCUSSION

In our series, we treated 17 patients utilizing surgery, radiosurgery, and endovascular techniques and achieved successful cure in 7 surgically managed patients and one more with combined therapy - embolization followed by radiosurgery.

The average age of presentation in this study was 32 years and is within the range as quoted in other studies, i.e. between 20 and 40 years of age.[33-51] Stoodly .et.al treated 53 patients of AVMs at Stanford Medical Center from 1985 to 1998. Patients ranged in age from 5 to 64 years (29±14).

We further found that incidence of posterior fossa AVMs is nearly twice in the male sex compared to female sex. This is against the literature reports of equal gender predilection[27,40] or slightly male predominance.[33-36,44] Although Multiple AVMs have rarely been described,[8,49] we had three patients of posterior fossa AVM with an associated AVM in suprasellar region. One was in parietal region and other two in occipital and suprasellar region.

Natural History: As with any procedure, the rational use of any therapeutic procedure must entail less risk than the natural history of the primary disease. With respect to posterior circulation AVMs, one might imagine that this would be hard to demonstrate in young patients. The data, however, suggest otherwise. Multiple recent analyses of unruptured AVMs have shown an annual rupture risk of 2% to 4%.[37,44,76] The mean age of our AVM patients would place them at approximately a 73% lifetime risk of rupture. With our own data, a good recovery/moderate disability outcome at hospital discharge occurred in 64.5% of patients who presented with hemorrhage and in 87.5%% of patients who presented with neurologic deficits. Thus, at least 35% of patients with AVMs that bleed can expect to be neurologically devastated by their lesion. The case for considering treatment of posterior circulation AVMs is therefore evident.

CLINICAL PRESENTATION

As shown in literature, this study too proves that hemorrhage is the most common presenting feature of posterior fossa AVM along with symptoms related to elevated intracranial pressure i.e. headache, nausea, vomiting, dizziness, altered sensorium etc.

Haemorrhage: The majority of patients in this series presented with intracranial hemorrhage. In particular, we observed intraparenchymal hemorrhage in 31(77.5%) patients followed by 16 (40%) patients presenting with intraventricular hemorrhage. Subarachnoid hemorrhage was found in 7(17.5%) patients. Many studies have shown that intraparenchymal hemorrhage is most common after rupture of posterior fossa AVM, followed by intraventricular and subarachnoid hemorrhage.[4,73,74]

Similar results showing a high incidence of hemorrhage associated with posterior fossa AVMs have been reported by Lesnaik et.al., where majority of patients (63%) with posterior fossa AVMs presented with intracranial hemorrhage. Drake et.al.,[3] reported a hemorrhage rate of 84% in their series, whereas Matsumura et.al.,[5] documented hemorrhage in 83% of patients. Similarly, in the Cooperative Study of Subarachnoid Hemorrhage and Intracranial Aneurysms, intracranial hemorrhage was the presenting sign in 72% of infratentorial AVMs.[1] In the same study, 66% of supratentorial AVMs were also associated with hemorrhage. Thus, it would appear that initial hemorrhage is as likely to occur in cases of supratentorial as infratentorial AVMs. Furthermore, studies of the natural history of AVMs suggest that the rate of rebleeding is likely similar in both locations,[7–12] although the prevention of new hemorrhage in the posterior fossa is likely to be more important. In current study those patient who refused treatment were lost to follow-up and hence, rebleeding rates could not be commented upon. In addition to hemorrhage, AVMs of the posterior fossa may also present with other symptoms as described below.

Headache: Headache was common presenting compliant in 75% of the patients with AVMs, associated with vomiting in nearly 50% of them. Out of these 15% patients had headache even in the absence of hemorrhage similar to published reports in other studies. [46]

Neurological deficit: In our series, 65% patients had presented with both transient loss of conciousness or in altered sensorium and few with transient or progressive neurological deficits. Eight (20%) patients presented altered sensorium, five (12.5%) with weakness, nine (22.5%) with ataxia, four (10%) with diplopia, four (10%) with dysarthria and two (5%) with dysphasia, three (7.5%) each with dizziness, facial numbness/spasm, facial deviation and one (2.25%) with hearing loss. In review of literature less than 10% of patients present with transient, permanent, or progressive neurological deficits not ascribed to hemorrhage or seizure.[33,36,46,71] Neurologic deficits most often presenting as a mild cerebellar syndrome have been seen in up to 30% of the patients.[3,4,6,75] Lesniak et.al., found that 37% of the patients with posterior fossa AVMs had neurological signs related to brainstem and/or cerebellar dysfunction at initial presentation. Chronic headache, vertigo, and ataxia were also reported by a significant number of their patients.

Seizures: Though 15% to 35% of patients with supratentorial AVMs present with seizures,[27,30,31,36,37,39,46] seizure as the presenting symptom of a cerebellar AVM is rare and was seen in 3(7.5%) of our cases. Out of these patients, one patient had hydrocephalus and one had a cerebellar AVM. The third patient presented with an occipital AVM which was embolized and on follow-up DSA had incidentally detected cerebellar hemispheric AVM.

Risk factor for Hemorrhagic presentation:-

Various AVM characteristic has described linked to risk for hemorrhage. Size of AVM nidus is a controversial factor. Guidetti and Delitala[18] reported that 89% of patients with small AVMs presented with hemorrhage, compared with 74% of patients with medium sized AVMs and 58% of large sized AVMs. In our study 73% of patients with small; 80% of medium and 100% of large AVMs presented with hemorrhage. In the current study relation of AVM size to hemorrhagic presentation did not reach statistical significance. Several large studies, including one of unruptured AVMs, found no difference in hemorrhage risk based on the size of AVM.[17,35,42,44,46,80] Others found that small AVMs(<3cm) pose a higher risk of hemorrhagic presentation.[37-39,43,45,72,84-86] Though deep venous drainage of AVM has been frequently linked with increase in the incidence of hemorrhage,[17,38,79,80,84,88,89] in our study out of total 31(77.5%) patients presenting with hemorrhage, deep venous drainage of AVM was present in 58% of the patients. No significance could be established between risk of hemorrhage with deep venous drainage. The Columbia AVM study group examined a large number of physiologic indices in 449 patients and the multivariate analysis revealed that size and deep venous drainage, were independent risk factors for bleeding.[90] In a multivariate model by Khaw. et.al.[92] AVM size, deep venous drainage, and the presence of AVM-associated arterial aneurysms were significantly associated with hemorrhagic AVM presentation. In our study, the patients presenting with hemorrhage had AVMs fed by multiple feeders in 84% (26 cases) as compared to only 16% (5 cases) fed by a single feeder.

AVM and aneurysm: The relationship between AVMs and aneurysms has been well established.[27,37,59,79,93-100] In our study, 12(30%) patients had an aneurysm associated with AVM. Depending on the series, 2.7% to 23% of patients have an aneurysms present with AVMs.[27,37,59,93,100] Miyasaka and coworkers found that the mean age of presentation was 41 years in those with aneurysms, versus 31 in those without.[94] In this study the mean age of patients with aneurysm was 43 years, while those without the aneurysm were 26. Also the aneurysms were three times more common in the males as compared to their female counterparts. While studies quote male to female ratio similar to that of entire AVM population.[98] We also saw that among 40 patients with 12 aneurysms, hemorrhage was the present in 77.5%. Seventy four percent of these patients had the hemorrhage related to AVM, 9.7% secondary to aneurysm, and in 16% the site of hemorrhage was unclear. Among 39 patients with 64 aneurysms, Cunha and colleagues found that intracerebral hemorrhage was the presentation in 63%.[97] Forty six percent of these bleeds were secondary to aneurysm, 33% were related to the AVM, and in 21% the site of hemorrhage was unclear. Majority of aneurysms are flow related.[27,59,79,94,95,97] In the current study all except one had distal feeding artery aneurysm. Only one had intranidal aneurysm. The frequency of distal, feeding artery aneurysms ranges from 37% to 69%[27,59,79,94,101] and intranidal aneurysms represents approximately 20% of aneurysms,[79] but this is variable, based on type of study performed.

Spetzler Martin grading: In this study of 40 patients with AVMs, 25(62.5%) patients were Spetzler-Martin(SM) grade I-II, 11(27.5%) patients were SM grade III, and 4(10%) patients SM grade IV-V. Also out of 31 patients presenting with hemorrhage, 17(54.8%) of the patients were SM Gr I-II, 10(32.25%) patients SM Gr III and 4(12.9%) patients SM GR IV-V. Independent researchers established, however, that the Spetzler-Martin garding variables are significant in influencing surgical outcome.[150,151] In our study the sample size of the patients undergoing surgical management for posterior fossa AVMs was small, therefore the statistical significance of Spetzler Martin grade to the outcome could not be commented upon. We further classified posterior fossa AVMs as located in the cerebellar vermis, cerebellar hemisphere, cerebellar tonsil, brain stem and in cerebellopontine angle (CPA). The location wise distribution of AVMs, cerebellum hemisphere including vermis (excluding tonsils, brainstem and CP angle) constituted the majority – 28(70%) cases and same has been reported in other studies.[3,4,7,52,152]

In the current study, large sized AVMs (>6cm) were present in 4(10%) of patients, all in the cerebellar hemisphere and constituting SM Gr IV-V. In the remaining locations, the most common grade was II(40%) constituting majority involving the cerebellar hemisphere and CP angle. Septzler Martin Gr-III AVMs were seen in 11(27.5) patients, four each in brainstem (majority) and cerebellar hemisphere, 2 in the vermis and one in CP angle. The medium- sized AVMs (3–6 cm) were present in 10 of the patients (25%). In the brainstem, however, all of AVMs were small (<3 cm). A similar breakdown of the cases by Lesniak, et.al[2] on location wise versus specific AVM characteristics showed 7 of 8 patients with vermian AVMs presented with grade IV lesions. In the remaining locations, the most common grade was II (40%). Medium-sized AVMs (3–6 cm) were present in 21 of the patients (55%). In the brainstem, however, the majority (75%) of AVMs were small (<3 cm).

Also the arterial supply of AVMs in majority of cases (especially in cerebellar AVMs) in the current study was from posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA). Brainstem AVMs were mainly supplied by SCA and AICA was the major supply in cases of CP angle AVMs. Vermian AVMs had bilateral supply from both superior cerebellar arteries (SCAs), or from the posterior inferior cerebellar artery (PICA) with occasional contributions from the anterior inferior cerebellar artery (AICA). In few cases directly from basilar artery (BA), vertebral artery (VA), or posterior cerebral artery (PCA), and four patients had dural component supplied by pharyngeal branch or occipital branch of external carotid artery (ECA). The most common venous drainage occurred directly into the deep venous system, with others emptying into the superficial venous system.

MANAGEMENT

In our study treatment for seventeen patients with posterior fossa AVMs included embolization, radiosurgery, and/or microsurgery. Preoperative embolization was used in two patients. One was located in CP angle had a diffuse nidus with size >3 cm while the other one was a vermian AVM having bilateral PICA supply with multiple arterial feeders and size <3cm. Seven more patients were treated by embolization alone with majority being SM grade I or II AVM in hemispheric location and in none complete cure could be achieved.

The combination of intraoperative embolization with surgical resection has been successful in the treatment of large cerebral AVMs. For instance, Zhao et al[155] treated 50 patients with giant AVMs by means of preoperative embolization and surgical resection and found excellent results in 88% of the patients. Similarly, Hongo et al[156] treated 27 cases of giant AVMs with preoperative embolization and found good results in 70% of the patients. When used alone, however, embolization has shown limited success and that only in the treatment of small AVMs.[160-165] Moreover, incomplete embolization is associated with worse clinical outcome as well as the potential for increased neovascularization.[164,165] Our results further corroborate the experience of others and help to reinforce the role of embolization in the management of patients with posterior circulation AVMs.

Radiosurgery represents a viable treatment modality for patients with inaccessible or small AVMs. Stereotactic radiosurgery has been widely applied to cerebral AVMs with a diameter of 3 cm or less and an obliteration rate of 65 to 85% is expected with no definite effect on bleeding rates during latency periods[166,169-172,174] In our series, two patients with hemispheric AVM had underwent radiosurgery. One patient had a diffuse nidus and initially underwent embolization (95% obliteration of nidus) followed by Gamma knife resulting in complete obliteration of nidus after combined modality of treatment. Other patient with SM grade-V AVM underwent primarily radiosurgery and repeat DSA showed residual AVM. We observed no permanent neurologic deficits caused by radiosurgery. None of them had rebleed during their followup period of 7-8 years. Although our results with respect to the obliteration rate and rebleeding rate are better than those published in the literature (50%–70% at 3 years),[64,166] we believe that this discrepancy can be explained by the relatively small number of patients treated with radiosurgery in our series as well as the extended follow-up time. Nevertheless, when chosen appropriately, radiosurgery can provide a useful adjunct in the management of posterior fossa AVMs.

The overall outcome was satisfactory in the majority of embolized and radiosurgically treated patients, but for a single mortality caused by intranidal rupture of aneurysm during embolization and one patient developed hemiparesis along with cerebellar dysfunction.

We operated upon eight properly selected AVMs within the posterior fossa, achieving total excision in 7 and near total in one patient. Total excision was performed in 3 cerebellopontine angle, 2 cerebellar, 1 vermian & 1 tonsillar AVM, while in one patient with cerebellopontine angle AVM only near total excision was achieved. Out of 4 patients having cerebellopontine angle, 2(50%) patients had good outcome and in other 2(50%) patients had severe disability. Solomon and Stein[10] reported nine surgically treated cases out of 12 patients with brainstem AVMs, including four dorsal midbrain, five pons, and three cerebellopontine angle AVMs. In this series, total resection was accomplished in two dorsal midbrain, four pons, and three cerebellopontine angle AVMs with a 22% morbidity rate. Drake et al[4] described total resection in one dorsal midbrain AVM, two pontine hemorrhage with cryptic vascular malformations, and seven cerebellopontine angle AVMs, with partial resection in one primarily extrapial ventral midbrain AVM extending into posterior perforated substance, in 15 brainstem and seven cerebellopontine angle AVMs. Five patients died related to the surgery. Batjer and Samson[1] described six surgically resected cases (four pons and two cerebellopontine angle AVMs) with single mortality. Recently, Lawton et al.[168] reported that eight brainstem AVMs were successfully resected with no serious neurological deterioration. They stated that brainstem AVMs typically reached a subarachnoid surface that could be accessed through several surgical approaches, but the location of the nidus was not described. Considering the reported cases and series by Kazuhiko Nozaki et.al[167], AVMs involving the brainstem are candidates for surgical extirpation when the nidus locates mainly sub or extrapially, such as dorsal midbrain AVMs and cerebellopontine angle AVMs. However, when most part of the nidus locates within the brainstem, considerable morbidity should be taken into account particularly in brainstem AVMs without intraparenchymal hemorrhage.

In our study, good results were obtained in 6(75%) of the surgically patients with acceptable morbidity in the remaining 2(25%) patients. A large surgical exposure, microsurgical dissection, and preservation of main drainage until total control of the nidus has been achieved all contribute to achieving complete and safe resection. Although postoperative hemorrhage accounts for most of the operative morbidity in AVM surgery, we have experienced this complication in only 1 patient (12.5%). Our patient subsequently made some recovery with persistence of lower cranial nerve paresis and cerebellar dysfunction following removal of the clot. This is within the range of 8% to 10% reported by Drake et al·[3]There was no operative mortality.

Finally, our cumulative morbidity (11.75%) and mortality (5.8%) rates reflect the increased risks associated with neurovascular lesions of the posterior fossa.

Conclusion

CONCLUSION

The AVMs of the posterior fossa are rare occurrence and surgery for these malformations is very challenging. The initial presentation, location, size, and grade of the AVM all play an important role in determining the most appropriate management and the need for neurosurgical intervention.

The current treatment modalities for posterior fossa AVMs include embolization, radiosurgery, and microsurgery. Embolization is often beneficial in reducing the vascularity of an AVM, reducing the size of nidus, and it often used preoperatively to minimize the risk of intraoperative bleeding. When used alone, however, embolization has shown limited success. Radiosurgery represents a viable treatment modality for patients with inaccessible or small AVMs, especially located in brainstem.

As in the case of supratentorial AVMs, surgery remains a safe treatment of choice for select lesions within the posterior fossa. Indeed, it may represent the most effective treatment for large AVMs that present with or without hemorrhage and for AVMs that lie outside the brainstem and are easily accessible via surgical exploration. Our results reinforce the literature evidence that properly selected cases of posterior fossa AVMs can be managed successfully with acceptable morbidity and mortality.

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Appendix

Appendix 1

PROFORMA

HOSPITAL No: …………… **NAME** :………………………………… **AGE**…… .**SEX**: M/F

<u>DIAGNOSIS:</u> **Inftratentorial** **Side**: Right / Left.
Posterior fossa: Vermian / Hemispheric / Tonsil / CPA / Brain stem / complex

<u>PRESENTING FEATURES</u>:

Bleed: Single / Recurrent (no:……...)
Headache: Y/N
Seizures: Single / Recurrent
Others:

<u>CLINICAL EXAMINATION</u>:

GCS: ……...: E…..M…..V….. = / 15
Cranial nerve deficits:
Lower cranial nerve:Y/N Facial: Y/N Vestibulocochlear: Y/N Others:
Motor deficits: Hemiparesis / Hemiplegia / Paraparesis / Paraplegia / Monoplegia / Monoparesis
Sensory deficits
Cerebellar signs
Others

<u>INVESTIGATIONS</u>:

<u>CT SCAN</u>:
Bleed: Size……….. Site……………………
Intraventricular: Y/N Serpigenous vessels Y/N Hydrocephalus Y/N
Mass effect: Y/N Midline shift: Y/N Aneurismal sac/ Venous sac Y/N. CTA Y/N

<u>MRI SCAN</u>:
Exact Location……….. Size :
<u>MRA</u>: Y/N

<u>DSA</u>:
Size…………..
Feeders: Single / multiple Superficial/ Deep Size of feeders: Normal / Enlarged
Aneurysm: Y/N Feeding artery / nidus No:
Draining vein : Superficial / Deep
Draining into: Sagital sinus / Transverse sinus / Sigmoid sinus / Straight sinus / Vein of galena / Basal vein / Cavernous / Galenic / Petrosal sinus
Venous sac Y/N
Nidus type : Compact / Diffuse Aneurysm: Y/N
Arterial Territory : SCA (……………..) / AICA(…………….) / PICA(……………..) / ECA(………..) / Vertebral / PCA(……………..) / ICA(……………..) / Others

SPETZLER MARTIN GRADE: 1/2/3/4/5/6

(contd.)

MANAGEMENT:

Embolization / Surgery / Radiosurgery / No intervention
Combined (Radiosurgery & Surgery / Surgery & Embolization / Embolization & Radiosurgery / All three)

Embolization: Y/N
No. of feeders embolized:
Postembolization Sequelae:

Material used: NBCA / IBCA / PVC / COIL
Post Embolization status (Percentage):

Surgery: Y/N
Procedure: Craniotomy / craniectomy: …………… / Approach: SOC / RMC / Others
Total / Partial / Subtotal
Management of *Aneurysm:* embolization / clipping
Radiotherapy: Y/N Radiosurgery / Gamma knife

POSTOP COURSE:

GCS Score:
GOS: I / II / III / IV / V

Fresh deficits:
Cranial nerve deficits:
Motor deficits:
Sensory deficits:
Cerebellar signs:
Others:

FOLLOW UP:

GOS: I / II / III / IV / V
Deficits:………………

Postoperative DSA: Residual / NIL

Management of Residual lesion: Resurgery / embolization / Radiotherapy

MASTER CHART

s no	hosp no	date	name	age	sex	side	site	head-ache	vomi-ting	alter sens	LOC	seizures	weak-ness	gait unsteadi-ness	diplo-pia	hearing loss	facial numbness	facial deviation	dizzi-ness	other	GCS	cran nerv	nystag-mus	motor deficit	sensory	cerebell	others	CT bleed	site	SAH	intra-ventricular	serpinginous vessel	hydro-cephalus	mass effect	ve sa
1	244229	3/10/2005	moncy	13	m	Lt	hemis	y	y	n	y	n	n	n	y	n	n	n	n	n	15	Lt. LR	-	-	-	Lt	-	y	Lt. cerebll	n	n	n	n	y	n
2	205546	12/7/2002	remya	19	f	Rt	CPA	y	y	y	n	n	n	n	n	n	n	n	n	dysphasia	10	-	-	-	-	-	neck stiff	y	Rt. Cerbll	n	y	n	y	n	n
3	216358	7/6/2003	sangeetha	6	f	Rt	tonsil	y	y	n	n	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	-	n	y	n	y	n	n
4	9903855	9/7/1999	elsy	38	f	Rt	hemis	y	y	n	y	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	Rt. Cerbll	n	y	y	y	n	n
5	9901459	29/4/99	sindhu	23	f	Rt	BS	y	n	y	n	n	n	n	n	n	n	n	y	n	12	b/l 3 , Rt 7	-	-	-	-	neck stiff	y	-	n	y	n	n	n	n
6	9900023	10/4/2000	saji anderson	30	m	Rt	hemis+tonsil	y	y	n	y	n	n	n	n	n	n	n	n	n	15	-	y	-	-	bl	-	y	Rt. Cerbll	y	y	y	n	n	n
7	184253	14-7-00	balakrishna	51	m	mid	verm	y	y	n	n	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	verm	y	y	n	y	n	n
8	180221	19-2-00	raghavan	51	m	mid	verm	n	y	n	n	y	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	verm	n	n	y	y	n	n
9	9801357	13/2/98	shylaja	45	f	Rt	hemis	y	n	n	y	n	n	n	n	n	n	n	n	n	15	b/l 6	-	-	-	-	neck stiff	y	-	y	n	n	y	n	n
10	9508604	11/4/1996	mohamm.	38	m	Lt	hemis	y	n	y	n	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	Lt. cerebll	n	n	n	n	n	n
11	9705082	5/8/1997	unni	16	m	Lt	hemis	n	y	n	n	n	n	n	n	n	n	n	n	vertigo	15	-	-	-	-	-	-	y	Lt cerebelll	n	n	y	n	n	n
12	9801720	16/9/04	saji S	23	m	Lt	hemis	y	y	n	n	y	n	y	y	y	n	y	n	n	15	Lt. 6,7,8b/l lcn	y	-	-	Lt	-	y	Lt. cerebll	n	n	y	n	n	n
13	9802340	10/8/1998	sasidharan	51	m	Rt	hemis	n	n	n	n	n	n	n	n	n	y	n	n	pontine haematoma	15	Lt. V 1,2,3	-	-	-	Lt	-	n	Rt cerbell	n	n	n	n	n	n
14	234769	3/12/2004	bhaskaran	44	m	Lt	hemis	y	y	n	n	n	n	y	n	n	n	n	n	n	15	Lt. 6,7umn	-	-	-	axial	neck stiff	n	-	n	n	n	n	n	n
15	203328	4/5/2004	nisha	13	f	Rt	hemis	y	n	n	y	n	n	n	n	n	n	n	n	n	9	-	-	-	-	-	neck stiff	y	Rt. Paravermian	n	n	n	n	n	n
16	206592	8/10/2002	donna mary	15	f	Lt	hemis	y	y	n	y	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	Lt cerebelll	n	n	n	y	n	n
17	9408648	10/3/1995	raveendram	42	m	Rt	hemis	n	n	n	y	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	-	y	y	y	y	y	n
18	191932	1/4/2001	anasmon	19	m	Rt	hemis	y	y	y	n	n	n	n	n	n	n	n	n	n	12	Rt ptosis	-	-	-	-	neck stiff	y	Rt Cerebell	n	y	y	n	n	n
19	195595	3/9/2001	abdul	38	m	Lt supra/infra	hemis+Rt. Pariet	y	y	n	n	n	n	n	n	n	n	n	n	gen weak	15	-	-	-	-	-	-	y	Rt. Parit/Lt cerebell	n	y	y	n	n	n
20	9906947	25/4/2000	babu	28	m	Lt	hemis	y	n	n	y	n	n	y	n	n	n	n	n	dysarthria	15	-	y	-	-	Lt.	-	y	Lt cerebelll	n	n	n	n	n	n
21	215400	4/2/2004	dhanushodi	52	m	Lt	hemis	n	n	n	n	n	n	n	n	n	Lt hemifacial spasm	n	n	n	15	lt ptosis, 7 lmn	y	-	-	Lt.	-	y	Lt cerebelll	n	n	n	n	n	n
22	217992	11/8/2003	shanmugam	39	m	Rt	CPA	y	n	n	n	n	n	n	n	n	n	n	n	n	15	-	-	-	-	b/l	-	y	Rt. Cerebell	y	n	y	n	n	n
23	217255	4/7/2003	balan	41	m	Rt	CPA	y	n	n	n	n	y	y	n	n	y	y	n	dysarthria	15	3,5,7,8,lcn	y	Lt Hemi 3/5	Lt hemisen all	b/l	-	y	pontine avm -ext to rt. Cpa	y	y	n	n	n	n
24	212220	30/4/04	ganesan	46	m	Lt	CPA	y	y	n	y	n	n	y	n	n	n	n	y	vertigo	15	-	y	-	-	Lt.	-	y	Lt cerebell	n	y	y	y	n	n
25	211836	22/1/03	bhooma	34	f	Rt	hemis	y	y	n	y	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	-	y	Rt cerebel	n	n	y	n	n	n
26	211817	20/1/03	indu	14	f	Lt	hemis	y	y	n	y	n	n	n	n	n	n	n	n	lt. cereb hem/ evc+vp shunt	15	-	-	-	-	Lt.	-	y	Lt cerebell	n	n	n	y	n	n
27	209428	8/11/2002	mercy	11	f	-	BS+suprsell	y	y	n	y	n	n	n	n	n	n	n	n	n	15	-	y	-	-	-	-	y	Rt. tect+midd cereb ped	n	y	n	y	n	n
28	195212	26/9/2001	santhosh	34	m	-	BS	y	n	n	n	n	y	n	y	n	n	n	y	vertigo	15	b/l ptosis, Rt 4	-	impr no wek	-	-	-	y	dors BS	n	y	n	n	n	n
29	195167	6/8/2001	chenthil	31	m	Lt	CPA	y	n	n	n	n	y	n	n	n	n	n	y	tinit,visual blur,propt Lt eye	15	Lt. 6,papill, Lt tempcut	y	-	-	-	Lt. temp swell	n	Lt. CPA,mass in lat ventr	n	n	y	n	n	n
30	9809911	8/12/1998	sukumaran	47	m	Lt	hemis	y	y	y	n	n	n	n	n	n	n	n	n	n	15	-	-	-	-	-	papilledema	y	-	n	y	-	-	-	-
31	182068	28/3/200	selvi	3	f	Rt	hemis	n	n	y	n	n	n	y	n	n	n	n	n	dysphasia	11	-	-	quadr	-	-	-	n	Rt. Cerebell	n	n	n	y	n	n
32	9508495	10/1/2001	fathima	16	f	Lt	verm	y	y	n	n	n	n	n	n	n	n	n	n	n	15	-	-	-	-	Lt	neck stiff	n	Lt. cerebll	n	n	y	n	n	n
33	190621	23/4/01	algraja	27	m	-	BS	n	n	n	y	n	y	y	y	n	n	n	n	dysarthria	15	lcn, Lt.7,6	-	Lt. hemi	-	Lt.	-	y	BS	y	n	n	n	n	n
34	191583	22/3/2001	ibrahim	51	m	Lt	hemis	y	y	n	y	n	n	y	n	n	n	n	n	n	15	-	-	-	-	y	-	n	Lt cerebelll	n	n	y	n	n	n
35	191217		vellayudhan	48	m	Lt	hemis	y	n	n	y	n	n	n	n	n	n	n	n	nausea	15	-	-	-	-	-	-	n	Lt cerebelll	n	n	y	n	n	n
36	196707	17/01/02	sudhiya	16	f	Lt	hemis+Rt. Occi	y	n	n	n	y	n	n	n	n	n	n	n	visual blurr	15	-	-	-	-	-	Lt hemianop	n	Rt. Occip	n	n	y	n	n	n
37	230712	10/8/2004	reena	36	f	-	BS	n	n	y	y	n	y	n	n	n	n	y	n	dysarthria	9	Lt 5,Rt.7,6,LCN	y	Lt hemipleg	-	y	-	y	pontine	n	y	n	y	y	n
38	244477	19/10/4	mohd. Hussain	60	m	Rt.	hemis	n	n	n	n	n	n	y	n	n	n	n	y	n	15	-	-	-	-	Rt	-	n	Rt. Cerebell	n	n	y	y	n	n
39	243210	30/7/05	sibi	30	m	Rt	hemisp+dural comp	n	n	n	y	n	n	n	n	n	n	n	n	n	15	-	y	Rt hand grip	-	Rt	neck stiff	y	Rt Cerebell	n	y	y	y	y	n
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Rt	hemisp+dural comp	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	n	n	n	n	n	y	n	15	-	-	-	-	-	neck stiff	y	Rt cerebel	n	n	n	n	n	n

CTA	MRI exct locat	DSA size	nidus	feeders	aneurysm	draining vein	artery	SM grade	manag	embol	surg	postop	GCS	GOS	cran nerv	motor	sens	cerebell	others	followup	GCS	GOS	cran nrv	motor	sens	cerebll	other	DSA	MRI	comments
n	y. subdural bleed	<3	compact	multiple	n	deep	sca,pica	2	surg	-	y	-	15	5	-	-	-	Lt	-	8mth	15	5	-	-	-	-	-	no resid	n	-
n	-	>3	diffuse	multiple	n	deep	sca,aica,pica	3	emb,surg	pica(60-70%)	Rt RMC	-	15	3	RT 6,7 b/l LCN	Rt hemi			-	4yr	15	4	Rt.5, 6,7,8, LCN	Rt hemi	-	-	-	resid sca dura component	n	-
n	y.avm c intravent bleed	<3	compact	multiple	n	super	aica,pica	1	surg	-	SOC avm exc	-	15	5	-	-	-	-	-	3yr	15	5	-	-	-	-	-	n	n	-
-	n	<3	compact	multiple	pica	deep	pica,aica,sca	2	surg	-	LT SOC	-	15	4	-	-	-	Rt	-	7yr	15	5	-	-	-	-	-	no resid	n	-
n	y. Rt midbr n pons	<3	compact	multiple	n	deep	sca	3	ref GK	-	-	-	15	4	INO	-	-	y	-	4yr	15	-	INO	-	-	y	-	n	y	-
y	n	>3	diffuse	multiple	n	super	pica	2	emb,GK	pica(95%)	-	-	15	5	-	-	-	bl	nystag	7yr	15	-	-	-	-	y	-	no resid	n	-
n	y.tortuous ver V	<3	compact	multiple	y,Lt. pica	super	b/l pica	1	embol,surg	pica(50%)	mid SOC	-	15	3	-	-	-	bl	staccato speech	2yr	15	4	-	-	-	y	-	no resid	n	-
n	n	>3	diffuse	multiple	n	deep	b/l pica L>R, L aica,sca	3	ref for emb	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
n	y. hydroceph	<3	compact	single	y. basilar top proj super	super	Rt sca	1	surg for aneury	-	Rt FT ZYG Cran	-	15	3	Rt 3	Lt. Hemi	-	-	-	7yr	15	-	3	-	-	-	-	no aneu resid avm	n	not willing of avm surg
n	n	>3	diffuse	multiple	n	super	Lt. aica,pica	2	ref fr embol	-	-	-	15	5	-	-	-	-	-	lost to foll	-	-	-	-	-	-	-	-	-	-
n	n	>6	diffuse	multiple	n	super	Lt aica,pica,sca,pca	3	ref fr RT	-	-	-	15	5	-	-	-	-	-	lost to foll	-	-	-	-	-	-	-	-	-	-
n	n	>6	diffuse	multiple	n	deep	Lt. aica, sca,b/l pica	5	RT	-	-	-	15	4	Lt. 6,7,8b/l lcn	-	-	Lt	-	6yr	15	-	Lt 7,8,LCN	-	-	y	-	resid	n	-
n	n	<3	compact	multiple	y.venous sac	deep	Rt sca- doub	2	emb	sca 25% bucrylate complication -	-	-	15	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
n	Lt. cerebll avm c aneury	<3	compact	single	y,aica	super	Lt. aica	1	-	-	-	-	15	4	-	-	-	axial	-	-	-	-	-	-	-	-	-	-	-	-
ı	n	<3	compact	single	n	super	Rt pica.	1	surg emer	-	SOC & transvermian appr evc of haemat	-	11	3	-	-	-	-	-	1yr	15	-	-	-	-	-	-	avm	-	ref for SRT
ı	Lt. cerbll avm fed Lt. aica	<3	compact	multiple	n	super	Lt. aica, pica	1	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	called fr surg
ı	y verm + rt cerebel	>3	diffuse	multiple	y basil top	deep	Rt. Pca, sca	4	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	lost to followup
ı	n	<3	compact	multiple	n	deep	Rt.aica,pica, b/vert	3	emb/surg	ibca 17% 6 feeders oblitr >90% aica,pica	Rt. Vp shunt	drowsy	12	3	-	-	-	-	-	3yr 3mth	15	-	-	-	-	-	-	Rt. sup br aica, dist br of pica		reembolised with IBCA & Dsa s/o minimal supply to avm frm Rt aica
	n	>6	diffuse	multiple	n	deep	Lt. sca,pica,aica, Rt choroidal	5	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	ref for SRT
	n	>3	diffuse	multiple	n	deep	Lt. pica,sca	3	emb	50% histocryl with intranidal aneur rupt	b/l EVD	-	exp 1/5/00	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	n	<3	diffuse	multiple	y,Lt. pica	deep	Lt.pica,sca,aica	2	emb	Lt. pica- GDC coil emb (30%)	n	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	n	>3	diffuse	multiple	y,sca-BA	super	Rt.aica,sca	2	surg	-	anery clippin later RMC & exc	postop haematoma evac	15	3	Rt. III Lcn, b/l XII	-	-	b/l	-	1yr	15	3	Lcn, dysarthria	-	-	b/l	-	no resid	-	-
	pontine avm	<3	compact	multiple	y,ba perf feeder	super	Rt.aica, Ba per	2	surg	-	exc of avm +clippin	V ner cut reanast	15	3	Rt. 5,7,lcn	Lt. hemi	Lt. hemis	b/l	-	1 yr 1mth	15	3	Lcn imprv	-	-	b/l	-	no resid	-	-
	n	<3	compact	multiple	n	super	Rt. aica,pica	2	surg	-	RMC exc	-	15	5	Lt. 7,8,Lcn	-	-	-	-	2yr+2mth	15	5	Lcn compensated	-	-	-	hoarsress	no resid	-	-
	resolv haemat	<3	compact	multiple	n	super	Rt.aica,pica	1	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	called fr surg
	n	<3	compact	single	n	deep	Lt. aica	2	-	-	-	-	15	4	-	-	-	Lt.	-	-	-	-	-	-	-	-	-	-	-	SRT
	tect+midd cereb pedunc&suprasell	<3	compact	multiple	n	deep	Rt.aica,pica,sca	3	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	SRT
	n	<3	compact	single	n	super	Lt.sca	2	emb	glue 17% 2 feed 100%	-	-	15	3	Lt 3,6Rt 47umn,Lcn	Rt hemi	-	b/l	-	4yr	15	-	Rt4	-	-	Rt>Lt		resid sca feeder	Small avm on dorsal midbrain c atrphy of cerebl & sup cerbll ped	not willing fr emb
	Lt. CPA, cntr NC, crisoid aneu	<3	compact	multiple	n	super	L pca,basil,Rt ECA	2	surg	-	Fr Centneur		13/15	3	-	Lt. Hemi	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	-	<3	compact	single	y. Lt sca	super	Lt. sca.	1	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	disch fr embl
	n	<3	diffuse	multiple	n	deep block b/l tr-sig	Rt sca,aica,b/lpica	2	emb tried	-	-	-	11	3	-	quadr	-	-	-	-	-	-	-	-	-	-	-	-	-	ref for SRT
b	Lt. cerebll	>3	diffuse	multiple	y,Lt. sca	deep	b/l sca, Lt. pica	3	-	-	-	-	15	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	ref for emb
	Lt. pons	<3	compact	multiple	n	deep	Lt pca p1,sca,basil art feed	3	-	-	-	-	15	3	lcn, Lt.7,6	Lt. Hemi	-	Lt.	-	-	-	-	-	-	-	-	-	-	-	ref for SRT
	n	<3	diffuse	multiple	n	deep	Lt sca, aica,pica,Lt.eca-mma	2	-	-	-	-	15	4	-	-	-	Lt.	-	-	-	-	-	-	-	-	-	-	-	ref for SRT
	n	>3	diffuse	multiple	y,2 intranidal	super	Lt.sca,aica,eca,pica	2	-	-	-	-	15	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	embol
	n	<3	compact	single	n	deep	Lt. sca	2	emb occip Gr3	glue 17% 2 feed	-	-	15	5	LT. homony	-	-	-	-	-	-	-	-	-	-	-	-	occip avm disapp & cerebell appear	-	ref for SRT
	Rt pont+midbr	<3	compact	multiple	n	deep	ba perf	3	-	-	-	-	9	3	LT.5,7	Lt. Hemi	-	-	-	-	-	-	-	-	-	-	-	-	-	ref for SRT
	Rt. Cerebel	<3	compact	multiple	n	deep	Rt.pica,sca,aica	2	-	-	-	-	15	4	-	-	-	Rt.	-	-	-	-	-	-	-	-	-	-	-	ref for emboli
	n	>6	diffuse	multiple	n	deep	Rt aica,pica, Lt pica,b/l vert,asc phary,	4	emb+surg	glue+alcoh rt. Pica, rt VA dural br,Rt. Occip & ascn phar br >90%	evac haemt &vp shunt	-	15	4	-	Lt hand gr	-	Rt.	-	3mths	15	-	-	mild Lt hand grp	-	-	-	resid AVM- rt aica /sca. aica , mma.	-	embol 2 feeder frm aica >90% reduct,1yr2mth embl occip & sca feeder >70%
	n	>3	diffuse	multiple	Rt. Aica feed	deep	Rt. Aica,sca,pica,Ft. Occip.	3	emb	glue NBCA>60%	-	-	15	4	-	-	-	ataxia	-	2mth	-	-	-	-	-	-	-	-	-	called fr reemb after 3mth