

**CLINICAL PROFILE AND ENDOVASCULAR MANAGEMENT OF
POSTERIOR CIRCULATION ANEURYSMS**

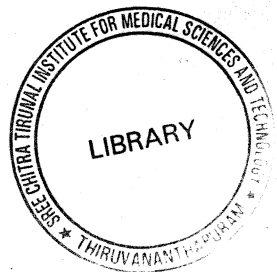
THESIS

**SUBMITTED IN PARTIAL FULFILLMENT FOR DEGREE
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INDIA**

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DECLARATION

I hereby declare that this thesis entitled “**CLINICAL PROFILE AND ENDOVASCULAR MANAGEMENT OF POSTERIOR CIRCULATION ANEURYSMS**”, has been prepared by me under the supervision and guidance of Dr. Arun Kumar Gupta, Professor and Head, Department of Imaging Sciences and Interventional Radiology, Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram.

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CERTIFICATE

This is to certify that the work incorporated in this thesis entitled, **CLINICAL PROFILE AND ENDOVASCULAR MANAGEMENT OF POSTERIOR CIRCULATION ANEURYSMS** for the degree of **DM (NEUROIMAGING AND INTERVENTIONAL NEURORADIOLOGY)** has been carried out by **Dr Keerthiraj B** under my direct supervision and guidance. The work done in connection with this thesis has been carried out by the candidate himself and is genuine.

Guide

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INTRODUCTION:

Intracranial aneurysms are localized pathological dilatation of cerebral arteries (1). They tend to occur at or near arterial bifurcations, mostly in the circle of Willis (2). Autopsy studies have shown that the overall frequency of aneurysms in general population ranges from 0.4 % to 10% (3). Aneurysms are classified based on their shape, size, location, and cause. Majority of intracranial aneurysms are saccular or berry aneurysms and contain all layers or components of normal vessel wall, whereas dissecting, fusiform, infectious, traumatic, and oncotic aneurysms are much rarer (4). Saccular, or berry aneurysms typically arise from arterial bifurcations, usually at the convexity of a curve, pointing toward the direction in which the flow would have continued if the curve had not been present (5). Nonsaccular aneurysms arise at nonbranching sites along the vessel trunks (6). Aneurysms can arise as solitary (70-75%) or multiple (25-30%), usually located at Circle of Willis (7). Intra cranial aneurysms result from a combination of hemodynamic stresses and acquired degenerative changes within the arterial wall, even if inherited diseases may predispose their formation by weakening the vessel wall (8). Other risk factors for development and rupture of aneurysms include hypertension, smoking, heavy alcohol consumption (7). Histopathologically, the wall of an aneurysm is thin and harbors only intima and adventitia, with internal elastic lamina and the tunica media usually terminating at the aneurysm neck and variable amount of fibrohyalin tissue and calcification forming the wall of the aneurysm with presence of thrombus and organized clot in the lumen of large aneurysms (9, 10, 11, 12).

Aneurysms of posterior circulation accounts for 10-15% of all intracranial aneurysms (13). Posterior intracranial circulation aneurysms arise from the intracranial portion of vertebral artery, basilar artery, or any of their branches (14,15). The goal of aneurysm treatment is complete, immediate, permanent, and safe exclusion of the aneurysm from circulation and preservation of the parent artery (16, 17). Although surgical clipping is considered to be the standard aneurysm treatment; microsurgical clipping of posterior circulation aneurysms is associated with several problems like deep anatomical position, narrow operation field,

contusion from brain retraction, and damage to the perforating arteries. Consequently, it may cause severe postoperative neurological deficits (18, 19, 20, 21).

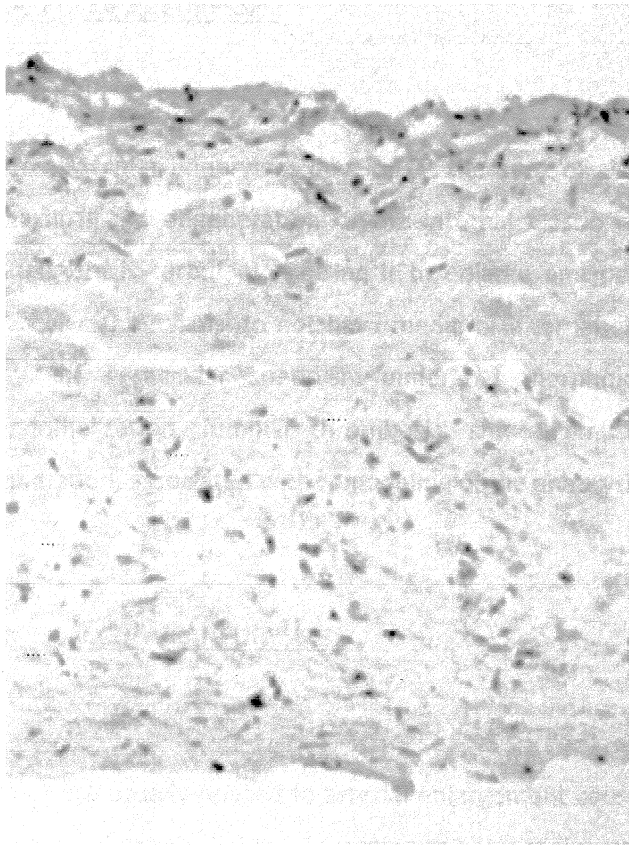
Several recent developments using Guglielmi detachable coils (GDCs) and micro catheter improvements, evolving coil technology with introduction of adjuvant devices such as intracranial stents, remodeling balloons have led to improved anatomic and clinical outcomes in endovascular management of posterior circulation aneurysms(22). Recently flow diverters like pipeline stent is being used as definitive constructive treatment option for large, giant, and fusiform posterior circulation unbled aneurysms achieving complete aneurysm occlusion without embolization coils with preservation of perforators(23). However, even though endovascular treatment is an essential modality for the treatment of patients harboring posterior circulation aneurysms, the main challenge continues to be the recurrence and the associated risks of rebleed (24).

REVIEW OF LITERATURE

Vast majority of intracranial aneurysms involve the anterior circulation and only 15-20% are located in posterior circulation (13). Half of these develop at basilar top with varying degrees of involvement of the P1 segment and other 50% from other posterior fossa vessels while aneurysms of anterior inferior cerebellar artery (AICA) and vertebral artery aneurysms without involvement of the VA-PICA junction or vertebrobasilar junction are extremely rare (7). In contrast to saccular aneurysms, dissecting aneurysms occur much more often in the vertebrobasilar system and are more often in man than in woman (24). Intracranial dissecting aneurysms often present with devastating subarachnoid hemorrhage (25). Ischemic infarction due to stenosis or occlusion by the intramural haematoma or by remote embolism occurs in about 42% of patients with posterior circulation dissections (24). The etiopathogenesis of cerebral aneurysms is uncertain and the cause of subsequent rupture is not evident in all cases (26). The congenital or medial defect theory hypothesizes a weakness in the artery wall due to maldevelopment (27). Support for a congenital origin comes from the frequency of multiple aneurysms, familial occurrence of aneurysms and association of systemic inherited diseases of vascular connective tissue, such as Marfan's syndrome, Ehlers-Danlos syndrome and polycystic kidney (28, 29). Alternately, the degenerative theory suggests an acquired defect in the vessel wall (30, 31, 32). This theory is supported by the increased frequency of aneurysms with age, hypertension, smoking and arteriosclerosis (33). A combination of hemodynamic stresses and acquired degenerative changes within the arterial wall often predispose to aneurysm formation, even if inherited diseases may predispose their formation by weakening the vessel wall (8). Histopathologically, the wall of an aneurysm harbors only intima and adventitia, with internal elastic lamina usually terminating at the aneurysm neck (9, 10, 11, 12). Tears in the internal elastic lamina (IEL) and associated vascular wall remodeling have been thought to precede IA formation, as the IA wall lacks the IEL, which normally provides structural strength for cerebral artery walls (34). The main factor contributing to the strength of cerebral arteries is an intact internal elastic lamina of the intima, which is the only elastic layer in cerebral arteries (35). It has

therefore been suggested that degenerative disruption of the internal elastic lamina due to hemodynamic stress is necessary for an aneurysm to develop (36).

Intracranial aneurysm



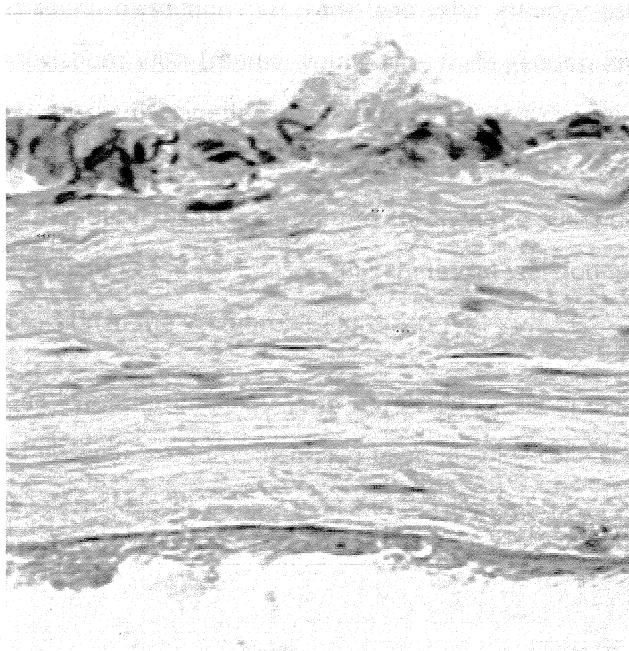
Adventitia

Myointimal hyperplasia

Missing IEL

← Missing endothelium

Normal cerebral artery



Adventitia

Medial layer

← IEL
← Endothelium

SACCULAR ANEURYSMS:

Most saccular aneurysms develop at the apex of a bifurcation, where hemodynamic stress is maximal and degeneration of the internal elastic lamina occurs. The blood flow inside the aneurysm is turbulent, causing it to enlarge and possibly to rupture. Therefore, the walls of saccular aneurysms do not have media or internal elastic lamina (37). Before the rupture, the wall of a saccular aneurysm undergoes morphological changes, such as apoptosis, de-endothelization, luminal thrombosis, smooth muscle cell proliferation, and macrophage infiltration, associated with remodeling of the aneurysm wall (38).

NON SACCULAR ANEURYSMS:

They are usually related to arterial enlargement caused by atherosclerosis but may also result from focal arterial injury. According to their radiographic appearance, nonsaccular intracranial aneurysms can be classified as fusiform, dolichoectatic, and transitional (39). Giant fusiform aneurysms present with symptoms of mass effect in 50% of cases and hemorrhage in 20% (40). Intradural dissections are rare, and their etiology usually remains unknown, though possible associations with trauma, young age, male gender, and vascular disorders have been reported (41). Dissecting aneurysms develop when the dissection proceeds through the media into the subadventitial layer and causes dilatation of the outer wall of the vessel (42). SAH may result if the intradural dissection ruptures through the adventitia (43). In a recent retrospective analysis by Ramgren et al (44), dissecting aneurysm was found in 69% of the cases with SAH caused by dissection in the vertebrobasilar territory.

Nonatherosclerotic aneurysms unrelated to the branching zones (including fusiform aneurysms and dissecting aneurysms) have been histologically classified based on the lesional patterns of the internal elastic lamina (IEL) and the state of the intima into 4 types (45):

- a) Type 1 corresponded to classic dissecting aneurysms, the pathogenesis of which was characterized by acute widespread disruption of the IEL without intimal thickening. Patients with Type 1 aneurysms had an ominous clinical course, and many presented with sudden subarachnoid hemorrhage with frequent rebleeding.
- b) Type 2 aneurysms were segmental ectasias, which had an extended and/or fragmented IEL with intimal thickening. Weakness of the arterial wall caused by the damaged IEL was assumed to be compensated by the intimal thickening. The luminal surface of the thickened intima was smooth without thrombus formation. The patients with Type 2 aneurysms had a placid clinical course.
- c) Type 3 aneurysms were dolichoectatic dissecting aneurysms, pathologically characterized by fragmentation of the IEL, multiple dissections of thickened intima, and organized thrombus in the lumen. Most of them were symptomatic and progressively enlarged over time.
- d) Type 4 aneurysms were saccular aneurysms unrelated to the branching zones. They arose in areas with minimally disrupted IEL without intimal thickening, and there was a risk of rupture.

TRAUMATIC ANEURYSMS:

Traumatic aneurysms account for 0.2% – 1.0% of all intracranial aneurysms. They are usually false aneurysms (all the wall layers are breached), where local bloods clot has organized to form a fibrous sac. According to the review by Fox (46), 75% of these cases occurred after closed head injury, 14% after penetrating trauma, and 11% after craniotomy and transphenoidal or other types of paranasal sinus surgery.

INFECTIOUS ANEURYSMS:

Infectious (formerly called "mycotic") aneurysms typically occur in patients with a history of drug abuse or with infective endocarditis. Infectious aneurysms tend to be located in peripheral branches distal to the circle of Willis and develop due to septic emboli, which cause a local inflammation, vessel wall disruption, and aneurysm formation (47). Spreading of an extravascular infection, such as meningitis, osteomyelitis, or sinusitis, may occasionally cause the formation of infectious aneurysms (48). The organisms causing the infection may be bacterial (*Streptococcus*, *Staphylococcus*, *Enterococcus*) or fungal (*Aspergillus*, *Candida*) (49).

FLOW-RELATED ANEURYSMS:

Flow-related aneurysms can be demonstrated in some patients with abnormally high flow in the cerebral arteries due to a pre-existing arteriovenous malformation (AVM), anatomical variant, or occlusion of ICA (50). They may regress spontaneously after the treatment of the underlying pathology and the consequent cessation of the high-flow state and the normalization of hemodynamic stress against the arterial walls (51). Flow-related aneurysms may rupture and cause hemorrhage (51). The presence of flow-related aneurysms further supports the theory of hemodynamic stress as a factor promoting aneurysm formation and growth (52)

GIANT ANEURYSMS:

Giant aneurysms, defined as larger than 25mm in diameter, account for 5% - 8% of intracranial aneurysms. Presenting symptoms, in addition to SAH, are commonly due to a mass effect, intracerebral hemorrhage, or thromboembolism. Giant aneurysms are frequently (in at least

60% of cases) associated with either partial or, less commonly, complete thrombosis (53). A completely thrombosed giant aneurysm may spontaneously recanalize (54). While the etiology of saccular aneurysms remains unclear, an interesting hypothesis concerning the pathogenesis of partially thrombosed giant aneurysms has been proposed (55). The formation of such lesions might be explained by an extra luminal inflammation process promoted by an enzyme called 5-lipo-oxygenase, which generates different forms of leukotriens. Leukotriens in turn are potent mediators of inflammation. Adventitial inflammation leads to weakening of the media, thereby degrading the extracellular matrix, the internal elastic lamina, and finally, the integrity of the vessel lumen. This in turn results in dilatation of the vessel and aneurysm formation. In addition to this biological cascade, neovascularization of vasa vasorum, promoted by 5-lipoxygenase activated macrophages, results in repeated subadventitial hemorrhages, creating new layers of intramural haematoma within the vessel wall. The increasing number of such hemorrhagic layers causes progressive growth of aneurysm size. A giant intracranial aneurysm can therefore be regarded as a proliferative disease of the vessel wall induced by extravascular activity (55)

PEDIATRIC ANEURYSMS:

Intracranial aneurysms in children are rare. In an analysis of 3000 ruptured aneurysms by Patel & Richardson (56), 2% of the patients were aged under 19 and only 0.1% under 5 years. There are several specific features of pediatric intracranial aneurysms that differ from those seen in the adult population. Compared with adults, posterior circulation aneurysms and infectious aneurysms are more common in the pediatric population (57) and aneurysms tend to be more frequently large or giant in size (58). Location of the aneurysm in the distal part of ACA, MCA, or PCA is much more common among children compared to adults (57). In children, a male predominance is seen, contrary to the female predominance in adults (57). In the study by Allison et al (57), 16% of the aneurysms in infants and children were associated with intracranial vascular variations or anomalies. In a consecutive series of 59 children with intracranial aneurysms, Lasjaunias et al (59) found dissecting aneurysms in 33 (56%), post-traumatic aneurysms in 2 (3%), infectious aneurysms in 8 (14%), and saccular aneurysms in 16 (27%)

patients. Most of the dissecting lesions were located in the posterior circulation, while the saccular lesions mostly occurred in the anterior circulation (59).

INTRACRANIAL ANEURYSMS AND HERITABLE DISEASES

A small fraction of intracranial aneurysms are associated with heritable connective-tissue diseases such as autosomal dominant polycystic kidney disease (ADPKD), Ehler-Danlossyndrome type IV, neurofibromatosis Type 1, and Marfan syndrome (60).ADPKD has been found to be a powerful risk factor for intracranial aneurysm formation (relative risk 4.4) indicating a need for aneurysm screening (61).

NATURAL HISTORY AND CLINICAL PRESENTATION OF ANEURYSMS:

The prevalence of intracranial aneurysms among the general population has been difficult to assess, because there used to be no non-invasive method available to diagnose intracranial aneurysms. According to autopsy studies, the prevalence of unruptured intracerebral aneurysm varies from 0.8% to 8.1% (62). The largest series included data from 87,772 autopsies, and the prevalence of intracerebral aneurysm was reported to be 1.6% (63).Bannerman et al (56) reviewed 51360 autopsies and reported a 1.43% prevalence of intracranial aneurysms, of which 0.34% were ruptured and 1.09% unruptured. Clinical presentation of posterior circulation aneurysms includes symptoms associated with major aneurysmal rupture (SAH), minor aneurysmal hemorrhage (warning leak or sentinel bleed), nonhemorrhagic manifestations (mass effect or cerebral ischemia), and asymptomatic scenarios (incidental aneurysm detection or identification through screening).

Most intracranial aneurysms remain undetected until the time of rupture and SAH presenting as abrupt onset of severe headache is by far the commonest initial clinical presentation (7).Nearly half of patients present with milder symptoms caused by warning leak before severe rupture of aneurysm (64). In addition to advanced age and female gender, cigarette smoking, hypertension, and alcohol abuse have also been recognized as risk factors for SAH (65). The

etiology of SAH includes a ruptured intracranial aneurysm in more than 80% of cases (66). Intracranial dissections, cerebral AVMs and other cerebral vascular malformations, dural AVFs, trauma, bleeding disorders, substance abuse, spinal origin of the hemorrhage, and other rare conditions explain less than 5% of primary SAH (66). Perimesencephalic nonaneurysmal SAH accounts for up to 15% of cases (67). In cases of perimesencephalic SAH, CT and MRI findings differ from those in aneurysmal SAH in that the maximum amount of blood is located anterior to the pons, possibly extending to the ambient cisterns or the basal parts of Sylvian cisterns (68). Although aneurysmal SAH has characteristic historical features, the constellation of symptoms may vary with location, size, shape, and direction of the aneurysm (69). Ruptured aneurysms of posterior circulation have a worse prognosis than patients with a ruptured aneurysm in another location (70).

Aneurysms may exert a mass effect on the adjacent neuronal and vascular structures and thereby produce progressive neurological deficits (71). Approximately half of the symptoms due to discovered unruptured aneurysms are attributable to cranial nerve compression (72). When such symptomatic aneurysms were embolized, Halbach et al (73) noticed that the size of the aneurysm markedly decreased in some patients but remained unchanged in others. However, alleviation of symptoms occurred both in the patients whose aneurysm was reduced in size and in those with no size reduction. Rodriguez-Catarino et al (74), noticed alleviation of symptoms (cranial nerve dysfunction and/or headache) in the patients whose aneurysms actually increased in size after the embolization. These findings suggest that the resolution or improvement of the symptoms may be related to the reduction of the pulsatile effect of the aneurysmal sac rather than the size of the aneurysm (74). Basilar tip or basilar trunk aneurysms can cause symptoms associated with brainstem compression such as lower cranial nerve palsies or tetraparesis, or they may obstruct the aqueduct of Sylvius and lead to hydrocephalus (75, 76). Aneurysms at the vertebrobasilar junction or the PICA origin may cause lower cranial nerve palsies or a partial lateral medullary (Wallenberg's) syndrome (77). Intracranial thromboembolic manifestations can occur with an unstable intraluminal thrombus migrating and thus causing thromboembolic symptoms in the vascular territory adjacent to the aneurysm, which increases the risk of complications in the treatment of such aneurysms (78).

UNRUPTURED ASYMPTOMATIC ANEURYSMS:

The natural course of unruptured aneurysms has been poorly understood because of the paucity of studies with sufficiently large patient series and sufficiently long follow-up. The studies have also been biased due to surgical selection (only old and poor grade patients remained untreated and constituted the patient series of the studies) (79). In the study by Juvela et al. (2000) (79), 181 unruptured aneurysms were followed for a mean of 19.7 years and the annual average incidence of rupture was 1.3%. The cumulative rate of bleeding was 10.5% at 10 years, 23.0% at 20 years, and 30.3% at 30 years after the diagnosis. Cigarette smoking, aneurysm size, and age (inversely) were found to be important factors determining the risk for subsequent aneurysm rupture (67).

Data from International Study Of Unruptured Intracranial Aneurysms (ISUIA) showed that 5year cumulative risk of aneurysm rupture in posterior circulation with no previous history of SAH was 2.5% and in those aneurysms <7mm with previous history of SAH was 3.4% while the 5year risk of rupture of 7-12 mm aneurysm size was 14.5%,13-24mm aneurysm size was 18.4% and >25mm aneurysm size was 50% , clearly indicating an increased risk of rupture with increase in size of aneurysm and increased risk of rupture of posterior circulation aneurysms compared to aneurysms in anterior circulation(80).

IMAGING OF INTRACRANIAL ANEURYSMS:

A) DIGITAL SUBTRACTION ANGIOGRAPHY AND 3D ROTATIONAL

ANGIOGRAPHY:

Digital subtraction angiography (DSA) is the gold standard for aneurysm detection (81). DSA is an invasive technique with a reported complication rate of 1.8-2.1% (82). During the past

decade, safer contrast agents have become available, and important technical advances have been made, including digital imaging systems, smaller catheters, and hydrophilic guide wires. In their prospective study, Bendszus et al (83), detected new bright lesions by diffusion weighted MRI in 26% of 66 consecutive patients after diagnostic cerebral angiography. No new neurological deficits were found in any of the patients, indicating that embolic events after diagnostic cerebral angiography are much more frequent than apparent neurological complications. The appearance of lesions correlated significantly with difficulties in catheterization, the amount of contrast media needed, and the fluoroscopy time (83). Three-dimensional rotational angiography (3DRA) involves a novel software application that reconstructs standard rotational angiographic data into a computer rendering that can be manipulated by the operator at any angle or viewpoint (84). A good correlation of 3DRA images with surgical anatomy has been demonstrated by Tanoue et al (85). The utility of this technique in planning either surgical or endovascular treatment of cerebral aneurysms has also been demonstrated (85).

B) COMPUTERIZED TOMOGRAPHY ANGIOGRAPHY (CTA):

Computerized tomographic angiography (CTA) is based on volumetric scanning of the brain after intravenous administration of contrast medium. The quality of CTA images has been improved by the multislice technology and the development of post processing hardware and software. Single-row helical CT scanners have been reported to yield 91% sensitivity and 95% specificity (86), when compared to DSA, whereas the reported rates of multislice CT scanners are 96% and 97%, respectively (87). CTA is less invasive than DSA and has the potential advantage that it can be performed at the time when SAH is detected by CT. However, small (<2mm), partially thrombosed aneurysms & small aneurysms close to skull base may be missed by CTA even in experienced centers.

The value of CTA is limited to postoperative imaging due to the artifacts caused by clip or coil mesh, which make the evaluation of the aneurysm base very difficult. In a series by Kivisaari

(88), a remarkably low rate, i.e. 2%, of incomplete closures after microsurgical clipping of intracranial aneurysms was detected by CTA, while in their previous series (89), DSA revealed an unexpected aneurysm residual in 7.3% of the clipped aneurysms. The low figures for residual necks raise a question of the reliability of CTA in the postoperative evaluation of treated aneurysms.

C)MAGNETIC RESONANCE ANGIOGRAPHY(MRA):

Magnetic resonance angiography (MRA) is a non-invasive method for the detection of intracranial aneurysms. MRA source images are usually acquired through the circle of Willis, using a three-dimensional single slab time of flight technique (3D TOF) (90). The advantages of MRA include non-invasiveness, lack of radiation, and good visualization of the intra-aneurysmal thrombus and the adjacent brain parenchyma. Its usefulness is, however, limited in the case of small aneurysms and aneurysms located close to the skull base. In the series of Okahara et al (91), the sensitivity of 3D TOF in detecting intracranial aneurysms varied from 60% to 79%, depending on the experience of the reader, and its specificity was 77-86%, respectively, when compared to 3D rotational angiography.

Contrast-enhanced MRA with ultra fast imaging sequences and image subtraction has been established as superior to 3D TOF in accurately depicting experimental lateral aneurysms in canines (92), and also in the assessment of recently ruptured intracranial aneurysms in a clinical trial (93).MRA has also been used in follow-up studies of previously coiled aneurysms. The sensitivity of 3D TOF MRA compared with DSA for the detection of residual aneurysm has ranged between 71% and 97% and the specificity in ruling out residual filling in aneurysms from 89% to 100%, respectively (94).

ENDOVASCULAR TREATMENT:

Endovascular techniques were first developed for aneurysms that were considered inoperable due to their location, size, intraluminal thrombosis, atherosclerosis in the neck or dome, or calcifications in the wall. Attempts to induce thrombosis of systemic aneurysms either by introducing foreign bodies or application of electrical or thermal injury date back to the first half of the nineteenth century. Velpaeu (1831) and Phillips (1832) independently described a method of introducing arterial thrombosis by inserting a needle into aneurysmal lumen and withdrawing it after thrombus has formed. Endovascular coiling for the treatment of intracranial aneurysms was first introduced into clinical use in 1990 (95). Detachable latex balloons which were earlier used for treatment of carotidocavernous fistulas were initially used in treatment of large and giant aneurysms such as cavernous carotid aneurysms which were otherwise treated by parent artery occlusion (96). Later this technique was extended to intracranial aneurysms (97). The technique of inflating the balloon inside a previously ruptured aneurysm led to hesitation of its use due to risk of rupture (98). In 1991, the Italian neurosurgeon Guido Guglielmi published his preliminary experience with electrolytically detachable platinum coils, opening a new era in aneurysm treatment. The introduction of Guglielmi Detachable Coils (GDC: Boston Scientific/Target, Fremont, California) in early 1990s revolutionized the treatment of intracranial aneurysms with the use of detachable platinum coils to densely fill the aneurysm sac thereby excluding the aneurysm sac and neck from circulation to prevent bleeding/rebleeding from the aneurysmal sac (99,100). Although initially endovascular occlusion of aneurysms was done in patients who were poor surgical candidates, endovascular treatment of intracranial aneurysms has made remarkable progress with low incidence of complications (101,102). Treatment with Guglielmi electrically detachable coils has been a less traumatic and more controllable endovascular method of aneurysmal occlusion. These devices have three main advantages: 1) they are very soft and less traumatic to the aneurysm wall; 2) they are detachable by application of electric current, which is also believed to induce thrombosis by attraction and accumulation of negatively charged blood elements; and 3) they are retrievable before detachment, which allows safe and accurate placement within the aneurysm aiding in complete occlusion in aneurysms with small necks (103). Coil embolization has limitation in the treatment of broad-

necked aneurysms because of possible coil herniation and migration into the parent artery lumen when coils are densely packed within the aneurysm(104,105).

FURTHER DEVELOPMENT IN COIL TECHNOLOGY:

In the late 1990's, softer GDC Soft coils were introduced as well as 2D and 3D shaped coils. Later, even softer GDC Ultra soft coils have become available. The development of softer coils enabled better filling of the aneurysms and safer deployment in small aneurysms compared to the original GDC. Three-dimensional coils (3D-GDC,360 degree coils) was developed for wide-necked aneurysms, to prevent coil protrusion when additional coils are inserted into the aneurysm (106). Later on, different platinum coils with different detachment technologies have become commercially available. Some of them are mechanically (IDC, Target Therapeutics, Fremont, CA and DCS, William Cook

Europe A/S, Denmark) and some hydraulically (TruFill DCS, Cordis, Miami, FL and Microplex, Microvention, CA) detached coils (107,108,109). Competitive platinum coils (Sapphire, Micro Therapeutics, Bochum, Germany and Micrus Microcoil System, Micrus Corporation, Mountain View, CA) with an electrolytic detachment system were developed (110). Despite the variety of detachment systems used, the basic procedure is similar to GDC, enabling repositioning, retrieval, and replacement of the coil before detachment. Cordis developed complex shaped TruFill coils, which enabled concentric packing of the aneurysm with 3D-shaped coils used alone. New TruFill Orbit (Cordis, Miami, FL) coils have recently been introduced and are the first 3D coils available in all sizes from 2mm to 20 mm. In a recent study by Slob et al (111), complex TruFill coils with a wire diameter of 0.012 inches enabled significantly better packing compared with helical GDC with a wire diameter of 0.010 inches, and the retreatment rate was lower for TruFill coils than GDC. Although GDC and other detachable platinum coils have proved to be reliable and effective in the short term aneurysm, recanalization due to coil compaction has emerged as a major long-term drawback of this technique. To avoid recanalization, several new types of coils have been under investigation. So far, clinical experience is available of biologically active coils (112), radioactive coils(113), and hydro gel-coated coils (114). Matrix coils (Boston Scientific, Target Therapeutics, Fremont, CA) consists of a thin platinum coil covered with bioabsorbable, polymeric material (polyglycolic acid/lactide) (112). A prospective, multicenter

study (called ACTIVE study) evaluated the potential benefits of Matrix coils in the treatment of cerebral aneurysms (114). During 12 months' follow-up, 16% of the

aneurysms were retreated and 7% of the aneurysms had bled. On the basis of the ACTIVE study, the benefits of Matrix over standard platinum coils remained questionable (115) The main cause for the poor initial occlusion grades was considered to be the high friction of Matrix coils, which might result in compartmentalization of the coils within the aneurysm and prevent dense packing. The absorbable nature of the bioactive coating may also explain the high recanalization rate. The Hydro coil Embolic System (MicroVention, CA) consists of a hybrid hydro gel-platinum coil with an initial diameter of 0.009 inch and an expanded diameter of 0.027 inch. In blood, the Hydro coil swells to its maximum diameter in about 20 minutes (116). Although these initial results of the hydro coils were promising& showing improvement of aneurysm packing, further larger series with follow-up will be necessary to investigate whether the findings ultimately correlate with a decreased rate of aneurysm recurrence and better clinical outcomes.

LIQUID EMBOLIC MATERIAL:

In the treatment of intracranial aneurysms, Onyx HD500 is constrained by the placement of a balloon over the neck of the aneurysm. It takes about ten minutes for Onyx to completely solidify(117).Despite the good overall initial and follow-up results of embolization with detachable coils, the results in cases of large and giant aneurysms are significantly inferior to those of small aneurysms due to coil compaction and recanalization In the Cerebral Aneurysm Multicenter European Onyx (CAMEO) trial, complete occlusion was achieved in 79% of the 71 aneurysms for which the results of 12-month follow-up angiography were available (117). In the reported clinical series, the use of Onyx for intracranial aneurysm embolization has been mainly targeted to treat these challenging lesions (117).The authors concluded that this treatment provides more stable results than any other treatment option in internal carotid artery aneurysms (118).

REMODELING TECHNIQUES:

The standard coil embolization technique is limited by its inability to occlude wide necked aneurysms (119). To overcome this problem, the balloon-assisted remodeling technique was first introduced by Moret et al (120). This technique consists of temporary inflation of a balloon across the aneurysm neck during embolization to avoid inadvertent coil protrusion into the parent artery. Traditionally, two types of balloon have been available to perform balloon-assisted coil placement in cerebral aneurysms: oval, guide-dependent balloons for sidewall aneurysms and round, flow-directed balloons for bifurcation aneurysms. A novel, more compliant, guide-dependent oval balloon (Hyperfoam, Micro Therapeutics, Irvine, CA) has been developed for the difficult anatomical situations where regular balloons are not feasible (121). The compliance of this balloon is a mechanical property defined by the propensity of the balloon to change its cylindrical shape to fit the anatomy of the vessel in which it is inflated. It is more effective than round, flow-directed balloons in the treatment of bifurcation aneurysms, because it is easier to position adequately (121). Moret et al reported the first large series of aneurysms treated with a balloon assisted technique. They were able to perform successfully 93% of the procedures with morbidity and mortality rates of 1% and 0%, respectively. According to follow-up data, 77% of the treated aneurysms were completely occluded, 17% of them were sub totally occluded, and only 6% of the aneurysms had incomplete occlusion (120).

Endovascular stents can be useful in the treatment of intracranial aneurysms in cases that otherwise could not be treated even with the balloon remodeling technique and when the parent artery cannot be sacrificed. Such aneurysms are either very wide-necked, fusiform, or dissecting (122). The stent placed across the aneurysm neck alters the blood flow and redirects the shear stress in such a way that it may lead to partial thrombosis (123) and in some cases stent(s) alone can be sufficient to occlude the aneurysm (124). However, both experimental and clinical results have led to the conclusion that an additional embolic device, for instance, coils or Onyx, is usually needed to occlude the aneurysm sac (125). The first intracranial stent placements were performed with balloon expandable stainless-steel stents developed for cardiac use, and the navigation of the device into the tortuous intracranial vasculature was challenging and frequently impossible.

Recently, technological developments have enabled the production of more flexible stents with better properties, allowing them to be advanced into the tortuous intracranial vasculature and also allowing treatment of complex wide-necked aneurysms in which endovascular reconstruction of the parent vessel is necessary (126). These new very-low profile self-expandable stents are made of nitinol, and they are specifically designed for intracranial use. The preliminary clinical results of these stents have been very favorable, but only limited follow-up data are available so far (126). Using the Neuroform stent (Boston Scientific/ Target Therapeutics, Inc., Natick, MA), Lylyk et al reported procedural morbidity and mortality rates of 8.6% and 2.1%, respectively, among the 48 aneurysms treated (127). The properties of these novel stents also enable Y-shaped reconstructions of parent vessels in cases of complex very wide-necked bifurcation aneurysms as well as retrograde access to the lesion, as first described by Moret et al (128).

There are limited data in the literature concerning the use of covered stents, called stent-grafts, in the treatment of intracranial aneurysms. When deployed across the aneurysm neck, the stent-graft simply hampers the entry of blood flow into the aneurysm sac and occludes the aneurysm with no need to place any embolic material inside the aneurysm. The use of intracranial stent-grafts is, however, limited to vessel segments where the arterial branches do not originate from the landing area of the device. The currently available devices are relatively stiff and have a poor profile for intracranial navigation, which restricts their use intracranially. Anecdotal case reports of successful stent-graft treatment of lesions in the intracranial vertebral artery and at the vertebrobasilar junction have also been published (129).

Balloon remodeling techniques and stent-assisted coiling have proved to be useful for some broad-necked aneurysms; however, each of these techniques introduces a small additional risk of parent artery ischemia, perforation, distal thromboembolism, and occlusion of adjacent perforators and branch arteries by the lattice of the stent (130,131).

HISTOLOGICAL FINDINGS IN COILED ANEURYSMS:

In experimental aneurysms in swine described by Hino et al (132), electron microscopic scanning immediately after the procedure showed platelets and fibrin adherent to the coil surface. A fibrin network had already been formed, but no migration of endothelial cells had occurred. At one week after the procedure, electron microscopic scanning revealed the development of a thick fibrin network, which extensively covered the coil surface, but there were also portions where the coil remained uncovered. Some endothelial cells had entered from the periphery toward the aneurysm orifice. At 3 weeks, the aneurysm orifice was macroscopically covered with a transparent neointima, and in 40% of the aneurysms, electron microscopic scanning showed the aneurysm orifice to be covered sufficiently by the vascular endothelial cells.

Castro et al reported histopathological findings in two aneurysms of a patient who died 33 months after embolization by GDC (133). In gross examination, the coils were so firmly attached in the aneurysmal lumen that they could not be removed. In microscopic study, the fundi of both aneurysms were filled with vascular fibrous connective tissue scars that were more dense on the periphery of the sac, where proliferation of inflammatory cells was evident. There was no evidence of residual thrombus. Sections through the neck of one of these aneurysms (OphtA, neck remnant in follow-up angiography) revealed that the ostium was covered by a neointima organized in two layers. The superficial endothelial layer was continuous with the endothelium extending from the lumen of the parent artery. The deeper layer consisted of dense, vascular, and collagenous fibrous tissue aligned parallel to the long axis of the parent vessel (133).

ANGIOGRAPHIC RESULTS OF ENDOVASCULAR TREATMENT:

The success of endovascular therapy is usually reported in terms of occlusion rates. Regardless of the ability of endovascular therapy to completely occlude the aneurysm initially, there is also concern regarding the long-term follow-up results for aneurysms that have been completely or incompletely coiled. It has been demonstrated that aneurysm recurrence, coil compaction, and enlargement of residual neck may occur after endovascular treatment. It is thus generally agreed that long-term angiographic follow-up is necessary (134,135).

PROCEDURAL COMPLICATIONS OF ENDOVASCULAR TREATMENT:

According to the recent literature, the reported procedural complication rates in clinical series range between 8.4% and 23% (136). In the meta-analysis of Brilstra et al., the complication rate was 9.6% for ruptured aneurysms and 20% for unruptured aneurysms (136). Most of the procedural complications are transient or reversible, and only a minority of them cause permanent morbidity and mortality.

THROMBOEMBOLIC COMPLICATIONS:

The endovascular treatment of intracranial aneurysms may be complicated by a transient ischemic attack (TIA) or ischemic stroke (137). The reported rates for procedural ischemic complications following endovascular treatment of cerebral aneurysms range from 2.7% to 17% (136,138). Most of these events can be attributed to thrombosis of the parent or branch arteries from which the aneurysm arises or to distal embolization of a thrombus from the treated aneurysm (99,100). The occurrence of ischemic attacks during the embolization procedure can also be caused by a thrombus from the catheter tip or by air embolism (139). Large aneurysm diameter and coil protrusion have been found to be independent risk factors for post procedural thromboembolic events (140).

Although there is no definitively proved benefit of heparin administration in neuroangiography or in neurointerventional procedures, the anticoagulant effect of heparin should be useful in preventing ischemic periprocedural complications.

IATROGENIC ANEURYSMAL RUPTURE:

Aneurysmal rupture during endovascular treatment is one of the most feared complications of endovascular aneurysm treatment. The reported percentage of aneurysmal rupture in clinical series varies between 2.0% and 8.8% (141). In the meta-analysis by Brilstra et al, the calculated

procedural perforation rate was 2.4%. Rupture may occur during any stage of the treatment procedure (136). Komiyama et al reviewed the aneurysmal ruptures during angiography and found that rebleeding occurred most often when the examination was performed on the day when the initial bleeding occurred, and the risk was highest during the first 6 hours from the onset of SAH (142). Fluctuations in blood pressure resulting from pain, anxiety, or anesthesia may be contributing factors (143). The pressure wave of an injection of contrast material may overcome the weak walls of an aneurysm (144). Perforation may be caused by the guide wire as it is steered into the aneurysm or subsequently by the micro catheter as it is advanced over the guide wire into the aneurysm. The coil itself or the delivery wire to which the coil is attached may perforate the aneurysm. Excessive packing of the aneurysm with coils for sealing the neck of the aneurysm may also result in its rupture. The alterations in flow dynamics inside the aneurysm after the introduction of coils may divert the flow toward the weak point of the aneurysm, resulting in bleeding either during the procedure or afterwards (143).

The basic management of iatrogenic aneurysm rupture involves immediate reversion of anticoagulation therapy by protamine sulfate and continuation of the embolization procedure (143). In a case of micro catheter perforation, Willinsky et al described a salvage maneuver including the use of a second micro catheter, with which the aneurysm was packed with coils, while the first micro catheter was temporarily left in place (145). If the balloon remodeling technique is used, the neck of the ruptured aneurysm can also be temporarily sealed by the balloon to control the bleeding (146). Aneurysm rupture during embolization may be accompanied by severe intracranial hypertension, which causes either a decrease or an arrest of cerebral perfusion, the duration of which determines the clinical outcome.

REBLEEDING RISK:

In the ISAT study, the rebleeding rates after treatment during a mean follow-up of four years were 1.2% in the surgical group and 3.2% in the endovascular group (147). A retrospective analysis of endovascularly treated ruptured aneurysms in a single center revealed a 1.4% incidence of early rebleeding (at 30 days after treatment) and a 1.27% incidence of late rebleeding (>30 days after treatment) during a mean follow-up time of 18.7 months (148). The

combined risk of rebleeding is thus approximately 2.7%. The risk factors for early rebleeding were aneurysm location in AcomA, incomplete aneurysm occlusion, and poor clinical condition at the time of treatment (149).

COMPARATIVE STUDIES OF ENDOVASCULAR AND SURGICAL TREATMENT:

Accurate comparison of endovascular and surgical treatment methods is possible only in a prospective, randomized study. By now, two studies comparing endovascular and surgical management of ruptured intracranial aneurysms fulfill that criterion. In a single center study performed in Kuopio University Hospital, Finland, 109 patients (52 in the endovascular and 57 in the surgical group) were included (150). The international subarachnoid aneurysm trial (ISAT) consisted of 2143 patients (1073 in the endovascular and 1070 in the surgical group) treated in 42 centers, mainly in the United Kingdom and elsewhere in Europe (147).

In the Kuopio study, 46% of the patients experiencing aneurysmal SAH during the study period were randomized. The most important reasons for exclusion were a large haematoma (27%) and aneurysm morphology unsuitable for coiling (25%). The initial angiographic results were significantly better after surgery in the patients with ACA aneurysms and after endovascular treatment in those with posterior circulation aneurysms. In MCA or ICA aneurysms, no significant differences were seen. The procedural mortality rate was 2% in the endovascular group and 4% in the surgical group. Clinical outcome at 3 months or at 1 year was not significantly different between the groups. One patient (2%) in the endovascular group had early rebleeding, while no rebleeding were detected in the surgical group. The patients eligible for neuropsychological analysis did not differ in their test scores. In post procedural MRI, superficial brain retraction deficits and ischemic lesions in the territory of the treated aneurysm were more common in the patients treated with surgical clipping than in those who had undergone endovascular treatment (150).

In the ISAT study, only 22.4% of the patients experiencing acute SAH during the study period could be randomized. The 1- year outcome in terms of survival free of disability turned out to be significantly better in the patients allocated to endovascular coiling (76.5%) compared to those allocated to neurosurgical clipping (69.1%). The absolute risk reduction in dependency or death in the endovascular versus surgical groups was 7.4%. The early survival advantage was

maintained for up to seven years and was statistically significant ($p=0.03$). The risk of seizures after the treatment procedure was substantially lower in the patients allocated to endovascular treatment. The risk for rebleeding was higher among the patients allocated to endovascular treatment (3.2%) than in the surgical group (1.2%). However, the results of the ISAT study reflect only the subgroup of SAH patients eligible for either of the two treatment options (147).

Only a few reports were found in the literature comparing the economic aspects of endovascular and surgical treatments of intracranial aneurysms. In another analysis, a subgroup of patients randomized to the ISAT study (10 patients in the endovascular and 12 patients in the surgical group) was analyzed by cost and outcome in a single center (151). While the endovascular procedure tended to be the more expensive in terms of the cost of consumables, this expense was more than compensated for by savings in staffing costs and the period and cost of hospitalization. Endovascularly treated patients returned to normal activity or paid employment sooner and had a favorable functional outcome compared with patients scheduled for a neurosurgical procedure (151).

One of the major concerns of endovascular treatment is substantial proportion of aneurysms that initially are occluded incompletely and possibility of reopening of coiled aneurysm over time with inherent risk of bleeding. Recently published results of long term follow up of ruptured intracranial aneurysms in International Subarachnoid Aneurysm Trial (ISAT) have demonstrated that although endovascular coiling of ruptured intracranial aneurysms has a significant advantage over neurosurgical clipping in the first year after treatment, after 5 years there was a small risk of recurrent bleeding from a coiled aneurysm compared with a clipped aneurysm, although the risk of death at 5 years was significantly lower in the coiled group than it was in the clipped group(95). Consequently, endovascular coiling of aneurysms has become accepted as an alternative to surgical clipping, with lower morbidity and mortality rates in some cases, particularly in posterior circulation aneurysms (152,153,154,155). There is ongoing progress in the field of endovascular therapy for intracranial aneurysms with development of new coil design and endovascular devices.

Aneurysms of intracranial vertebral artery

Vertebral artery (VA) aneurysms constitute 0.5 to 3% of intracranial aneurysms and 20% of posterior circulation aneurysms (156). They pose significant challenge due to difficulty in the surgical approach, which is further enhanced by the relative inexperience of most neurosurgeons with these lesions due to their rarity. These aneurysms are usually located in intracranial segment of vertebral artery (V4) at the vertebral artery - posterior inferior cerebellar artery (VA-PICA) junction or slightly distal to the origin of PICA (157). These aneurysms are mostly dissecting in nature. Acute dissecting aneurysms are usually small in size and presents with hemorrhage while chronic dissecting aneurysms are usually large or giant in size and present with mass effect (158). Due to high risk of rebleeding and poor prognosis of ruptured acute dissecting aneurysms, treatment is mandatory (159). Although endovascular selective treatment of aneurysmal sac is usually feasible, the best option remains endovascular occlusion of the parent artery at the dissection site and trapping of aneurysm using coils (160,161,162). Intradural dissecting aneurysms should be occluded at the dissection site to minimize the risk of recanalization from collateral vessels (161). Stent-supported coil embolisation has been advocated, especially in cases in which the occlusion of the parent artery is not tolerated during the occlusion test or contraindicated because of a disease involving the contra lateral vertebral artery (162,163,164). The use of double stent placement with two overlapping stents without additional positioning of coils also has been reported (165,166). The reconstructive approach using stenting maintains the patency of the parent artery (158). As also reported in surgical experience, parent artery occlusion (PAO) remains the elective treatment in most chronic dissecting aneurysms (167,168).

Aneurysms of vertebrobasilar junction:

Aneurysms located at the vertebrobasilar junction are uncommon lesions and are often associated with fenestration of the proximal basilar artery (169,170). Most patients present with SAH (171).

The relation of vertebrobasilar junction aneurysm with basilar fenestration is well established and explained by intrinsic defects in the medial vessel wall of fenestrated arteries. In the fetus, the basilar artery is formed by fusion of bilateral longitudinal neural arteries during the fifth gestational week. During this fusion process, temporary bridging arteries connecting the longitudinal neural arteries regress as fusion is completed. If these bridging arteries persist, they result in fenestration of the basilar artery (172,173). The lateral walls of the fenestrated artery have a normal intrinsic architecture. The medial walls, however, have focal defects at both ends of the fenestration that may lead to aneurysm formation similar to cerebral artery bifurcations (171).

Surgical access to the vertebrobasilar junction is difficult, and local anatomy is complex due to the presence of perforators to the brain stem and lower cranial nerves (174,175,176,177). Although surgical approach to the vertebrobasilar junction is difficult, endovascular access is easy & therefore, coil occlusion of aneurysmal sac is the treatment of choice with good initial and midterm results (178,179). Endovascular occlusion of the vertebral artery following test occlusion is a safe and effective treatment for aneurysms of the vertebrobasilar junction (180). In selected cases with insufficient caliber of the posterior communicating arteries, unilateral or bilateral stent placement over the vertebrobasilar junction can be considered to preserve ante grade flow to the basilar artery (171).

Aneurysms involving posterior inferior cerebellar artery(PICA):

Aneurysms of the posterior inferior cerebellar artery (PICA) are uncommon, comprising approximately 0.49% of all intracranial aneurysms (181) and 20% of the posterior fossa aneurysms (182). In contrast to vertebral aneurysms located at origin of PICA, true PICA aneurysms are located either proximally or distally at PICA itself (7). The PICA has the most complex, tortuous and variable course, as well as supplying area, of all cerebellar arteries (183).

In most cases, it arises from the VA at its intracranial portion (80-95% of cases), passes laterally to the medulla and inferiorly to the olive, thus taking a posterior direction to the biventral lobule of the cerebellum (184,185).

In Yasargil series, from 15 aneurysms of PICA, 10 were originated from the origin of the vessel (186). The PICA can be divided as five segments based on its relationship to the medulla oblongata and the cerebellum. The five segments are 1) anterior medullary segment, extending from the origin of PICA at VA to inferior olivary prominence 2) lateral medullary segment, extending to the origins of ninth, 10th, 11th cranial nerves, 3) tonsillomedullary segment, extending to the level of the tonsillar midportion, 4) telovelotonsillar segment, extending to the cortical surface of cerebellum and 5) cortical segment, extending to the cerebellar vermis and hemisphere (187,188).

Surgery of PICA aneurysms is challenging because they are deeply located in front of the brain stem and surrounded by the lower cranial nerves. Postoperative temporary or permanent lower cranial nerve palsies occur in almost half of the cases(9) with prolonged need for intensive care and inherent risk of developing pneumonia (189). Geometry of some PICA aneurysms precludes selective occlusion of the aneurysm with coils preserving flow to the PICA. General techniques to treat wide-necked aneurysms such as balloon or stent-assisted coiling are often impossible in PICA aneurysms; deconstructive techniques with PICA occlusion or vertebral occlusion are the only endovascular options in these wide neck aneurysms(190). Occlusion of PICA is associated with a very low morbidity due to excellent collateralization of the cerebellum via the AICA and SCA(7).

Aneurysms involving basilar trunk:

Saccular aneurysms of the midbasilar segment (basilar trunk) are rare lesions with an incidence of less than 1% of intracranial aneurysms (191). Among vertebrobasilar aneurysms, approximately 8% are on the basilar trunk (192). Direct surgical clipping of basilar trunk aneurysms is technically highly challenging and carries significant surgical risks, mainly due to limited exposure and view of the aneurysm and the vulnerability of vital perforating arteries

arising from the basilar artery (193). Surgical or endovascular basilar or vertebral artery occlusion as a treatment has proved an effective alternative in certain cases, such as in fusiform aneurysms (194,195,196). For saccular aneurysms or those not suitable for parent vessel occlusion, selective endovascular treatment with detachable coils can be considered (197,198,199). General drawbacks of coiling are the proportion of aneurysms that cannot initially be occluded completely and the possibility of coil compaction or thrombus resolution over time leading to reopening of the aneurysm lumen, particularly in large and giant aneurysms (191). In case of broad based aneurysms stent assisted coiling to bridge the neck might be necessary. During last few years it has become obvious that majority of basilar trunk aneurysms are probably of dissecting nature and stent assisted coiling might be necessary in these cases to avoid further recanalization and regrowth of aneurysmal sac (7).

Giant aneurysms by definition are larger than 2.5 cm in maximal diameter (200). When a fusiform aneurysm is located in the basilar trunk, its treatment is quite challenging (201). In a large surgical series of 32 cases of giant basilar trunk aneurysms of the fusiform type, the main treatment strategy was proximal basilar artery occlusion or trapping, resulting in seven cases of mortality and five cases of morbidity (167). In another series, there were three mortalities among eight cases of basilar artery fusiform aneurysm (202). There have been several reports of fusiform basilar artery aneurysms treated by endovascular means, using coils and/or stents (203,204,205). In eccentrically bulging, broad neck aneurysms, the placement of a flexible stent, followed by coil embolization, may be a legitimate treatment option (203,204). In giant fusiform aneurysms, stent placement followed by stent graft placement may also be an alternative treatment option (204). Stent placement has the advantage of allowing the maintenance of the parent arterial lumen, but clinical use of this technique is limited by the difficulty associated with the endovascular navigation of stents into the tortuous cerebral vasculature (204).

Parent vessel occlusion by surgical or endovascular means may be another treatment option (195,206). In a series of vertebrobasilar aneurysms treated by parent vessel occlusion, delayed neurological complication was reported in 45 % of the cases and the majority of these complications were found to be fatal (195). Furthermore, in aneurysms involving the basilar artery, complete thrombosis is often not achieved by the occlusion of the parent vessel (206). In determining the safety of basilar artery occlusion, it is known that the most important factor is

the presence and size of the PCoAs, and those patients in whom both PCoAs were more than 1 mm in diameter showed better outcomes (195).

Flow diverter devices like pipeline embolisation device and silk stent provides a safe and definitive constructive treatment option for large, giant, and fusiform/circumferential aneurysms. The PED can achieve complete aneurysm occlusion without embolization coils. When applied judiciously, the PED may be used safely in vascular segments that give rise to eloquent perforators (23).

Basilar top aneurysms:

Basilar top aneurysms are the most common type of aneurysms in the posterior circulation (51%), although they are relatively rare and only account for 5%–8% of all intracranial aneurysms(70,207). Since the introduction of detachable coils in the early 1990s, endovascular coiling of basilar tip aneurysms has rapidly replaced surgery for these lesions. Surgical approaches for basilar tip aneurysms are more challenging than for aneurysms at other locations, as these aneurysms are deeply located, confined in the narrow interpeduncular fossa in close proximity to the midbrain, and surrounded by important perforating arteries. As a consequence, surgery is associated with substantial morbidity (208,209,210). In several previous studies, the safety and effectiveness of coiling of basilar tip aneurysms has been established, in addition to the safety and effectiveness of large aneurysms and for patients in poor clinical grades (211,212,213). Coiling of basilar tip aneurysms is associated with low morbidity and adequately protects against recurrent or primary hemorrhage (214).

The problem posed by a wide-necked basilar bifurcation aneurysm is different from that of a wide-necked, sidewall internal carotid artery (ICA) aneurysm. Prior stent-assisted coiling of basilar apex aneurysms involved deploying a stent into only one PCA, with the proximal end in the basilar artery thereby "jailing" the contra lateral PCA. This technique provides only partial protection from coil herniation into the parent vessel (215). Another hypothetical concern regarding this asymmetric stent deployment would be the alteration of flow favoring the PCA in which the distal end of the stent was deployed (215). To solve the

problem of partial protection, Y-configuration double-stent technique may reconstruct the basilar apex by deploying a second stent through the interstices of the first stent thereby reconstructing the major parent vessel and protecting the entire aneurysm neck from coil herniation and coil compaction(215). Many investigators believe that incompletely coiled basilar terminus aneurysms are particularly prone to re-canalization secondary to the hemodynamics of the parent artery and the resultant high-pressure pulsatile flow directly at the coil mass (216). The placement of both stents may alter the flow pattern at the aneurysm neck, redirecting flow toward the PCAs to potentially reduce the likelihood of recanalization(215).

Aneurysms of posterior cerebral artery:

Aneurysms of the posterior cerebral artery (PCA) represent approximately 1% of all intracranial aneurysms (217). Aneurysms arising from the PCA have a predilection for the P1 and P2 segments (218). Aneurysms on the proximal P1 segment may impede on the oculomotor nerve which courses between the P1 and the proximal superior cerebellar artery (219).

The surgical approach and dissection of the PCA is technically challenging owing to the complexity of its perforating branches and their intimate relationship with the cranial nerves and upper brain stem (218). Selective catheterization of the PCA and endovascular treatment of aneurysms arising from its different segments is technically feasible, offering a viable alternative to the surgical approach (220). Endovascular treatment with the GDC coils varies depending on the nature of the aneurysm (ie, saccular, fusiform, or dissecting), and its location along the different anatomic segments of the PCA(218).

A precise knowledge of the segmental anatomy of the PCA and its branches is essential when the surgical or endovascular approach to an aneurysm is planned, particularly if parent vessel occlusion is contemplated (218).

The PCA can be subdivided into four anatomic segments: P1 segment extends from the tip of the basilar artery to the origin of the posterior communicating artery. The P2 segment extends from the posterior communicating artery to the dorsal aspect of the midbrain; this P2 segment can be further subdivided into anterior (P2A) and posterior (P2P) segments. The P3 segment

extends from the lateral aspect of the quadrigeminal cistern at the origin of the posterior temporal artery, to the anterior limit of the calcarine fissure. The P4 segment consists of the terminal cortical branches of the PCA, after the take off of the parietooccipital and calcarine arteries. Each of these segments gives off groups of branches that supply distinct anatomic territories: brain stem and thalamic branches, ventricular branches, and cortical branches (221,222,223)

PCA aneurysms frequently affect young patients, with an average age of 38 years (224). This is younger than the average age of 50 to 60 years for aneurysms occurring at other anatomic sites. Also, there is a higher incidence of large and giant aneurysms (almost 23% of PCA aneurysms, versus 3–5% at other anatomic sites) affecting the PCA (225).

The most common clinical presentation of PCA aneurysms described in the literature is the SAH (80%) (217,226). Other presentations include, oculomotor palsy, visual field deficit, or a combination (219).

Saccular aneurysms can safely and effectively be treated with selective occlusion with coils and sparing the parent PCA with good long-term anatomic results (219). Occlusion of the aneurysm together with the parent PCA with coils is an effective treatment for fusiform and dissecting aneurysms that is usually tolerated well as a result of adequate collateral circulation via the anterior choroidal artery, the superior cerebellar artery, and leptomeningeal collaterals from middle and anterior cerebral artery (219). Dissecting aneurysms that occlude the PCA and saccular aneurysms with intramural thrombus and dissection may reopen exposing the patient to the risk of recurrent SAH (219).

AIMS AND OBJECTIVES:

1) TO ANALYSE THE CLINICAL MANIFESTATIONS AT PRESENTATION & OUTCOME AT FOLLOW UP OF ENDOVASCULAR TREATMENT OF POSTERIOR CIRCULATION ANEURYSMS.

2) TO ANALYSE THE RESULTS, OUTCOME AND REBLEED IN PATIENTS WITH POSTERIOR CIRCULATION ANEURYSMS WHO UNDERWENT ENDOVASCULAR MANAGEMENT AS PRIMARY TREATMENT OPTION.

MATERIALS AND METHODS:

From October 1998 to September 2010, a total of 33 patients with mean age of 43 years (range of 7-70 years) consisting of 20 males and 13 females presenting with various clinical manifestations and clinical/imaging features suggestive of posterior circulation aneurysms to department of Imaging Sciences and Interventional Radiology (previously Department of Radiology), Sree Chitra Tirunal Institute for Medical Sciences And Technology, Thiruvananthapuram underwent preprocedural diagnostic evaluation and endovascular treatment of intracranial aneurysm with use of coils with/without stent/balloon assisted techniques. The study was retrospective as well as prospective in nature.

The patient selection for endovascular treatment was made by combined opinion consisting of neurointerventional radiologist(s) and neurosurgeon(s). The treatment decision was made on the basis of the location, size, shape and neck of the aneurysm and the patient's clinical status and co morbidities. The patient and his/her relatives were informed about the disease, the treatment options available, and the risks included in the treatment. Endovascular treatment was usually chosen if it seemed feasible. There were no absolute exclusion criteria for endovascular treatment, but surgical treatment was favored if :1) a large intracranial hematoma is present in a poor-grade patient necessitating emergency craniotomy and haematoma evacuation 2) one or more parent artery branches originating from the aneurysm sac and 3) when the posterior circulation vessels are very tortuous.

All patients hospital records, outpatient reports, procedural records, all angiographic, computed tomography and magnetic resonance imaging were carefully subjected to retrospective analysis and were systematically reviewed. All the patients were followed up with clinical examination, repeat angiography when required & Time of Flight Magnetic Resonance Angiography (TOF MRA) sequence were scheduled depending upon clinical course of patient and whether the aneurysm was fully/partially occluded with coils in first sitting. All the coiled patients were advised to come for follow up after 3 months, 6 months and thereafter every 6th month for 1 year and then every yearly. Close follow up were done in ruptured aneurysms with initial incomplete coiling.

Out of 33 patients, five patients had aneurysms of distal vertebral artery, one patient had aneurysm at vertebrobasilar junction with fenestration, one patient had PICA aneurysm, five had aneurysms of basilar trunk, 15 had aneurysms of basilar top and 6 had aneurysms of posterior cerebral artery. Endovascular treatment of these aneurysms were done using coil

with/without stent/balloon assisted techniques along with parent artery sacrifice in certain aneurysms.

CLINICAL EVALUATION: Patients' clinical status was retrospectively analyzed from patient records. The H&H (Hunt & Hess 1968) grading system was used in the evaluation of the pre-embolization clinical status of patients with ruptured aneurysms. Within the group of 32 patients, those aneurysms with evidence of bleed presenting with subarachnoid hemorrhage/intraparenchymal hemorrhage due to a ruptured aneurysm, the amount of blood visible in CT images was graded using Fisher grading scale (Fisher et al. 1980). Follow up analysis of endovascularly treated patients was done using Modified Rankin Score & Glasgow Outcome Scale.

Table 1:

Hunt & Hess grading scale for evaluating the clinical condition of patients with a history of subarachnoid hemorrhage (Hunt & Hess 1968).

Grade Description

- 0 No bleeding
- 1 Asymptomatic, or minimal headache and slight nuchal rigidity
- 2 Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
- 3 Drowsiness, confusion, or mild local deficit
- 4 Stupor, moderate to severe hemiplegia, possibly early decerebrate rigidity, and vegetative disturbances
- 5 Deep coma, decerebrate rigidity, moribund appearance

Table 2:

Fisher grading scale for evaluating the amount of blood in CT scan (Fisher et al. 1980).

Grade Description

0	No CT scan
1	No SAH
2	Diffuse SAH, less than 1 mm thick
3	Localized clots of layers of hemorrhage 1mm or thicker
4	IVH or ICH with or without SAH
5	SAH, unknown grade

Table 3:

Glasgow Outcome Scale for assessing clinical outcome after treatment (Jennet & Bond 1975).

<u>Grade</u>	<u>Brief description</u>	<u>Full description</u>
5	Good recovery	Full and independent life, no or minimal neurological deficit
4	Moderately disabled	Moderately disabled, neurological deficit, or intellectual impairment, but independent life
3	Severely disabled	Conscious, but totally dependent on others
2	Vegetative stage	Vegetative survival
1	Dead	Dead

Table 4:

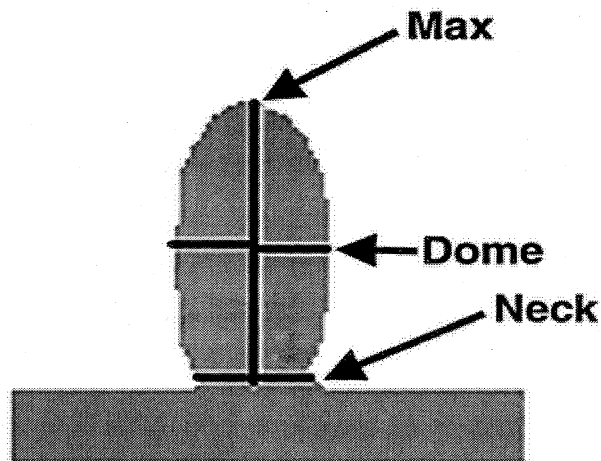
Modified Rankin Score

SCORE	DESCRIPTION
0	No symptoms at all
1	No significant disability despite symptoms; able to carry out all usual duties and activities
2	Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance
3	Moderate disability; requiring some help, but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent and requiring constant nursing care and attention
6	Dead

ANGIOGRAPHIC ANALYSIS:

Diagnostic cerebral angiogram was done in all patients prior to endovascular treatment to evaluate the aneurysm characteristics, presence or absence of vasospasm, presence of vascular anomalies and excessive vascular tortuosity. All the aneurysms visible on DSA images were analyzed by location, size, shape, rupture, and etiology. If the patient had had SAH, and multiple aneurysms were found, the ruptured one was judged according to the location of hemorrhage in CT images and the angiographic appearance of the aneurysms in DSA images (larger, proximal, aneurysm with a tail on its margin). Aneurysms visualized on DSA were characterized based on

morphology as saccular, fusiform or fusosaccular aneurysms. The size of the aneurysm was measured directly from 2D DSA images 3D or rotational angiographic reconstruction images, when available, calibrated to the extra cranial markers. Radiopaque rings 10 mm in diameter were used for calibration, and they were taped on the patient's skin on one side of the head. Dome neck ratio was calculated from DSA images as width across the dome divided by width across the neck for each aneurysm prior to coiling and aneurysms were classified as narrow neck aneurysms(dome neck ratio >2) and wide neck aneurysms(dome neck ratio <2).



SCHEMATIC REPRESENTATION OF DOME NECK RATIO MEASUREMENT OF ANEURYSM.

ANGIOGRAPHIC EQUIPMENT:

During majority of study period, cerebral angiographies and endovascular treatments were performed on a angiographic unit GE ADVANTIX SINGLE PLANE DSA system. A biplane angiographic unit (GE INNOVA 3131, BIPLANE UNIT) was installed in January 2010, and thereafter all cerebral angiographies and neurointerventional procedures have been performed in this suite.

ANESTHESIA AND PERIPROCEDURAL MEDICATION:

Since 1997, all the embolizations were performed under general anesthesia and were actively monitored for vital parameters. Systemic heparinization was used routinely. Initially, 5000 units of heparin were usually administered intravenously. Heparinization was individually decided by the treating interventional radiologist case by case. The level of heparinization was monitored by measuring activated clotting time (ACT) values (target 250-300 seconds) during the procedure, and additional doses of heparin every hourly were given as needed. This was considered essential as risk of thromboembolism is regarded as dominant over the risk of hemorrhage even in acute subarachnoid hemorrhage. At the end of the procedure, heparinization was partially reversed by intravenous protamine sulphate, if needed. For wide neck aneurysms in which stent assisted coiling was planned, preprocedural aspirin 75-325 mg per day and clopidogrel 75 mg per day was given for 5 days before procedure. In case of emergency aspirin 75-325 mg and loading dose of 300 mg clopidogrel was given 6 hrs prior to procedure.

EMBOLIZATION METHODS AND DEVICES:

Transfemoral catheterization (via unilateral or bilateral femoral artery puncture) was used in all cases. The sizes of the transfemorally deployed introducer sheaths ranged from 5 to 7 French (F). After confirming therapeutic anticoagulation, thin walled straight tip guiding catheters (range, from 5F to 7F) were carefully placed in dominant vertebral artery in distal V2 segment avoiding flow occlusion. Guiding catheter offers a combination of very soft flexible distal tip combined with reasonably stiff shaft providing a stable position in parent artery during micro catheter manipulations.

Using magnified real time fluoroscopy and digital road map techniques , preshaped micro catheter was placed coaxially through the guiding catheter into distal artery lumen and directed into aneurysm with help of microguidewire. At the beginning of this study period, Tracker-10 (Target Therapeutics, Boston Scientific, Fremont, CA, USA) micro catheters were exclusively

used, but later on, a large variety of micro catheters mostly Excelsior micro catheter and 0.014 inch microguidewire were used to catheterize aneurysms. Initially only GDC-10 and GDC-18 (Boston Scientific, Target Therapeutics, Fremont, CA, USA) were used for the coiling procedure and later due to availability of bioactive coils, Matrix coils (Boston Scientific, Target Therapeutics, Fremont, CA, USA) were also used along with GDC coils to pack the aneurysm fundus tightly without impinging on parent vessel. First coil was a 3D coil which was accurately matched with the fundus of aneurysm with diameter more than neck of aneurysm to form a stable basket within aneurysm forming a platform for further coil deposition. After successful detachment of first coil, subsequent coils were chosen with decreasing diameter to achieve central packing of initial basket. Before detaching each coil, DSA acquisition was obtained in AP and lateral views to detect any coil prolapse into parent vessel, to detect aneurysmal sac rupture and to guide adequacy of aneurysmal sac occlusion. Detachment was obtained by connecting ground needle electrode to groin of patient and positive electrode to end of coil wire followed by application of direct current through a battery driven compatible system and coil detachment usually occurred in 1-3 minutes. Towards the end of coiling procedure softer coils like GDC 10 system coils were used to allow for dense packing and avoiding endotension and rupture of aneurysm.

In saccular aneurysms with narrow neck, only coil embolisation was done. In cases of wide neck saccular aneurysms, either balloon assisted or stent assisted coiling of aneurysms was done. During the study period, several occlusion balloons were used for this purpose: Endeavour non-detachable balloon (Boston Scientific); Solstice Balloon Occlusion System (Medtronic MIS, Sunnyvale, CA); Sentry balloon (Boston Scientific); and Hyperform balloon (EV3, Irvine, CA).

In cases where the balloon remodeling technique was used, the balloon catheter (Sentry/hyperfoam balloon) was introduced through a 6F guiding catheter to an appropriate position in the parent artery at the level of the aneurysm neck prior to aneurysm catheterization.

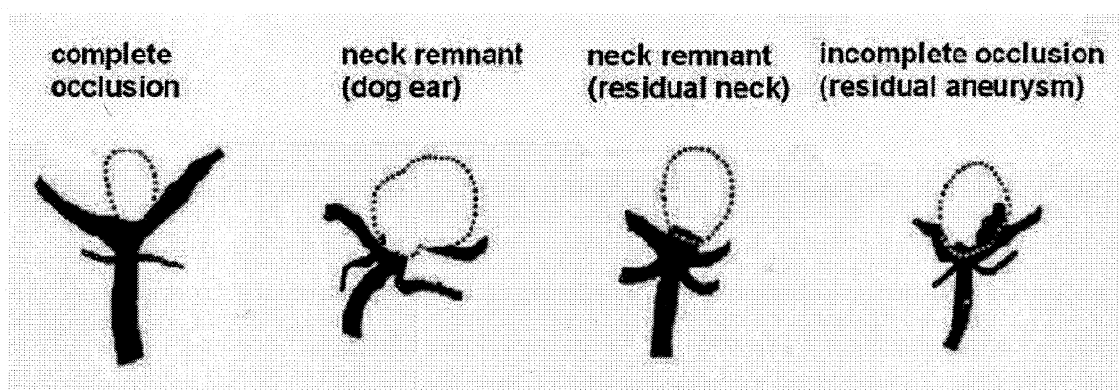
When the stent remodeling technique was used, various stents like Neuroform stent (Boston Scientific) & Leo stent (Balt, Montgomery) was deployed first, and the aneurysm was subsequently catheterized through the stent interstices with a micro catheter and packed with coils or micro catheter was jailed within aneurysm followed by deployment of stent. In fusiform

aneurysms either stent assisted coiling or occlusion of aneurysm with parent artery occlusion was done with coils.

TABLE SHOWING MATERIALS USED IN CATHLAB FOR COILING:

1.	Sheath – Radifocus, Cordis	6, 7,8 French size
2.	Diagnostic catheters – Vertebral glide (Terumo), Right coronary (Cordis), Mani cerebral (Cordis), Simmons (Cordis), Multipurpose (Cordis)	5, 6 French size
3.	Guide wire – Terumo exchange length, Terumo standard	150 cm – Standard 260 cms - Exchange
4.	Guiding catheters – Vistabrite, Launcher (Cordis), Fasguide catheter	6, 7 French size
5.	Micro catheters –Tracker, excelsior, echelon	1.5 to 1.8 French
6.	Microguidewire – Transcend, dasher, roadrunner	.010, .014
7.	Embolic materials –GDC 360degree,GDC soft, matrix coils	Coils varying from-10 system to 18 system.
8	Balloons-Hyperform,Sentry,Solistice	Various sizes
9	Stents-Neuroform,Leo stent	Various sizes

The degree of initial aneurysm occlusion was visually graded using modified Raymond Classification, where the “dog ear” and “residual neck” groups were combined as “neck remnant” (Raymond *et al.* 1997) Frontal and lateral DSA projections were also routinely obtained at the end of the treatment. A qualitative assessment of aneurysm occlusion was performed using a three-point Raymond scale: (1) “complete occlusion” meant that no contrast could be visualized within the aneurysm; (2) “neck remnant” meant when residual filling was noted in the region of the aneurysm neck only, typically between the interstices of coils in this region; and (3) “residual aneurysm” meant if any contrast was visualized extending beyond the immediate parent vessel–aneurysm interface into the aneurysm fundus. In all cases, aneurysm occlusion was interpreted on the basis of the image obtained immediately after placement of the last coil.



COMPLICATIONS, MORBIDITY AND MORTALITY:

All procedural complications, both transient and permanent, were clinically verified & corroborated with imaging findings and were recorded in case sheet.

Post-procedural complications were also verified and recorded, if they could be related to the endovascular treatment. Lethal procedure-related complications were included in procedural

mortality. All procedure-related complications causing clinical symptoms were included in procedural morbidity, if the symptoms persisted until discharge.

FOLLOW-UP

The follow-up protocol was individualized for each patient depending upon initial angiographic result at the end of the treatment and thus there was interindividual variation. The patients were followed up with clinical examination, repeat DSA angiography when required & Time of Flight Magnetic Resonance Angiography (TOF MRA) and CEMRA sequence when doubt existed on TOFMRA due to artifacts. Close follow up were done in ruptured aneurysms with initial incomplete coiling. In case of ruptured aneurysms with complete initial occlusion of aneurysm on angiography, follow up was done every 6 months for 1 year followed by every yearly evaluation & TOFMRA was done at 6 months and every yearly. In case of ruptured aneurysms with incomplete initial occlusion of aneurysm on angiography, first follow up was done within 2-3 months, then at 6th month, followed by every yearly evaluation & DSA/TOFMRA was done at first visit and TOF MRA at 6 months and then every yearly to evaluate for interval opening of aneurysm neck secondary to coil compaction. Unruptured aneurysms were usually checked at least once every yearly after the endovascular treatment. Longer follow-up was scheduled if considered indicated. Clinical follow-up of patients who refused or were unable to undergo follow-up angiography was assessed by contacting the patient's relatives by telephone or in writing at various follow-up intervals. The changes observed on follow-up angiograms were classified as further thrombosis, which was defined as a decrease in the amount of contrast agent filling the aneurysm; unchanged, defined as a similar degree of aneurysm occlusion in multiple projections; and recanalization, defined as an increase in the amount of contrast material filling the aneurysm compared with the immediate postembolization angiographic results.

The clinical status of the patients was retrospectively recorded from the patient files available at medical records section of hospital at every follow up examination. All hospital visits were analyzed. The patient outcomes were graded using the Glasgow Outcome Scale and modified

Rankin Score. The end points of clinical follow-up were death and surgical treatment of an endovascularly treated aneurysm.

DATA COLLECTION:

The following significant parameters related to patient history, aneurysm features, endovascular treatment, and follow-up findings were included for evaluation:

Patient history:

- Name, gender, and date of birth
- History of intracranial bleedings and previous endovascular or surgical interventions
- Clinical presentation of the aneurysm(s)
- Pre-procedural clinical findings of the patient (H&H)
- CT findings on Fisher grading scale

Aneurysm features:

- Location and size (length and width)
- Neck width and dome-to-neck ratio
- Involvement of parent artery
- Aneurysm margin (regular/irregular, secondary pouch)
- Date when found and imaging method used (DSA/MRA/CTA)
- Ruptured/unruptured
- Etiology (saccular/dissecting/flow-related)

Endovascular treatment:

- Date of first treatment
- Time interval from rupture to treatment
- Dates of retreatments
- Number, type, and manufacturer of device used
- Techniques used (single catheter/ balloon remodeling/ stent remodeling/ parent artery occlusion)
- Procedural and delayed complications
- Angiographic result
- Clinical outcome (GOS)
- Treatment abandonment and reason for abandonment

Follow-up findings:

- Date
- Angiographic result and imaging method (DSA/MRA)
- Clinical recovery (GOS)
- Indication for additional treatment
- Need and time for further follow-up

RESULTS:

This study was conducted in the department of Imaging Sciences and Interventional Radiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology, Thiruvananthapuram over a period of 12 years. Study was undertaken in 33 patients presenting with various clinical manifestations and clinical/imaging features suggestive of posterior circulation aneurysms who underwent endovascular management of aneurysms. Indications for referral for endovascular therapy included anticipated surgical difficulty, poor neurological or medical condition of the patient, and failed attempt at previous surgical clipping. Aneurysms were deemed potentially surgically difficult or believed to carry a higher-than-average risk for operative morbidity if one or more of the following conditions were present: difficult surgical location (vertebrobasilar junction aneurysms), high basilar artery bifurcation or posteriorly projecting dome (for basilar top aneurysms), moderate- to-severe vasospasm documented on the diagnostic angiogram, or an aneurysm neck that was wide or partially calcified.

AGE DISTRIBUTION OF PATIENTS (N=33):

TABLE 5:

AGE(YEARS)	NO. OF PATIENTS	PERCENTAGE
<20	3	10
20-40	9	30
40-60	19	60
60-80	2	10

In our study youngest patient was 7 years old and eldest patient was 70 years old. Majority of patients were in 40-60 years age group and mean age was 43 years (range of 7-70 years).

SEX DISTRIBUTION OF PATIENTS (N=33):

TABLE 6:

SEX	NO OF PATIENTS	PERCENTAGE
MALE	20	60
FEMALE	13	40

Majority of patients in our study with posterior circulation aneurysms were males (60%) and rest were represented by females (40%)

PRETREATMENT CLINICAL STATUS OF PATIENTS WITH RUPTURED ANEURYSMS GRADED BY HUNT HESS SCALE(N=33):

TABLE 7:

GRADE	DESCRIPTION	NO OF PATIENTS	PERCENTAGE
0	No bleeding	14	42
1	Asymptomatic, or minimal headache and slight nuchal rigidity	1	3
2	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy	11	33

3	Drowsiness, confusion, or mild focal deficit	7	22
4	Stupor, moderate to severe hemiplegia, possibly early decerebrate rigidity, and vegetative disturbances	0	0
5	Deep coma, decerebrate rigidity, moribund appearance	0	0

Majority of patients in our study with ruptured aneurysms prior to endovascular treatment were in Hunt Hess Grade 2 with moderate to severe headache, nuchal rigidity with/without cranial nerve palsy.

SEVERITY OF BLEEDING IN 32 PATIENTS GRADED BY FISHER GRADING SCALE.(N=33)

TABLE 8:

GRADE	DESCRIPTION	NO.OF PATIENTS	PERCENTAGE
0	No CT scan	9	28
1	No SAH	5	15
2	Diffuse SAH, less than 1 mm thick	13	39
3	Localized clots of layers of hemorrhage 1mm or thicker	6	18
4	IVH or ICH with or without SAH	0	0
5	SAH, unknown grade	0	0

Most of patients with posterior circulation aneurysms presenting with SAH were in grade 2 showing CT evidence of diffuse SAH, less than 1mm thick accounting to 39%.

CHARACTERISTICS OF ANEURYSMS (N=33):

TABLE 9:

MORPHOLOGY	NO OF PATIENTS	PERCENTAGE
SACULAR	20	60
FUSIFORM	8	25
FUSOSACCULAR	5	15

Majority (60%) of posterior circulation aneurysms in our study were sacular in morphology and were located mostly in basilar artery tip.

TABLE 10(N=33):

NECK OF ANEURYSM	NO. OF PATIENTS	PERCENTAGE
NARROW NECK(DOME NECK RATIO>2)	11	33
WIDE NECK(DOME NECK RATIO<2)	22	67

Posterior circulation aneurysms with narrow neck constituted approx 33% o all aneurysms in our study and narrow neck aneurysms were best suited for endovascular management using coils only.

MANIFESTATION OF ANEURYSMS (N=33):

TABLE 11:

MANIFESTATION	NO OF PATIENTS	PERCENTAGE
RUPTURE	19	58
MASS EFFECT	5	15

Rupture of aneurysms manifesting as SAH was seen in approx 58% of patients with posterior circulation aneurysms and mass effect as manifestation was present in 15% of patients. Majority (11) of ruptured aneurysms were saccular in nature located at basilar top and one saccular aneurysm was located at basilar trunk between SCA and PCA & one ruptured aneurysm of basilar trunk (patient no. 17) was exerting mass effect on pons in addition to SAH and 6 ruptured aneurysms were fusiform in morphology located at PCA, intracranial vertebral artery and vertebrobasilar junction.

CLINICAL MANIFESTATIONS AT TIME OF PRESENTATION(N=33):

TABLE 12:

CLINICAL FEATURES	NO OF PATIENTS	PERCENTAGE
Sudden severe headache with vomiting,	20	60
Recurrent mild headache	6	18
Giddiness & vertigo	3	9

diplopia	1	3
seizures	1	3
Hemi facial spasm	1	3
Numbness&paresthesia	1	3
Left hemiparesis and diplopia along with severe headache	1	3

Maximum number of patients (20 patients) in our study had sudden onset severe headache with vomiting and drowsiness and these patients had evidence of ruptured posterior circulation aneurysms. One patient (patient no. 20) had presented with SAH due to rupture of saccular aneurysm located at basilar trunk between SCA and PCA and due to severe vasospasm of PCA, developed left hemiparesis and diplopia on 6th day.

LOCATION OF ANEURYSMS(N=33):

TABLE 13:

LOCATION	NO OF PATIENTS	PERCENTAGE
BASILAR TOP	15	46
POSTERIOR CEREBRAL	6	18
BASILAR TRUNK	5	15
V4 VERTEBRAL	5	15
PICA	1	3
VERTEBROBASILAR JUNCTION	1	3

Approx 46% of aneurysms in our study were located at basilar top followed by posterior cerebral artery. Approx 15% of aneurysms were located at basilar trunk and intracranial segment of vertebral artery. PICA aneurysm was seen in 1 patient with cerebellar AVM wherein the aneurysm was located at origin of left PICA. One aneurysm was located at vertebrobasilar junction associated with fenestration. Basilar top aneurysms were saccular in morphology whereas basilar trunk, PCA and vertebral artery aneurysms were mostly fusiform or fusosaccular in morphology. PICA aneurysm was saccular in morphology and was flow related aneurysm in relation to left cerebellar hemispheric AVM. Vertebrobasilar junction aneurysm associated with fenestration was saccular in morphology.

TIME INTERVAL BETWEEN CLINICAL MANIFESTATION AND ENDOVASCULAR TREATMENT OF ANEURYSM(N=33):

TABLE 14:

DURATION	NO OF PATIENTS	PERCENTAGE
0-3 DAYS	3	9
4-7 DAYS	4	13
8-20DAYS	9	27
21-30 DAYS	7	21
>30 DAYS	10	30

After the onset of bleed, majority of patients underwent endovascular coiling within 8-20 days from ictus. However overall majority of patients underwent treatment more than 1 month after onset of clinical symptoms.

SIZE OF ANEURYSM(MAXIMUM DIAMETER) ON DSA(N=33):

TABLE 15:

MAXIMUM DIAMETER	NO. OF PATIENTS	PERCENTAGE
<7mm	6	18
7-12mm	15	46
13-24mm	8	24
>25mm	4	12

Maximum diameter of aneurysms in our study as analyzed by DSA was 7-12mm constituting approx 46% of aneurysms.

ENDOVASCULAR TREATMENT OF ANEURYSMS(N=33):

TABLE 16:

ENDOVASCULAR TREATMENT	NO OF PATIENTS	PERCENTAGE
COILS ONLY TO OCCLUDE ANEURYSM SAC	13	40
STENT ASSISTED COILING	6	18
BALLOON ASSISTED COILING	5	15
PARENT ARTERY OCCLUSION AS PRIMARY TREATMENT	9	27

Coils alone were used to occlude the aneurysmal sac in 13 patients particularly in aneurysms with narrow neck (dome to neck ratio >2). Stent assisted coiling was done in 6 patients and balloon assisted coiling was done in 5 patients with wide necked aneurysms. Parent artery occlusion was done as primary modality of treatment using coils in cases of fusiform aneurysms. In one patient (patient no. 19), stent assisted coiling of giant basilar trunk aneurysm was done with extension of stent into right VA along with parent artery occlusion of left VA distal to PICA origin.

INITIAL (IMMEDIATE POSTPROCEDURE) ANGIOGRAPHIC OCCLUSION (N=33) :

TABLE 17:

ENDOVASCULAR TREATMENT	COMPLETE OCCLUSION	INCOMPLETE OCCLUSION
COILS ONLY TO OCCLUDE ANEURYSM SAC	7(21%)	6(18%)
STENT ASSISTED COILING	2(6%)	4(12%)
BALLOON ASSISTED COILING	3(9%)	2(6%)
PARENT ARTERY OCCLUSION AS PRIMARY TREATMENT	7(21%)	2(6%)

Coils alone were used in 13 patients to occlude the aneurysmal sac and complete occlusion was angiographically achieved in 7 patients immediate post procedure and in remaining 6 patients there was residual small neck remnant which was not treated due to risk of coil prolapse into parent artery and subsequent parent artery occlusion. Stent assisted coiling was done in 6 patients out of which immediate complete angiographic occlusion was achieved in 2 patients and in remaining 4 patients there was minimal persisting filling of residual aneurysm and

aneurysmal neck. Complete angiographic occlusion was achieved in 3 patients after treatment with balloon assisted coiling in wide neck aneurysms. Complete occlusions among aneurysms treated with the balloon remodeling technique were less common than among the cases where standard coiling was used (9% vs. 22% Table 17). However the data is small to fully validate the results. The percentages of complete occlusion among the aneurysms treated with parent artery occlusions (22.0%) was same as that of standard coiling (22%) in our study.

TREATMENT RELATED COMPLICATION IN PATIENTS TREATED WITH ENDOVASCULAR OCCLUSION OF ANEURYSMS(N=33):

TABLE 18:

TREATMENT RELATED COMPLICATION	NO OF PATIENTS	PERCENTAGE
PROCEDURAL:		
microguidewire tip break and lodged in parent artery	2	6%
POSTPROCEDURAL		
left 3 rd nerve palsy	1	3%
left hemiparesis	2	6%
left hemianopia	1	3%

In 2 patients(patient no. 16 &19) there was breakage of roadrunner 0.014inch microguidewire tip used for stent assisted coiling of basilar trunk aneurysm and got dislodged in right PCA P2 segment. However these patients were on antiplatelet agents post procedure and no post procedure neurodeficits developed on follow up. One patient(patient no. 25)who had presented with SAH secondary to rupture of right PCA fusiform aneurysm developed left hemiparesis and hemianopia postproceure due to occlusion of right PCA. Another patient (patient no. 16) who

had presented with partially thrombosed basilar trunk aneurysm with mass effect on pons and midbrain with signal changes in pons and midbrain underwent stent assisted coiling of fusosaccular aneurysm and was on antiplatelet agents postprocedure, however on 6th day postproceure he developed acute onset weakness of left upper and lower limb with slurring of speech and MRI diffusion weighted images showed acute infarct in right half of pons and midbrain with MRA showing good distal flow .

FOLLOW UP OF COILED ANEURYSMS:

The lengths of follow-up for 28 posterior circulation aneurysms are summarized in Table 19. Majority (18 patients) had follow up of more than 1 year. At least one DSA was performed at follow-up on 11 treated aneurysms (40% of treated aneurysms,). The remaining aneurysms were followed up with TOFMRA images in 10 patients (37%) and CEMRA in 6 patients (22%) when significant artifacts occurred in TOFMRA or when doubt existed on TOFMRA. The causes for missing angiographic follow-up are summarized in Table 20.

DURATION OF FOLLOW UP OF COILED ANEURYSMS(N=28):

TABLE 19:

DURATION OF FOLLOW UP	NO OF PATIENTS	PERCENTAGE
<6MONTHS	2	7
6MONTHS-1 YEAR	8	29
1-2YEARS	4	14
2-4YEARS	4	14
4-6 YEARS	5	18
6-8 YEARS	2	7
8-10 YEARS	3	11

REASONS FOR MISSING FOLLOW UP (N=5):

TABLE 20:

REASON FOR MISSING FOLLOW UP	NO OF PATIENTS
Patient died before follow up angiography	3
Follow up angiography schedule after 3 months at later date in 2010.	2

One patient (patient no. 3) with basilar top & SCA aneurysm who underwent coiling of basilar top aneurysm rebled and died 6 days postprocedure. Another patient (patient no. 8) presenting with unruptured basilar top aneurysm died 2 days post coiling due to myocardial infarction. Another patient (patient no. 29) with ruptured intracranial vertebral artery aneurysm died 3 days post procedure due to cardiac arrest.

FOLLOW UP ANGIOGRAPHIC (DSA/TOFMRA/CEMRA)RESULTS IN 27 COILED ANEURYSMS OF POSTERIOR CIRCULATION:

TABLE 21:

<u>TREATMENT</u>	<u>IMMEDIATEPOSTPROCEDURE(n=33)</u>			<u>AT FOLLOW UP(n=27)</u>		
	complete occlusion	incomplete occlusion residual neck	residual aneurysm	Complete Occlusion	incomplete occlusion Residual Neck	residual aneurysm
COILS ONLY TO OCCLUDE ANEURYSM SAC						
-RUPTURED						
-UNRUPTURED	4(12%)	4(12%)	1(3%)	2(7%)	6(21%)	
	3(9%)		1(3%)	3(10%)	1(3%)	
STENT ASSISTED COILING						
-RUPTURED			2(6%)	1(3%)		
-UNRUPTURED	2(6%)		2(6%)	1(3%)		2(7%)
BALLOON ASSISTED COILING						

-RUPTURED					
-UNRUPTURED					
	2(6%)	2(6%)	2(7%)	2(7%)	
	1(3%)				
PARENT ARTERY OCCLUSION AS PRIMARY TREATMENT					
-RUPTURED					
-UNRUPTURED	5(15%)		4(12%)		
	2(6%)	2(6%)	2(6%)	1(3%)	

GLASCOW OUTCOME SCALE AT FOLLOW UP IN ALIVE PATIENTS(N=28) :

TABLE 22:

GRADE	DESRPTION	NO. OF PATIENTS	PERCENTAGE
5	Good recovery	27	96%
4	Moderately disabled	1	4%
3	Severely disabled	-	-
2	Vegetative stage	-	-
1	Dead	-	-

MODIFIED RANKIN DISABILITY SCORE AT FOLLOW UP IN ALIVE PATIENTS(N=28):

TABLE 23:

GRADE	DESCRIPTION	NO OF PATIENTS	PERCENTAGE
0	No symptoms		
1	No significant disability despite symptoms	26	92%
2	Slight disability; unable to carry out all previous activities	1	4%
3	Moderate disability; requiring some help, but able to walk without assistance	1	4%
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance	-	-
5	Severe disability; bedridden, incontinent and requiring constant nursing care and attention	-	-
6	Dead	-	-

REBLEEDING (N=29):

TABLE 24:

ANEURYSM	NO OF PATIENTS	PERCENTAGE
BASILAR TOP +SCA ANEURYSM	1	3%

1 patient (patient no. 3) with basilar top aneurysm and SCA origin saccular aneurysm had presented with SAH and DSA revealed aneurysm of SCA and Basilar top and stent assisted coiling of basilar top aneurysm was done of basilar top aneurysm 4 days post ictus as it thought that basilar top aneurysm was cause of bleed due to tit on its surface. Immediate post procedure DSA showed small residual neck filling .However patient rebled 6 days later with CT showing diffuse SAH and patient died due to diffuse cerebral edema, cardiac dysfunction.

BASILAR TOP ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

Out of 33 patients, 15 patients had presented with basilar top aneurysms. The study population with basilar top aneurysms consisted of 10males (66%) and 5 females (34%) with mean of 49 years and range of 35-66 years. Out of 15 patients, 11 patients (73%) had ruptured aneurysms and 4 patients (27%) had unruptured aneurysms, with CT Fischer score of 2 in majority (7 patients) and majority of patients (7 patients) were in Hunt Hess grade 2 at presentation. The mean time between SAH and presentation in 11 patients with ruptured aneurysms was 30 days with a range of 3-150 days. Two patients (13%) had presented with multiple aneurysms. Vasospasm was seen in 5 patients with ruptured aneurysms. Clinical presentation of patients with basilar top aneurysms included sudden severe headache, vomiting, vertigo, giddiness and loss of consciousness in varying combination. In one patient (patient no. 14) endovascular coiling was done following clipping due to residual aneurysm after clipping

ANEURYSM CHARACTERISTICS AND MORPHOLOGY:

Out of 15 patients with basilar top aneurysms, all were saccular in morphology and 8 basilar top aneurysms were narrow neck with dome-neck ratio of >2.10 (66%) out of 15 aneurysms were between 7-12 mm in maximum diameter and 4 (27%) aneurysms were <7 mm in maximum diameter and 1 (6%) aneurysm was 13-24 mm in maximum diameter. Vasospasm was seen in 5 (33%) patients with ruptured aneurysms. In one patient (patient no. 1) there was persistent hypoglossal artery connecting basilar artery with basilar top aneurysm.

ENOVASCULAR MANAGEMENT:

Endovascular coiling was done in all 15 basilar top aneurysms. Endovascular occlusion of saccular aneurysm was done using coils only in 7 aneurysms with narrow neck (dome-neck ratio >2) and in one wide neck aneurysm (dome-neck ratio <2). Stent assisted coiling using Leo stent and Neuroform stent was used in 2 patients respectively (patient no. 2 & 3) with wide neck basilar top aneurysms with one or both PCA adjacent to neck of aneurysm. Balloon assisted coiling was done in 4 patients (patient no. 6, 8, 9, 11) using Sentry/Solstice/Equinox balloons in moderately wide neck basilar top aneurysms with one or both PCA adjacent to neck of aneurysm. In one patient (patient no. 14) with narrow neck saccular aneurysm, balloon assisted coiling was done using hyper form balloon to prevent coil prolapse into PCA. Parent artery occlusion was not done in any patient. In patient no. 1, basilar artery coiling was done through persistent hypoglossal artery due to hypoplastic proximal vertebral artery.

ANGIOGRAPHIC OUTCOME:

Immediate post procedure angiographic occlusion of aneurysm was obtained in 8 patients (53%) particularly in those aneurysms where coils only or balloon assisted coiling of aneurysms was undertaken. Immediate post procedure residual neck remnant was seen in 5 patients (33%) and residual aneurysm was seen filling in 2 patients (14%) with stent assisted coiling. Out of 7 incompletely coiled aneurysms, one patient (patient no. 3) had an episode of rebleed with diffuse cerebral edema 6 days later and died. Only one treatment session was done in all 15 patients.

FOLLOW UP OF COILED BASILAR TOP ANEURYSMS:

One patient (patient no. 3) had an episode of rebleed with diffuse cerebral edema 6 days later and died .Another patient (patient no. 8) died 2 days later after successful coiling due to myocardial infarction. Rest of 13 patients had a range of follow up period extending from 6months to 10 years. During follow up all the alive patients had Glasgow Outcome Score of 5 and Modified Rankin Score of 1 .No patient rebleed during the time of follow up period.

Details of 15 basilar top aneurysms with angiographic (DSA/TOFMRA/CEMRA) follow up are summarized in table 25.

TABLE 25;

PATIENT NO	IMMEDIATE ANGIOGRAPHIC RESULT	DURATION OF FOLLOW UP	FOLLOW UP ANGIOGRAPHIC RESULT	MODALITY USED IN FOLLOW UP	
1	Complete occlusion	3 years	Complete occlusion	CEMRA	
2	Residual aneurysm	3 years	Complete occlusion	CEMRA	
3	Residual neck+residual aneurysm	Died after 6 days due to rebleed	-----	-----	
4	Complete occlusion	4 ½ years	Residual neck(0.29 x0.22cm)	TOFMRA	
5	Complete occlusion	10 years	Residual neck(0.34 x0.5cm)	TOFMRA	
6	Complete occlusion	7 months	Complete occlusion	DSA	
7	Complete occlusion	9 months	Residual neck(0.32x0.38cm)	DSA	

8	Complete occlusion	Died after 2 days due to MI	-----	-----	
9	Residual neck(3x4.2mm)	7 ½ years	Residual neck(0.6x0.5cm)	TOFMRA	
10	Complete occlusion	7 months	Complete occlusion	DSA	
11	Residual neck	6 years	Residual neck(0.6x0.5cm)	TOFMRA	
12	Residual neck	5 years	Residual neck(0.4x0.3cm)	CEMRA	
13	Residual neck	6 months	Residual neck(0.3x0.4cm)	DSA	
14	Complete occlusion	2 years	Complete occlusion	CEMRA	
15	Residual neck	2 years	Residual neck(0.53x0.35cm)	TOFMRA	

POSTERIOR CEREBRAL ARTERY ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

Out of 33 patients, 6 patients had presented with posterior cerebral artery aneurysms. The study population with posterior cerebral artery aneurysms consisted of 4 males (66%) and 2 females (34%) with mean of 29 years and range of 7-53 years. Out of 6 aneurysms right PCA aneurysms was seen in one (patient no.25) and left PCA aneurysms were seen in 5 patients

(patient no. 21,22,23,24,26). Out of 6 patients, 2 patients (34%) had ruptured aneurysms and 4 patients (66%) had unruptured aneurysms, with CT Fischer score of 3 & 2 in ruptured aneurysms (patient no. 25,26 respectively) and Hunt Hess grade 2 at presentation in ruptured. The mean time between SAH and presentation in 2 patients with ruptured aneurysms was 10 days with a range of 6-14 days. Clinical presentation of patients with posterior cerebral artery aneurysms included sudden severe headache with vomiting & altered sensorium in 2 patients (patient no. 25,26), recurrent headache in 2 patients (patient no. 21,24), numbness and paresthesia due to left thalamic infarct in 1 patient (patient no. 22), and seizure in 1 patient (patient no. 23).

ANEURYSM CHARACTERISTICS AND MORPHOLOGY:

Out of 6 patients with posterior cerebral artery aneurysms, 4 (patient no 22, 24, 25, 26) aneurysms were fusiform in morphology, one (patient no. 23) saccular in morphology and 1 (patient no. 21) aneurysm was fusosaccular in morphology. Five aneurysms had no definable neck and 1 saccular aneurysm was narrow neck (patient no. 23). 3 (50%) out of 6 aneurysms were between 7-12 mm in maximum diameter and 2 (33%) aneurysms were 13-24 mm in maximum diameter and 1 (17%) aneurysm was 25 mm in maximum diameter. Vasospasm was seen in 1 patient (patient no. 25) with ruptured aneurysm. P2 segment of PCA was involved in 5 (83%) patients and P1 PCA was involved in 1 (17%) patient.

ENDOVASCULAR MANAGEMENT:

Endovascular coiling was done in all 6 posterior cerebral artery aneurysms. Endovascular occlusion of saccular aneurysm was done using coils only in 1 patient (patient no. 23) with narrow neck (dome-neck ratio > 2) preserving the left PCA and in remaining 5 patients parent artery occlusion along with occlusion of aneurysm was done as primary modality of treatment. Test occlusion before permanent PCA occlusion was not performed.

ANGIOGRAPHIC OUTCOME:

Immediate post procedure angiographic occlusion of aneurysm was obtained in 5 patients out of 6 patients (83%). One patient (patient no. 25) developed left hemiparesis and hemianopia post procedure due to right PCA territory infarct. Only one treatment session was done in all patients.

FOLLOW UP OF COILED POSTERIOR CEREBRAL ARTERY ANEURYSMS:

Out of 6 patients, 5 patients had a range of follow up period extending from 2 to 10 years. In one patient (patient no. 26) follow up was not done as follow up is scheduled after 3 months in October 2010. During follow up all the patients had Glasgow Outcome Score of 5 and Modified Rankin Score of 1. No patient rebled during the time of follow up period.

Details of 5 posterior cerebral artery aneurysms with angiographic (DSA/TOFMRA/CEMRA) follow up are summarized in table 26.

TABLE 26:

PATIENT NO	IMMEDIATE ANGIOGRAPHIC RESULT	DURATION OF FOLLOW UP	FOLLOW UP ANGIOGRAPHIC RESULT	MODALITY USED IN FOLLOW UP
1	Complete occlusion	10 years	Complete occlusion	DSA
2	Complete occlusion	5years	Complete occlusion	DSA
3	Complete occlusion	3 years	Complete occlusion	DSA
4	Residual aneurysm filling	2years	Complete occlusion	TOFMRA
5	Complete occlusion	7 ½ years	Complete occlusion	TOFMRA

BASILAR TRUNK ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

Out of 33 patients, 5 patients had presented with basilar trunk aneurysms. The study population with basilar trunk aneurysms consisted of 3 males (60%) and 2 females (40%) with mean of 32 years and range of 15-56 years. Out of 5 patients, two (patient no. 17 & 20) patients (40%) had ruptured aneurysms and 3 patients (60%) had unruptured aneurysms, with CT Fischer score of 2 in both ruptured aneurysms and Hunt Hess grade 2 (patient no. 17) and Hunt Hess grade 3 (patient no. 20) at presentation in ruptured aneurysms. The mean time between SAH and presentation in 2 patients with ruptured aneurysms was 21 days with a range of 14-28 days. Aneurysms in 4 patients (patient no. 16, 17, 18, 19) also exerted mass effect on brainstem structures. Clinical presentation of patients with basilar trunk aneurysms included sudden severe headache with vomiting & altered sensorium in 1 patient (patient no. 20) who developed left hemiparesis and diplopia secondary to vasospasm 5 days later, recurrent headache with vertigo in 2 patients (patient no. 16, 18), sudden severe headache & vomiting in 1 patient (patient no. 17) and headache with diplopia in one patient (patient no. 19).

ANEURYSM CHARACTERISTICS AND MORPHOLOGY

Out of 5 patients with basilar trunk aneurysms, 4 (patient no. 16, 17, 18, 19) aneurysms were fusosaccular in morphology, 1 (patient no. 20) saccular in morphology and saccular component of fusosaccular aneurysms were wide neck and 1 saccular aneurysm was also wide neck (patient no. 20). Three (60%) out of 5 aneurysms were 25mm in maximum diameter and 1 (20%) aneurysm was 13-24mm in maximum diameter and 1 (20%) aneurysm was 7-12 mm in maximum diameter. Vasospasm was seen in 1 patient with ruptured aneurysm (patient no. 20). Fusosaccular aneurysm was partially thrombosed in 3 patients (patient no. 16, 18, 19). Patient no. 17 had dissecting fusosaccular aneurysm of basilar trunk ending just proximal to SCA. In patient no. 16 & 19, the aneurysm involved the whole of basilar trunk sparing PCA. In one patient (patient no. 18), the fusosaccular aneurysm was involving the basilar trunk distal to AICA and sparing PCA. Saccular aneurysm with wide neck involving the basilar trunk between origin of SCA and PCA and projecting to right side was seen in one patient (patient no. 20).

ENDOVASCULAR MANAGEMENT

Endovascular coiling was done in all 5 basilar trunk aneurysms. Endovascular occlusion of saccular aneurysm was done using coils only in 1 patient (patient no. 20) with moderate wide neck (dome-neck ratio<2) with small residual neck persisting and in one patient (patient no.18), occlusion of saccular component of aneurysm and basilar artery at level of aneurysm was done after successful balloon occlusion test. In remaining 3 patients (patient no. 16, 17, 19) stent assisted coiling of aneurysm was done along with occlusion of left VA in patient no 19. Neuroform stent was used in 2 patients (patient no. 16&19) and Leo stent was used in one patient (patient no.17). In 2 patients (patient no. 16 &19), roadrunner 0.014inch microguidewire used to deploy the stent broke at its tip and got dislodged in right PCA, however no neurodeficits occurred as patients were on antiplatelet agents.

ANGIOGRAPHIC OUTCOME:

Immediate post procedure angiogram showed complete occlusion in 1 patient (patient no. 19) with residual aneurysm filling in 3 patients (patient no.16,17&18) and residual neck filling in one patient (patient no. 20) One patient (patient no.16) developed left hemiparesis and slurring of speech 6 days post procedure while on antiplatelets due to brainstem infarcts on right side. Only one treatment session was done in all patients except in one patient (patient no. 19) where second treatment session was done to occlude the residual aneurysmal sac, but was unsuccessful.

FOLLOW UP OF COILED BASILAR TRUNK ANEURYSMS :

TABLE 27:

PATIENT NO	IMMEDIATE ANGIOGRAPHIC RESULT	DURATION OF FOLLOW UP	FOLLOW UP ANGIOGRAPHIC RESULT	MODALITY USED IN FOLLOW UP
1	Residual aneurysm filling	2 years	Residual aneurysmal filling(1.2 x0.9cm)	CEMRA

2	Residual aneurysmal filling	7months	Complete occlusion	TOFMRA
3	Residual aneurysm filling distally	10 years	Residual aneurysmal filling(8.8x7.2mm)	DSA
4	Complete occlusion	2 1/2years	Residual aneurysmal filling(3 x1cm)	DSA
5	Residual neck	10months	Residual neck of 0.34 x0.46mm filling	CEMRA

INTRACRANIAL VERTEBRAL ARTERY ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

Out of 33 patients, 5 patients had presented with intracranial vertebral artery aneurysms. The study population with vertebral artery aneurysms consisted of 2 males (40%) and 3 females (60%) with mean of 45 years and range of 25-56 years. Out of 5 patients, 3 patients (patient no.27, 28&29) had ruptured aneurysms and one patient (patient no.30) had unruptured aneurysm presenting with mass effect, with CT Fischer score of 2 in all 3 ruptured aneurysms and Hunt Hess grade 2 at presentation in ruptured aneurysms. The mean time between SAH and presentation in 3 patients with ruptured aneurysms was 24 days with a range of 12-30 days. Clinical presentation of patients with vertebral artery aneurysms included sudden severe headache with vomiting&altered sensorium in 3 patients ((patient no.27, 28 and 29) and recurrent headache with episodes of vomiting in two patients (patient no. 30, 31).

ANEURYSM CHARACTERISTICS AND MORPHOLOGY

4 patients with intracranial vertebral artery aneurysms had fusiform aneurysms with no definable neck with one patient(patient no. 31) having left V4 wide neck saccular aneurysm

Three (60%) out of 5 aneurysms were involving the V4 segment of right vertebral artery and 2 aneurysms (patient no. 30, 31) were involving the V4 segment of left VA with partial thrombus in its wall in 1 patient (patient no. 30). Four aneurysms were in range of 13-24mm in maximum diameter and one saccular aneurysm in range of 7-12mm in maximum diameter. RtVA aneurysms were distal to right PICA origin and one left VA aneurysm was proximal to PICA origin and other distal to PICA origin. Both VA were dominant in all patients.

ENDOVASCULAR MANAGEMENT

Endovascular coiling was done in all 5 intracranial vertebral artery aneurysms. Endovascular occlusion of wide necked saccular aneurysm was done using stent (Neuroform stent) assisted coiling in 1 patient (patient no. 31) preserving the left VA and in remaining 4 patients parent artery occlusion along with occlusion of aneurysm was done as primary modality of treatment.

ANGIOGRAPHIC OUTCOME

Immediate post procedure angiographic occlusion of aneurysm was obtained in 3 patients (patient no. 27, 28, 29) and minimal residual filling of aneurysm was seen in 2 patients (patient no. 30, 31). Only one treatment session was done in all patients.

FOLLOW UP OF COILED INTRACRANIAL VERTEBRAL ARTERY ANEURYSMS

TABLE 28:

PATIENT NO	IMMEDIATE ANGIOGRAPHIC RESULT	DURATION OF FOLLOW UP	FOLLOW UP ANGIOGRAPHIC RESULT	MODALITY USED IN FOLLOW UP
1	Complete occlusion	4 months	Complete occlusion	TOFMRA
2	Complete occlusion	5 years	Complete occlusion	DSA/TOFMRA
3	Residual aneurysmal filling	1 year	Complete occlusion	TOF MRA

1 patient (patient no. 29) died 3 days later due to cardiac arrest and in another patient (patient no 31) follow up angiography is planned after 3 months in October 2010.

PICA ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION

One male patient 52 years old (patient no. 33) had presented with aneurysm at origin of left PICA in association with left cerebellar hemispheric AVM and aneurysm was unruptured aneurysm. Clinical presentation of patients included left hemi facial spasm.

ANEURYSM CHARACTERISTICS AND MORPHOLOGY

The aneurysm was flow related saccular aneurysm with narrow neck involving left PICA origin along with left cerebellar AVM and aneurysm was less than 7mm in maximum diameter

ENDOVASCULAR MANAGEMENT

As it was decided to coil the aneurysm first followed by AVM embolisation, coils were used to selectively pack the aneurysmal sac excluding the aneurysm from circulation

ANGIOGRAPHIC OUTCOME:

Complete angiographic occlusion of aneurysm was obtained which was persistent on DSA done 1 year later. After exclusion of aneurysm, the cerebellar AVM was partially embolized with glue.

VERTEBROBASILAR JUNCTION ANEURYSM WITH FENESTRATION:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

One female patient 70 years old (patient no 32) had presented with saccular aneurysm involving vertebrobasilar junction with fenestration. Clinical presentation was with SAH with CT Fischer score of 3 and Hunt Hess score of 2.

ANEURYSM CHARACTERISTICS AND MORPHOLOGY

The aneurysm was narrow neck involving vertebrobasilar junction associated with fenestration and maximum diameter was <7mm.

ENDOVASCULAR MANAGEMENT

Endovascular management of aneurysm was done using coils only with minimal residual aneurysm filling immediate post procedure.

FOLLOW UP:

At follow up of 1year the patient was clinically asymptomatic with Glasgow Outcome Score of 5 and modified Rankin Score of 1. However no imaging was done.

DISCUSSION:

The present study has been done to analyze the clinical manifestations at presentation and at follow up of endovascular treatment of posterior circulation aneurysms and to analyze the results, outcome and rebleed in patients with various posterior circulation aneurysms who underwent endovascular management as primary treatment option. Most of the data of the present study was collected retrospectively.

DEMOGRAPHIC PROFILE:

During a period of 12 years, a total of 33 patients presenting with various clinical manifestations and clinical/imaging features suggestive of posterior circulation aneurysms underwent endovascular management of aneurysms. Majority(60%) of patients were in age group of 40-60 years and mean age was 43 years with range of 7-70 years. Males constituted 60 % (20 patients) and females 40 % (13 patients). Range of age group, sex, mean age (range) and study duration by various authors is shown below:

TABLE 29:

Authors	Duration of study	No of patients	Mean age	range	sex
Mordasini,P et al ¹³¹	1993-2003	46	54 years	15-78years	27 males 19 females
Kyung-Soo Sung et al ²⁷	2004-2008	13	51.7years	15-66years	7males 6females

Hilman J et al132	1993(1 year)	49	55 years	19-75 years	20 males 29 females
Present study	1998-2010	33	43 years	7-70 years	20 males 13 females

CLINICAL PRESENTATION:

Out of 33 patients, 14 patients had not bled. The remaining 19 patients had ruptured aneurysms with Hunt Hess Grade 2 in 11 patients (33%), 7 patients were in Hunt Hess Grade 3 (22%) and 1 patient in Hunt Hess Grade 1 at presentation. CT Fischer score was 2 in 13 patients (39%) and 3 in 6 patients presenting with subarachnoid haemorrhage. Twenty patients (59%) had presented with sudden severe headache, vomiting and altered sensorium. Mordasini P et al (227) reported that in their series of 46 patients with posterior circulation aneurysms that 11 patients were in Hunt Hess Grade 2 (23.9%) and 10 patients were in Hunt Hess Grade 3(21.7%) which was similar to our study with majority of patients in Hunt Hess Grade 2. They also reported that CT Fisher score in patients with ruptured aneurysms were score 2 in 1 patient(2.2%), 3 in 14 patients(30.4%) and 4 in 16 patients(34.8%) which was different from our study where in majority (40%) of patients had CT Fisher score of 2. Mass effect was seen in 5(16%) out of 14 unruptured aneurysms while Mordasini P et al(227) reported 4 patients presenting with mass effect out of 10 unruptured aneurysms.

After onset of bleeding majority (9 patients) underwent endovascular management between 8-20 days. Douglas AN et al (228) in their study reported that eight patients were treated 0 to 3 days after subarachnoid hemorrhage (SAH), 17 patients were treated 4 to 11 days after SAH, and one patient was treated 30 days after SAH in 26 posterior circulation aneurysms. This was similar to our study where in majority underwent endovascular coiling within 8-20 days following an episode of bleed.

ANEURYSM LOCATION AND MORPHOLOGY OF POSTERIOR CIRCULATION ANEURYSMS:

In our study there were 15 basilar top aneurysms, 6 PCA aneurysms, 5 basilar trunk, 5 intracranial vertebral artery aneurysms, 1 vertebrobasilar junction aneurysm and 1 PICA aneurysm. Douglas AN et al (228) in their study of 26 posterior circulation aneurysms found that nineteen aneurysms were located at the basilar top, two at the P1 segment of the posterior cerebral artery, two at the origin of the superior cerebellar artery, one at the midbasilar trunk, three at the vertebrobasilar junction, and one at the origin of the posterior inferior cerebellar artery (PICA) which was more or less similar in location as compared to our study. Mordasini P et al (227) in their study also found that among 46 posterior circulation aneurysms, basilar top aneurysms were 34 (72.3%), basilar trunk aneurysm was 1 (2.1%), posterior inferior cerebellar artery aneurysms were 5 (10.6%), vertebral artery aneurysms were 3 (6.4%) with majority of aneurysms being basilar top aneurysms similar to our study.

Maximum aneurysmal diameter as analyzed on DSA in our study was divided into 4 groups according to ISUIA trial and <7mm aneurysms were 6(18%), 7-12mm aneurysms were 15(46%), 13-24mm were 8(24%) and giant aneurysms were 4 (>25mm maximum diameter) with 5 of 33 aneurysms showing partial thrombosis. Douglas AN et al (228) in their study of 26 posterior circulation aneurysms found that 12 aneurysms were 3 to 6 mm in maximum diameter, 12 aneurysms were 7 to 10 mm, and four aneurysms were 11 to 25 mm with 2 of the 28 aneurysms were partially thrombosed which was similar to our study with majority of posterior circulation aneurysms being 7-12mm in maximum diameter.

Neck size was estimated according to the projection showing the widest neck on DSA and was classified as <4 mm or >4 mm with dome neck ratio >2 for narrow neck and dome neck ratio <2 for wide neck aneurysms. Among 33 posterior circulation aneurysms 11(33%) were narrow neck and 22 were wide neck (67%) in our study. Mordasini P et al (227) in their study of 46 posterior circulation aneurysms found that twenty-nine (61.7%) aneurysms had small necks (<4 mm), whereas 18 (38.3%) had large necks (>4 mm) which was different from our study.

Out of 33 aneurysms in our study, 20 aneurysms (60%) were saccular in morphology, 8 were fusiform (25%) and 5 were fusosaccular in morphology (15%) in our study. Out of these, 13 saccular aneurysms (39%), 1 fusosaccular (3%), 5 fusiform (15%) aneurysms were ruptured at

presentation. Todd E. Lempert et al (229) in their study of 112 patients with ruptured posterior circulation aneurysms found 40 (37%) saccular aneurysms having a narrow neck (<4 mm), 14 (13%) were saccular harboring a wide neck (>4 mm), 25 (23%) were fusiform, 19 (18%) were irregularly shaped or multilobed, and 8 (7%) were giant aneurysms. In their study also majority of ruptured aneurysms were saccular similar to our study although the study population was larger. Majority of ruptured saccular aneurysms (8 out of 13) were narrow neck similar to study by Todd E. Lempert et al (229) wherein 40 out of 54 saccular aneurysms were having narrow neck and were ruptured.

BASILAR TOP ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

In our study, 15 patients had presented with basilar top aneurysms, consisting of 10 males (66%) and 5 females (34%) with mean of 49 years and range of 35-66 years. Tateshima S et al (213) in their study of 73 basilar top aneurysms reported that the average age of the patient population in their study was 48.3 years (range, 28–82 yr) with fifty-one patients women, and 22 men and majority of patients in age group of 40-61 years. In our study also the majority of patients (12 out of 15) were in age group of 40-60 years.

Out of 15 patients, 11 patients (73%) had ruptured aneurysms and 4 patients (27%) had unruptured aneurysms, with CT Fischer score of 2 in majority (7 patients) and majority of patients (7 patients) were in Hunt Hess grade 2 at presentation. The mean time between SAH and presentation in 11 patients with ruptured aneurysms was 30 days with a range of 3-150 days. Peluso JPP et al (214) in their series of 114 patients noted that a total of 114 (74%) had ruptured and 40 (26%) were unruptured & 68 patients were in Hunt Hess grade 2 & timing of treatment after subarachnoid hemorrhage (SAH) was 0–3 days in 43 patients, between 4 and 14 days in 53 patients, and >14 days in 18 patients. Although their study population was large compared to ours, in our study also majority were ruptured basilar top aneurysms and patients with ruptured aneurysms were in Hunt Hess grade 2 at presentation.

ANEURYSM CHARACTERISTICS AND MORPHOLOGY:

Out of 15 patients with basilar top aneurysms in our study, all were saccular in morphology and 8 basilar top aneurysms were narrow neck with dome-neck ratio of >2 . 10 (66%) out of 15 aneurysms were between 7-12 mm in maximum diameter and 4 (27%) aneurysms were <7 mm in maximum diameter and 1(6%) aneurysm was 13-24mm in maximum diameter. Cameron G et al (230) in their study of 33 basilar top aneurysms reported that aneurysms in their study ranged in angiographic size from 3 to 25 mm with 16 small (< 10 mm) and 17 large aneurysms (>10 mm)& thirteen of the aneurysms had narrow necks (<4 mm) and 20 had wide necks. There were more narrow neck aneurysms which were 7-12mm in our study contrary to the previous study .Total of 7 aneurysms (46%) in our study were coiled with help of supporting device .Of 154 aneurysms, Peluso JPP et al (214) in their series reported that 40 (26%) were primarily coiled with a supporting device with twenty-six aneurysms primarily coiled with a temporary supporting balloon.

INITIAL ANGIOGRAPHIC OCCLUSION:

In our study immediate post procedure angiographic occlusion of basilar top aneurysm was obtained in 8 patients (53%) particularly in those aneurysms where coils only or balloon assisted coiling of aneurysms was undertaken. Immediate post procedure residual neck remnant was seen in 5 patients (33%) and residual aneurysm was seen filling in 2 patients (14%) with stent assisted coiling. Peluso JPP et al (214) reported that in their series initial angiographic occlusion of 154 basilar tip aneurysms was complete or near-complete in 144 (94%) and incomplete in 10 (6%) aneurysms. Tateshima S et al (213) in their study of 73 basilar top aneurysms reported that complete aneurysm occlusion was achieved in 34 aneurysms (45.3%), a small neck remnant was observed in 30 aneurysms (40%)&incomplete occlusion was detected in 7 aneurysms (9.3%). This was similar to our study where majority 8(53%)of aneurysms were completely occluded.

IMMEDIATE CLINICAL OUTCOME:

Out of 15 basilar top aneurysms, 13 patients were neurologically intact or remained unchanged and 2 patients died with one patient death 6 days later due to rebleed in partially coiled aneurysm and other patient died 2 days after procedure due to myocardial infarction. Tateshima S et al (213) in their study of 69 patients treated with the GDC system reported that 63 patients

(91.5%) were neurologically intact or remained unchanged, four patients (5.7%) experienced clinical deterioration, and two patients (2.8%) died.

LONG TERM ANGIOGRAPHIC (DSA/CEMRA/TOFMRA) OUTCOME:

In our study follow up period ranged from 6m-10 years with a mean of 3.8 years The follow-up period ranged from 3 to 40 months (average, 18.6 mo) in their study of 69 patients by Tateshima S et al(213).We found that in our study out of 8 initial completely occluded basilar top aneurysms ,4(50%) were completely occluded on follow up and 3(38%) were showing residual neck on follow up due to coil compaction.1 aneurysm with residual neck was completely occluded on follow up with remaining 5 aneurysms showing persisting residual neck .Tateshima S et al (213) in their study of 73 basilar top aneurysms reported that thirty aneurysms (71.4%) demonstrated complete or near-complete occlusion with further thrombosis observed in one small aneurysm with a small neck and in one large aneurysm with aneurysm recanalization attributable to coil compaction occurring in 10 aneurysms (23.8%).The percentage of completely occluded aneurysms was more than in our study although their study population was large.

LONG-TERM CLINICAL OUTCOME:

Long-term clinical outcomes were obtained in 13 patients in our study &the mean follow-up period was 3.8 years. Two patients who died during the acute phase were excluded from the analysis of long-term clinical outcome. The outcome was classified according to the Glasgow outcome scale and modified Rankin score and during follow up all the alive patients had Glasgow outcome score of 5 and modified Rankin score of 1 .No patient rebled during the time of follow up period after discharge. Tateshima S et al (213) in their study of 73 basilar top aneurysms reported that long-term clinical outcomes were obtained in 71 patients with mean follow-up period was 31.3 months with fifty patients (70.4%) having a good recovery, 11 (15.5%) moderately disabled, 3 (4.2%) severely disabled, and 1 patient (1.4%) remaining in a vegetative state with death of six patients (8.5%).The clinical outcome in our study was different from the previous study with all alive patients having a good recovery.

Samson et al. (231) reported their surgical results in 302 patients with ruptured and unruptured basilar top aneurysms. They reported a technical complication rate of 14% and a procedure-related combined morbidity and mortality rate of 7%. In our series, technical complications were

observed in none of cases, and the morbidity and mortality related to the procedure were 0 %and 6% respectively.

The procedure-related morbidity and mortality observed in our series indicate that treatment of basilar top aneurysms with GDCs can be performed safely in most patients owing to relatively easy endovascular access to the aneurysm. The high and deep location of the tip of the basilar artery does not influence the endovascular treatment .There is no need for brain and cranial nerve retraction, and the possibility of untoward occlusion of a perforating artery is small. These arteries usually are incorporated at the level of the neck of the aneurysm& this anatomic disposition allows tight GDC packing of the basilar top aneurysm without compromising the lumen of a perforator. There were no complications related to a perforator injury in our series of 15 basilar top aneurysms,

In our series, no rebleeding occurred in the acute phase of the SAH except in 1 patient, despite an incomplete embolisation.In our series, approx 40% complete occlusion rate was observed at follow up, and no recanalization occurred in 4 out of 8 completely occluded aneurysms in the long-term angiographic follow-up studies. Incomplete occlusion with residual neck remnant or residual filling of aneurysm immediate post procedure was associated with recanalization in our study in 38%of cases.

Newly designed coils and the use of the balloon-assisted technology popularized by have also exerted a positive influence on the recent anatomic results. In series of Moret et al (232), balloon-assisted technique was used in two patients, and 3-D GDCs were used in one patient achieving complete occlusion. In our study, stent assisted coiling was done in 2 patients and balloon assisted coiling in 5 patients with complete occlusion at follow up in one stent assisted and two balloon assisted cases and one case each of balloon and stent assisted cases died post procedure. Due to presence of residual neck, careful follow-up is mandatory for the patients with an incompletely coiled basilar top aneurysms

B) POSTERIOR CEREBRAL ARTERY ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION:

In our study there were 6 (18%) patients with PCA aneurysms with males constituting 4(66%) and females 2(34%) with mean age of 29 years(range of 7-53years) with majority in age group of 30-50 years. In a study by Hong Gee Roh et al (233), PCA aneurysms were seen in 13 patients (eight females and five males) with 17 PCA aneurysms with age of the patients ranging from 20 to 67 years, with a mean age of 44 years. The patients in our study were younger than the previous study with a mean age of 29 years although the study population was smaller in our study. In a study 10 patients with PCA aneurysms by Paul Hallacq et al (234), 10 patients had PCA aneurysms involving P2 segment with five patients being female and five male, with an age range of 18–70 years and mean age of 43 years..

Out of 6 aneurysms in our study, 2 were ruptured and 4 were unruptured with CT Fischer score of 2&3 in ruptured and Hunt Hess score of 2 in ruptured aneurysms. In a study 10 patients with PCA aneurysms by Paul Hallacq et al(234) , 2 patients had presented with SAH due to ruptured aneurysms with grade of 0 in eight patients and a grade of I in two patients which was almost similar to our study.

ANEURYSM CHARACTERISTICS AND MORPHOLOGY:

There were 4 fusiform, 1 fusosaccular and 1 saccular aneurysm in our study with narrow neck of saccular aneurysm and no definable neck in rest of aneurysms with size of aneurysms being 7-12mm in 3 patients(50%), 13-24 mm in 2 patients(33%) and 25mm in 1 patient(17%) and right PCA was involved in 1 patient and left PCA in 5 patients with P1 segment involved in 1 patient with saccular aneurysm and P2 PCA in rest of 5 patients. In a study by Hong Gee Roh et al(233) , out of 17 PCA aneurysms nine were fusiform and eight were saccular and P2 segment was involved in 11 patients (65%), the P1 segment in three patients (18%), the P3 segment in one patient (6%), and the P4 segment in one patient(6%); with maximum diameter of aneurysms in range of 7-12mm. These results were similar to our study where maximum diameter was in range of 7-12mm and majority involved P2 segment PCA.

INITIAL ANGIOGRAPHIC OCCLUSION:

In our study ,one saccular aneurysm was treated with coils only preserving the parent artery and in rest 5 patients with P2 segment PCA fusiform/fusosaccular aneurysms ,proximal aneurysm as well as parent artery was occluded and immediate post procedure angiogram showed occlusion of aneurysm in 4 patients and faint filling in one patient. Hong Gee Roh et al (233) in their study of 17 PCA aneurysms in 13 patients treated by endovascular approach reported that 7 patients were treated by occlusion of the aneurysm and parent artery together; five patients by selective embolization of the aneurysm with complete occlusion of aneurysm which was similar to our study in which saccular aneurysm was coiled completely with preservation of parent artery and fusiform /fusosaccular aneurysm was completely occluded except in 1 patient following proximal of aneurysm and parent artery

IMMEDIATE CLINICAL OUTCOME:

Out of posterior cerebral artery aneurysms in our study, 5 patients were neurologically intact or remained unchanged and 1 patients developed left hemi paresis and hemianopia due to right PCA infarct immediate post procedure. Hong Gee Roh et al (233)in their study of 17 PCA aneurysms in 13 patients treated by endovascular approach reported that infarctions were found in two patients treated with selective embolization and in three patients treated with parent artery occlusion& one patient with a ruptured P2 aneurysm treated with parent artery occlusion developed transient amnesia as an ischemic symptom. The complication rate in our study was less compared to previous study although the number of PCA aneurysms was less.

LONG TERM ANGIOGRAPHIC (DSA/CEMRA/TOFMRA)OUTCOME:

In our study follow up period ranged from 2-10 years with a mean of 4.5 years The follow-up period ranged from mean time of 27 months; range 5– 72 months in the study by Hong Gee Roh et al (233) of 17 PCA aneurysms in 13 patients. We found that in our study, all coiled PCA aneurysms (100%) were completely occluded on follow up. In the study by Elisa F. Ciceri et al (218), out of 20 patients treated by endovascular approach angiographic follow-up was available in 11 of the 14 aneurysms that were obliterated with GDC and where the parent artery was preserved and they reported that there was no recanalization of the treated aneurysm on any of the follow-up studies and of the seven patients who were treated with parent artery occlusion, two underwent follow- up MR imaging and MRA studies showing thrombosis and

shrinkage of the aneurysm. The percentage of completely occluded aneurysms was more in our study although their study population was large.

LONG-TERM CLINICAL OUTCOME:

Long-term clinical outcomes were obtained in 5 patients in our study & the mean follow-up period was 4.5 years. The outcome was classified according to the Glasgow outcome scale and modified Rankin score and during follow up all the alive patients had Glasgow outcome score of 5 and modified Rankin score of 1. No patient rebled during the time of follow up period Hong Gee Roh et al (233) in their study of 17 PCA aneurysms in 13 patients noted that there was no delayed deficit or rebleeding during the clinical follow-up period (mean time 27 months; range 5–72 months) and the clinical outcomes available at the follow up time were therefore excellent (GOS 5, modified Rankin scale 0 or 1) for all patients. These results were similar to our study.

Aneurysms arising from the PCA have a predilection for the P1 and P2 segments as described by Ferrante L et al (235). This is the case in our series as well as in other previously reported series by Gerber C J et al (236) and Pia H W et al (237). These aneurysms have some peculiar morphologic features and present with specific clinical findings that distinguish them from aneurysms occurring at other anatomic locations of the intracranial circulation. They frequently affect young patients, with an average age of 38 years as described by Ferrante L et al (235). This is younger than the average age of 50 to 60 years for aneurysms occurring at other anatomic sites. Also, there is a higher incidence of large and giant aneurysms (almost 23% of PCA aneurysms, versus 3–5% at other anatomic sites) affecting the PCA as described by Gerber C J et al (236). In our series, however there was only one aneurysm with maximum diameter of 25mm among all PCA aneurysms which could be due to less number of PCA aneurysms in our study. The most common clinical presentation of PCA aneurysms described in the literature is the SAH (80%) as reported by Pia H W et al (237). In our series, 2 patients of PCA aneurysms presented with SAH, In our patients in whom the treatment of the PCA aneurysm required permanent artery occlusion, there was a relatively low incidence of visual field defect noticed in only 1 out of 6 patients. The low incidence of visual field defect complicating parent artery occlusion is related to the rich anastomotic collaterals that exist between the territory of the PCA and that of other arteries as reported by Elisa F. Ciceri et al (218). Hamada et al (238) reported a 15-year experience with 21 cases of PCA aneurysms. These investigators performed open surgery in 16 patients, and 11 (69%) of these showed a good recovery. In our series with

endovascular treatment option, good recovery (Glasgow Outcome Score of 5) was obtained in 5 out of 6 patients (83%) patients at discharge.

C) BASILAR TRUNK ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION

In our study , 5 out of 33 patients had presented with basilar trunk aneurysms consisting of 3males (60%) and 2 females (40%) with mean of 32 years and range of 15-56 years.Out of 5 patients, 2 (40%) had ruptured aneurysms and 3 patients(60%) had unruptured aneurysms ,with CT Fischer score of 2 in both ruptured aneurysms and Hunt Hess grade 2 &grade 3 at presentation in ruptured aneurysms .Aneurysms in 4 patients also exerted mass effect on brainstem structures. Uda K et al (199) in their study of basilar artery trunk aneurysms in 39 patients reported that 29 were women and 10 were men, with ages ranging from 28 to 76 years (mean 51 years) and 27 patients had presented with SAH and six suffered from intracranial mass effect& sixteen patients presenting with SAH were classified according to the Hunt and Hess scale with three patients in Grade I, four in Grade II, eight in Grade III, and one in Grade IV. Compared to our study where patients with ruptured aneurysms were in Hunt Hess grade 2 and 3 at presentation there were majority of patients in Hunt Hess grade 3 in their study.

ANEURYSM CHARACTERISTICS AND MORPHOLOGY

In our study, out of 5 patients with basilar trunk aneurysms ,4 (80%)aneurysms were fusosaccular in morphology with partial thrombosis in 3 patients,1(20%)was saccular in morphology with wide neck. Three (60%)out of 5 aneurysms were 25mm in maximum diameter and 1(20%) aneurysm was 13-24mm in maximum diameter and 1(20%) aneurysm was 7-12 mm in maximum diameter.Uda K et al (199) in their study of basilar artery trunk aneurysms reported that out of 41 aneurysms twenty-eight lesions were small (diameter <11 mm), seven were large (11–25 mm), and six were giant (>25 mm) with fifteen aneurysms having small necks , 22 having wide necks , and four were fusiform& in three patients giant aneurysms were partially thrombosed.This highlights the fact that majority of basilar trunk aneurysms are wide neck or fusiform in morphology as evident in our study.

INITIAL ANGIOGRAPHIC OCCLUSION

Immediate post procedure angiogram showed complete occlusion in 1 patient with residual aneurysm filling in 3 patients and residual neck filling in one patient. Stent assisted coiling was done in 3 patients and coils only were used to occlude the saccular aneurysm in one patient and coils only were used to occlude the aneurysm and basilar artery in another patient with fusosaccular aneurysm in our study. Uda K et al (199) in their study of basilar artery trunk aneurysms reported that thirty-six aneurysms were selectively occluded using GDCs, and in five patients concomitant occlusions of the aneurysm and parent artery were performed with thirteen aneurysms (31.7%) completely occluded, 22 (53.7%) showed neck remnant, and six (14.6%) were incompletely occluded and complete occlusion was achieved in one (14.3%) of seven large aneurysms, whereas in none of the giant aneurysms was complete occlusion achieved. This was similar to our study where incomplete occlusion was achieved in large fusiform /fusosaccular aneurysms.

IMMEDIATE CLINICAL OUTCOME: One patient developed left hemiparesis and slurring of speech 6 days post procedure while on antiplatelets due to brainstem infarcts on right side in our study. Uda K et al (199) in their study of basilar trunk aneurysms reported that technical complications observed in their study were aneurysm perforation (2.6%), ischemic complications (7.7%), including two thromboembolic and one hemodynamic events while post procedure complications were less in our study although the study population was smaller

LONG TERM ANGIOGRAPHIC (DSA/CEMRA/TOFMRA)OUTCOME)

In our study mean follow up period ranged from 7 months -10 years with a mean of 3.2 years. In our study, in one patient who underwent stent assisted coiling for fusosaccular aneurysm showed complete occlusion of aneurysm on follow up. In rest of 4 cases residual filling of aneurysm was seen which was however less in diameter compared to that prior to treatment. Uda K et al (199) in their study of basilar artery trunk aneurysms reported that recanalization caused by coil compaction into the body of the aneurysm was seen in 9 patients with fusiform aneurysms at follow up. These results are similar to our study wherein fusiform/fusosaccular aneurysms have a high chance of coil compaction and subsequent recanalization.

LONG-TERM CLINICAL OUTCOME:

Long-term clinical outcomes were obtained in 5 patients in our study & the mean follow-up period was 3.2 years, the outcome was classified according to the Glasgow outcome scale and modified Rankin score and during follow up all patients had Glasgow outcome score of 4- 5 and Modified Rankin Score of 1 except in 1 patient where it was 3 .No patient rebled during the time of follow up period. Uda K et al (199) also reports in their study of basilar artery trunk aneurysms that no aneurysm rebleeding occurred during the perioperative period; however, a large BA trunk aneurysm reruptured 8 years after the initial treatment. Ten of 11 patients treated in the nonacute phase after SAH had excellent outcomes and one died.

Follow-up studies have reported a poor prognosis especially in patients harboring fusiform BA trunk aneurysms. In 1989, Echiverri et al (239) published a retrospective evaluation of the clinical features and therapeutic outcome in 13 consecutive patients with the diagnosis of fusiform aneurysm of the vertebrobasilar system. Brain stem compression (31%) and ischemic symptoms (77%) were the most common complications in this series. In three out of five patients that died, the cause of death was progressive brain stem ischemia. However no patient died on follow up in our study & were mostly asymptomatic In the series of Steinberg et al(240), surgical occlusion of the BA in the lower portion yielded a good clinical result in 83% while an occlusion of the upper portion led a good result in only 64%. In one of our patient, saccular component of aneurysm occlusion was done along with basilar artery occlusion proximally which was uneventful. In one patient in our study ,stent assisted coiling of proximal basilar fusosaccular aneurysm was done along with stenting of right VA and occluding of left VA.This was similar to technique reported by Thomas L et al (241) in cases of fusosaccular vertebrobasilar aneurysms.Stent assisted coiling was done in 3 patients in our study which was first described by Higashida et al (242) & Thomas L et al (241) reported that stent-assisted coil occlusion or stenting as a sole treatment option is a valuable alternative to surgery and to endovascular parent vessel occlusion .

D) INTRACRANIAL VERTEBRAL ARTERY ANEURYSMS:

PATIENT DEMOGRAPHICS AND CLINICAL PRESENTATION

Vertebral artery aneurysms were mostly dissecting in nature. The study population with vertebral artery dissecting aneurysms consisted of 5 patients with 2 males (40%) and 3 females (60%) with mean age of 45 years and range of 25-56 years. Out of 5 patients, 3 patients had ruptured aneurysms and 1 patient had unruptured aneurysm presenting with mass effect and another 1 had chronic headache, with CT Fischer score of 2 in all 3 ruptured aneurysms and Hunt Hess grade 2 at presentation in ruptured aneurysms. In their series of 25 patients with ruptured vertebrobasilar dissecting aneurysms by Lee J M et al (243) fourteen patients were male and 11 were female the mean patient age was 45.3 years (range, 22–66 years) with Hunt and Hess grade of 2 in eight patients (32%) had grade 2 and grade 3 in 8 patients. This shows the younger age group of dissecting vertebral artery aneurysms which was also evident in our study.

ANEURYSM CHARACTERISTICS AND MORPHOLOGY

In our study, 4 patients with intracranial vertebral artery aneurysms had fusiform aneurysms with one patient having left V4 wide neck saccular aneurysm & 3 (60%) out of 5 aneurysms were involving the V4 segment of right vertebral artery and 2 aneurysms were involving the V4 segment of left VA with 4 aneurysms in range of 13-24mm in maximum diameter and one saccular aneurysm in range of 7-12mm in maximum diameter with right VA aneurysms located distal to right PICA origin and one left VA aneurysm proximal to PICA origin and other distal to PICA origin. Lee J M et al (243) reported that of the 25 patients with ruptured vertebrobasilar dissecting aneurysms, 16 aneurysms were classified as supra-PICA lesions, 7 as infra-PICA lesions, and 3 as involving the PICA.

INITIAL CLINICAL & ANGIOGRAPHIC OCCLUSION:

Endovascular occlusion of wide necked saccular aneurysm was done using stent (Neuroform stent) assisted coiling in 1 patient preserving the left VA and in remaining 4 patients parent artery occlusion along with occlusion of aneurysm was done as primary modality of treatment with complete occlusion in 3 patients and residual aneurysm filling in 2 patients in our study. Lee J M et al (243) reported that of the 25 patients with ruptured vertebrobasilar dissecting

aneurysms complete occlusion of dissected arterial and aneurysm segments (internal trapping) was achieved in 21 (91.3%) of 23 patients with supra PICA and infra PICA locations. The two patients with posterior inferior cerebellar artery involvement underwent double stent only placement with partial occlusion. All aneurysms in our series were either distal or proximal to PICA and hence parent artery occlusion was done as primary treatment in 4 patients. Clinical outcome was good in all patients except in 1 patient died 3 days later due to cardiac arrest.

LONG TERM ANGIOGRAPHIC AND CLINICAL OUTCOME:

Long-term clinical outcomes were obtained in 3 patients in our study & the mean follow-up period was 2.1 years with range of 4 months to 5 years with complete occlusion of aneurysm in all 3 patients & the clinical outcome was classified according to the Glasgow outcome scale and modified Rankin score and during follow up & all patients had Glasgow outcome score of 5. and modified Rankin Score of 1. Clinical outcomes were favorable in 17 (68%) of 25 patients, 2 (8%) had severe disability, and 6 (24%) patients died in series of ruptured vertebrobasilar dissecting aneurysms by Lee J M et al (243).

When the dissection site is located in the vertebral artery proximal to the PICA division, proximal occlusion may be effective and safe. However, when the dissecting site is located in the vertebral artery distal to the PICA endovascular internal trapping may be more reliable to prevent rerupture; however, it may precipitate the lateral medullary syndrome or brain stem infarctions. Theoretically, for the preservation of perforators, treatment with a combination of stents and coils or with a stent alone, which can preserve the parent artery, may be an alternative to parent artery occlusion, especially for patients with high risks of complications after parent artery occlusion as described by Lee J M et al (243). However, none of our patients developed brainstem infarct/lateral medullary syndrome post trapping of involved vertebral artery

E) VERTEBROBASILAR JUNCTION ANEURYSM WITH FENESTRATION:

Out of 33 posterior circulation aneurysms in our study, one aneurysm involved vertebrobasilar junction associated with fenestration & age of patient was 70 years with ruptured aneurysm with CT Fischer score of 3 and Hunt Hess score of 2. Peluso J.P.P et al (171) reported that 10

aneurysms in 10 patients were located on the vertebrobasilar junction with a mean age of 49.6 years (range, 29–75 years & 5 were males and 5 females & out of 10 aneurysms, 9 (90%) had ruptured and one (10%) presented with symptoms of mass effect & clinical conditions at the time of treatment were Hunt and Hess (HH) I-II in 3, HH III in 3, and HH IV-V in 3 & seven of 10 aneurysms (70%) were associated with proximal basilar fenestration.

Aneurysm was narrow necked saccular aneurysm which was treated with coil occlusion only with minimal residual neck immediate post procedure. Immediate post procedure clinical outcome was good with no neurodeficits and at 1 year follow up the patient had Glasgow outcome score of 5 and modified Rankin Score of 1. Peluso J.P.P et al (171) reported that initial aneurysm occlusion was complete in 7 and near complete in 2 aneurysms & 6 patients had angiographic follow-up at a mean of 17.5 months (range, 6–30 months) & aneurysm occlusion at follow-up was complete in 4 and near-complete in 2 aneurysms.

Basilar artery fenestrations are most commonly located in the proximal basilar trunk close to the vertebrobasilar junction. In the presence of a basilar fenestration, the incidence of an aneurysm is reported to be 7% (96). On the other hand; the incidence of fenestration in the presence of a vertebrobasilar junction aneurysm is reported to be 35%. Although surgical approach to the vertebrobasilar junction is difficult, endovascular access is easy; therefore, coil occlusion is the treatment of choice with good initial and midterm results as reported by Graves VB et al (244) which was also done in our patient with satisfactory immediate and midterm outcome.

F) POSTERIOR INFERIOR CEREBELLAR ARTERY ANEURYSMS:

Out of 33 posterior circulation aneurysms in our study, 1 aneurysm involved the origin of at origin of left PICA in association with left cerebellar hemispheric AVM and aneurysm was unruptured aneurysm in 1 male patient, 52 years old who had presented with left hemi facial spasm & the aneurysm was flow related saccular aneurysm with narrow neck involving left PICA origin along with left cerebellar AVM and aneurysm was less than 7mm in maximum diameter. Complete angiographic occlusion of aneurysm was obtained with coils which was persistent on DSA done 1 year later. After exclusion of aneurysm, the cerebellar AVM was partially embolized

with glue. Peluso J P et al (190) in their study reported that incidence of PICA aneurysms was 2.8% (60 of 2169) of all treated aneurysms and 18% (60 of 334) of posterior circulation aneurysms and there were 8 men (17%) and 38 women (83%) with a mean age of 54.7 years and range of 24–83 years and 37 were ruptured PICA aneurysms (79%) and 10 were unruptured PICA aneurysms. PICA aneurysms were less represented in our study compared to previous study & mean aneurysm size was 6.8 mm.

Thirty-seven aneurysms in 36 patients in their study by Peluso J P et al (190) were treated with selective occlusion of the aneurysm, sparing the parent PICA origin and ten aneurysms with origin of the PICA from the aneurysm sac or with a wide neck based on the PICA were treated with various techniques like coil occlusion of the aneurysm including the parent PICA coil occlusion of the aneurysm including the VA and PICA origin in and coil or balloon occlusion of the VA proximal to the PICA. However in our study the narrow necked saccular aneurysm was selectively occluded sparing the PICA with good clinical immediate post procedure and at follow up.

SUMMARY:

The present study was retrospective and prospective in nature undertaken to analyze the clinical manifestations at presentation & outcome at follow up of endovascular treatment of posterior circulation aneurysms and to analyze the results, outcome and rebleed in patients with posterior circulation aneurysms who underwent endovascular management as primary treatment option. The data was collected, analyzed and results were compared with literature in 33 patients of ruptured and unruptured posterior circulation aneurysms.. The observations were summarized as follows:

- 1) Majority of patients with posterior circulation aneurysms were in 40-60 years age group and mean age was 43 years (range of 7-70 years) with 20 patients being males and 13 being females (male: female ratio=3:2).
- 2) Majority (33%) of patients with ruptured aneurysms were in Hunt Hess Grade 2 with moderate to severe headache, nuchal rigidity with/without cranial nerve palsy.
- 3) Most of patients with posterior circulation aneurysms presenting with SAH were in CT Fisher grade 2 showing CT evidence of diffuse SAH, less than 1mm thick accounting to 39%.
- 4) Majority (60%) of posterior circulation aneurysms in our study were saccular (60%) in morphology and were located mostly in top of basilar artery (45%).
- 5) Posterior circulation aneurysms with narrow neck constituted approx 33% of all aneurysms in our study and narrow neck aneurysms were best suited for endovascular management using coils only.
- 6) Rupture of aneurysms manifesting as SAH was seen in approx 58% of patients with posterior circulation aneurysms and mass effect as manifestation was present in 15% of patients.
- 7) Maximum number of patients in our study had sudden onset severe headache with vomiting and drowsiness and these patients had evidence of ruptured posterior circulation aneurysms

- 8) Basilar top aneurysms were saccular in morphology whereas basilar trunk, PCA and vertebral artery aneurysms were mostly fusiform or fusosaccular in morphology. PICA aneurysm was saccular in morphology and was flow related aneurysm in relation to left cerebellar hemispheric AVM. Vertebrobasilar junction aneurysm associated with fenestration was saccular in morphology.
- 9) After the onset of bleed, majority of patients underwent endovascular coiling within 8-20 days from ictus. However overall majority of patients underwent treatment more than 1 month after onset of clinical symptoms.
- 10) Maximum diameter of aneurysms (15 aneurysms) in our study as analyzed by DSA was 7-12mm constituting approx 46% of aneurysms.
- 11) Coils alone were used to occlude the aneurysmal sac in 13 patients particularly in aneurysms with narrow neck (dome to neck ratio >2). Stent assisted coiling was done in 6 patients and balloon assisted coiling was done in 5 patients with wide necked aneurysms. Parent artery occlusion was done as primary modality of treatment using coils in cases of fusiform aneurysms.
- 12) Coils alone were used in 13 patients to occlude the aneurysmal sac and complete occlusion was angiographically achieved in 7 patients immediate post procedure and in remaining 6 patients there was residual small neck remnant. Stent assisted coiling was done in 6 patients out of which immediate complete angiographic occlusion was achieved in 2 patients and in remaining 4 patients there was minimal persisting filling of residual aneurysm and aneurysmal neck. Complete angiographic occlusion was achieved in 3 patients after treatment with balloon assisted coiling in wide neck aneurysms. Parent artery occlusion as primary treatment resulted in complete occlusion in 7 patients
- 13) Majority (18 patients) had follow up of more than 1 year. At least one DSA was performed at follow-up on 11 treated aneurysms (40% of treated aneurysms). The remaining aneurysms were followed up with TOFMRA images in 10 patients (37%) and CEMRA in 6 patients (22%) when significant artifacts occurred in TOFMRA or when doubt existed on TOFMRA.

- 14) Majority of patients at follow up had a Glasgow outcome scale of 5 and modified Rankin disability score of 1.
- 15) One patient with partially coiled basilar top aneurysm along with SCA aneurysm rebled and died in immediate post procedure period before discharge. No other patient rebled during period of follow up despite residual neck
- 16) Out of 33 aneurysms, 15 were located at basilar top and out of these 11 were ruptured and all were saccular in morphology.
- 17) In our study follow up period of basilar top aneurysms ranged from 6m-10 years with a mean of 3.8 years. Immediate post procedure angiographic occlusion of basilar top aneurysm was obtained in 8 patients (53%) particularly in those aneurysms where coils only or balloon assisted coiling of aneurysms was undertaken. Immediate post procedure residual neck remnant was seen in 5 patients (33%) and residual aneurysm was seen filling in 2 patients (14%) with stent assisted coiling .Out of 8 initial completely occluded basilar top aneurysms, 4(50%) were completely occluded on follow up and 3(38%) were showing residual neck on follow up due to coil compaction. One aneurysm with residual neck was completely occluded on follow up with remaining 5 aneurysms showing persisting residual neck.
- 18) In our study there were 6 patients with PCA aneurysms, 2 ruptured and 4 unruptured with 4 fusiform, 1, fusosaccular and 1 saccular aneurysm in morphology. One saccular aneurysm was treated with coils only preserving the parent artery and in rest 5 patients with P2 segment PCA fusiform/fusosaccular aneurysms, proximal aneurysm as well as parent artery was occluded and immediate post procedure angiogram showed occlusion of aneurysm in 4 patients and faint filling in one patient. All coiled PCA aneurysms (100%) were completely occluded on follow up.
- 19) In our study, 5 out of 33 patients had presented with basilar trunk aneurysms consisting of 3males and 2 females. 4 aneurysms were fusosaccular in morphology with partial thrombosis in 3 patients, 1 was saccular in morphology with wide neck. Stent assisted coiling was done in 3 patients and coils only were used to occlude the saccular aneurysm in one patient and coils only were used to occlude the aneurysm and basilar artery in another patient with fusosaccular aneurysm in our study Immediate post

procedure angiogram showed complete occlusion in one patient with residual aneurysm filling in three patients and residual neck filling in one patient. In 1 patient, who underwent stent assisted coiling for fusosaccular aneurysm showed complete occlusion of aneurysm on follow up. In rest of 4 cases, residual filling of aneurysm was seen which was however less in diameter compared to that prior to treatment

- 20) The study population with intracranial vertebral artery aneurysms consisted of 5 patients with 2 males and 3 females with three aneurysms ruptured. Endovascular occlusion of wide necked saccular aneurysm was done using stent (Neuroform stent) assisted coiling in 1 patient preserving the left VA and in remaining 4 patients parent artery occlusion along with occlusion of aneurysm was done as primary modality of treatment with complete occlusion in 3 patients and residual aneurysm filling in 2 patients. Complete occlusion of aneurysm in all 3 patients at follow up was noted.
- 21) One ruptured aneurysm involved vertebrobasilar junction associated with fenestration which was treated with coil occlusion only with minimal residual neck immediate post procedure and at 1 year follow up, the patient had Glasgow outcome score of 5 and modified Rankin Score of 1.
- 22) One aneurysm involved the origin of left PICA in association with left cerebellar hemispheric AVM and aneurysm was unruptured aneurysm & the aneurysm was flow related saccular aneurysm with narrow neck. Complete angiographic occlusion of aneurysm was obtained with coils which was persistent on DSA done 1 year later. After exclusion of aneurysm, the cerebellar AVM was partially embolized with glue
- 23) Periprocedural complications occurred in 6% of our patients, without permanent morbidity related to the procedure in our study.
- 24) The risk of recurrent hemorrhage during follow up after discharge appears to be extremely low; it was 0% in our series however; longer follow-up periods are required to assess the effectiveness and durability of endovascular therapy.

CONCLUSION:

From our results, we can conclude the following: First, GDC technology with further advances in technology with use of stent and balloon assisted coiling constitutes safe treatment for ruptured & unruptured aneurysms, with early successful anatomic and clinical results and low complication rates achieved. Second, recanalization of the aneurysm lumen is an important limitation in large, giant, wide necked aneurysms & narrow neck aneurysms with initial incomplete coiling. Third, complete embolization does not guarantee permanent occlusion after complete occlusion and regular long term imaging follow up is essential particularly in ruptured aneurysms. Fourth, mass effect symptoms can be effectively decreased by using the GDC technique.

The primary goal of treatment of a ruptured aneurysm is to prevent rehemorrhage. The endovascular treatment of posterior circulation aneurysms has a high rate of technical success, producing an excellent clinical outcome in the majority of cases, and is associated with low rates of morbidity and mortality in comparison with surgery.

It appears that endovascular treatment provides a reasonably safe and effective method of treating ruptured and unruptured posterior circulation aneurysms. We believe that the decision to treat these aneurysms should be a joint process, involving an experienced team of cerebrovascular neurosurgeons and interventional neuroradiologists who can weigh alternative treatment risks and efficacy. In our opinion, endovascular coil occlusion of aneurysms will play a major role in the treatment of posterior circulation aneurysms in the future.

Long-term clinical and angiographic follow-up after coiling of ruptured aneurysms confirms its efficacy as a primary treatment technique. Rebleeding rates after treatment are low, but recanalization remains an issue, even in aneurysms that are initially completely occluded. Long-term imaging follow-up is required particularly in incompletely coiled and ruptured aneurysms.

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ANNEXURES

PATIENT PROFORMA

PATIENT PARTICULARS:

Patient history:

- Name, gender, and date of birth
- History of intracranial bleedings and previous endovascular or surgical interventions
- Clinical presentation of the aneurysm(s)
- Pre-procedural clinical findings of the patient (H&H)
- CT findings on Fisher grading scale

Aneurysm features:

- Location and size (length and width)
- Neck width and dome-to-neck ratio
- Involvement of parent artery
- Aneurysm margin (regular/irregular, secondary pouch)
- Date when found and imaging method used (DSA/MRA/CTA)
- Ruptured/unruptured
- Etiology (saccular/dissecting/flow-related)

Endovascular treatment:

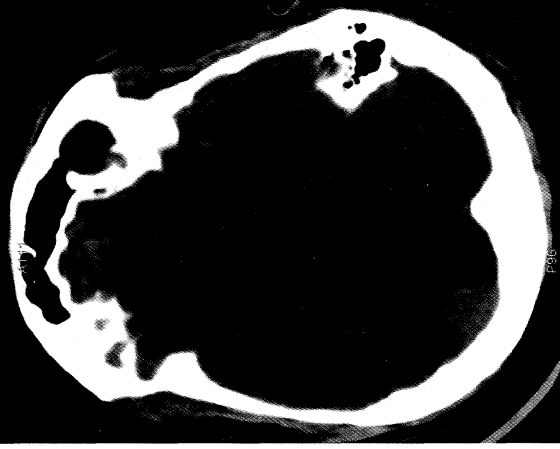
- Date of first treatment
- Time interval from rupture to treatment
- Dates of retreatments
- Number, type, and manufacturer of device used
- Techniques used (single catheter/ balloon remodeling/ stent remodeling/ parent artery occlusion)
- Procedural and delayed complications
- Angiographic result
- Clinical outcome (GOS)
- Treatment abandonment and reason for abandonment

Follow-up findings:

- Date

- **Angiographic result and imaging method (DSA/MRA)**
- **Clinical recovery (GOS)**
- **Indication for additional treatment**
- **Need and time for further follow-up**

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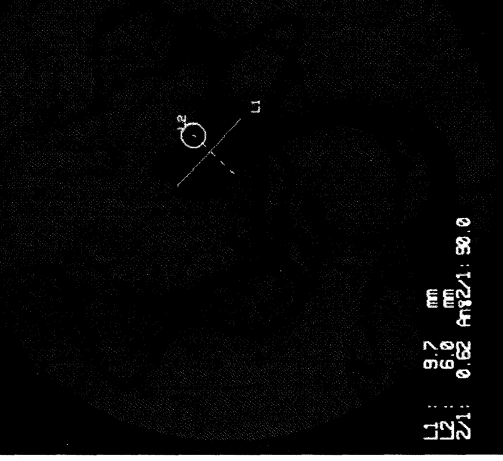
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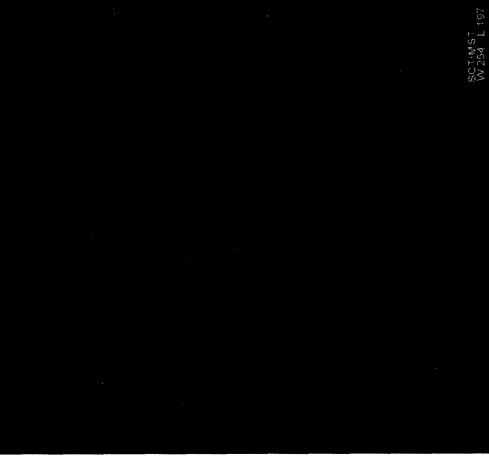
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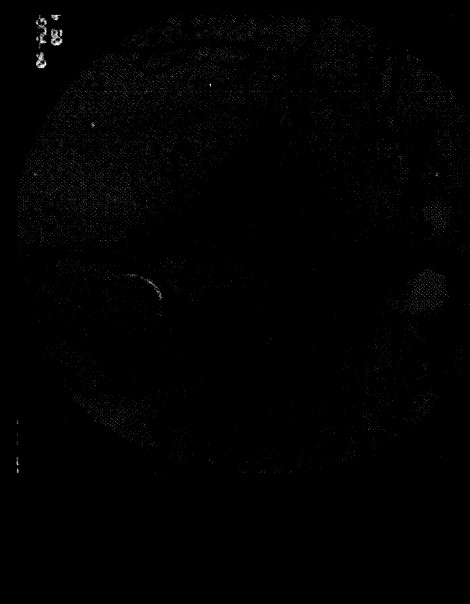
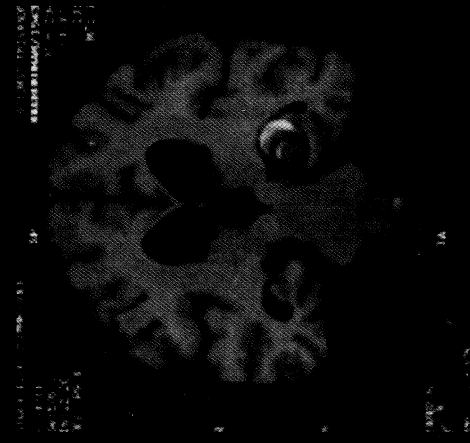
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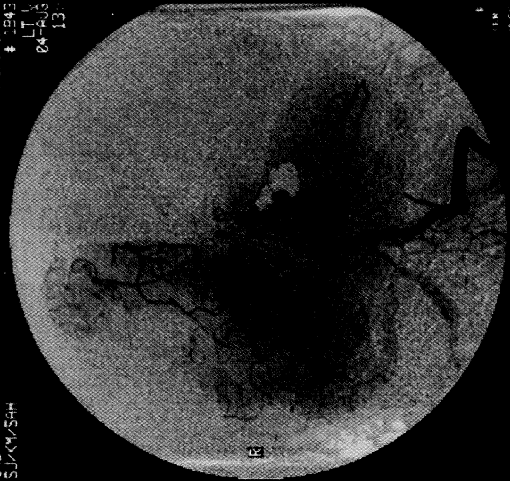


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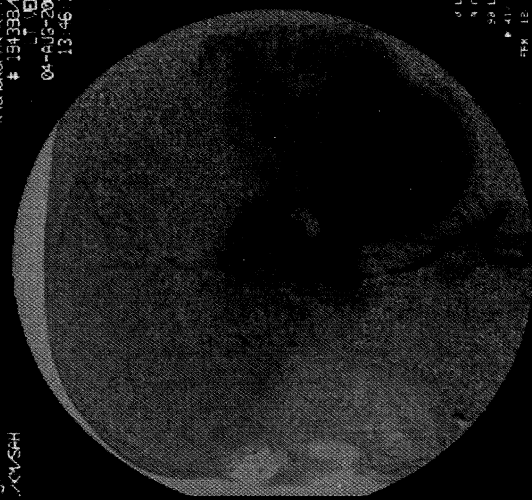
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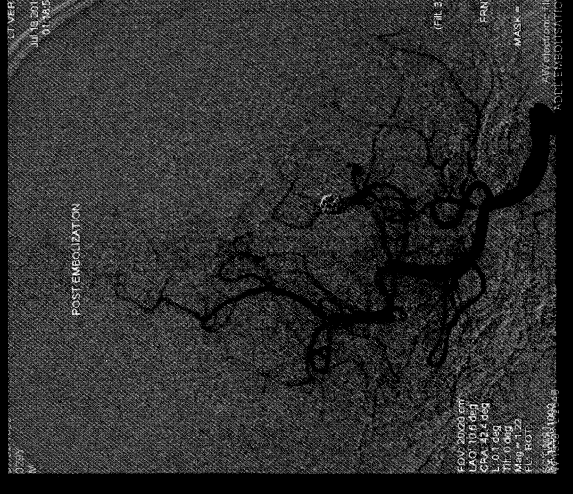
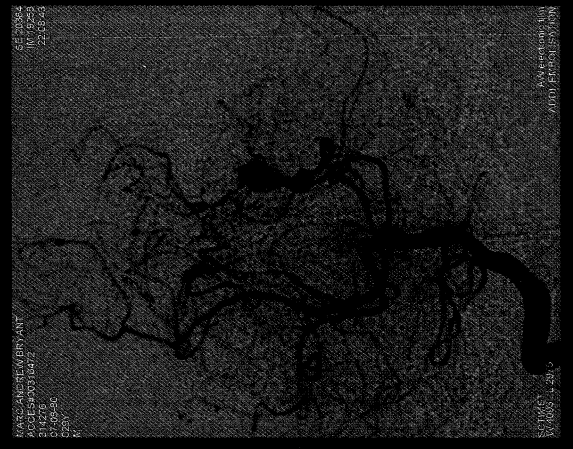
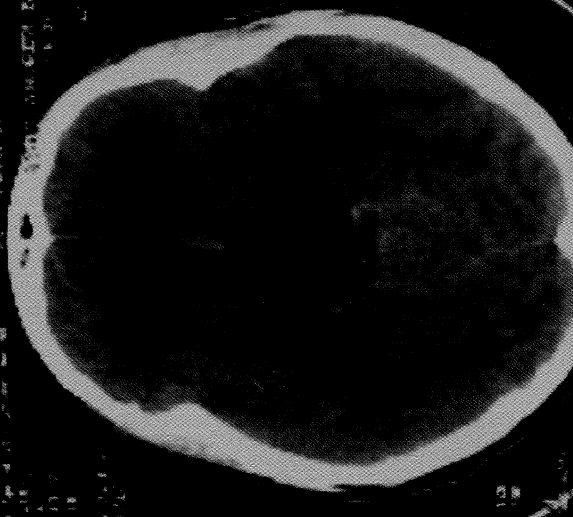


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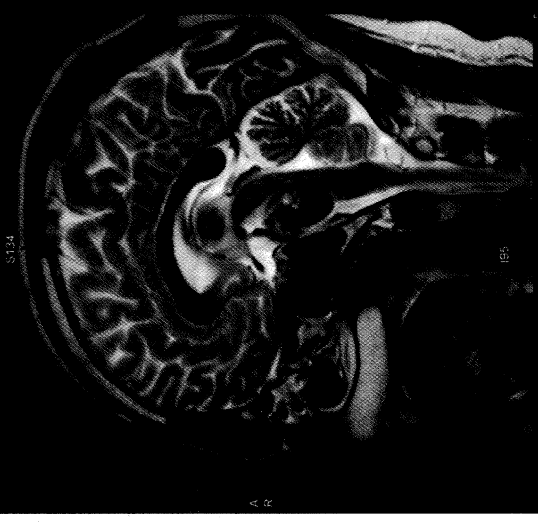
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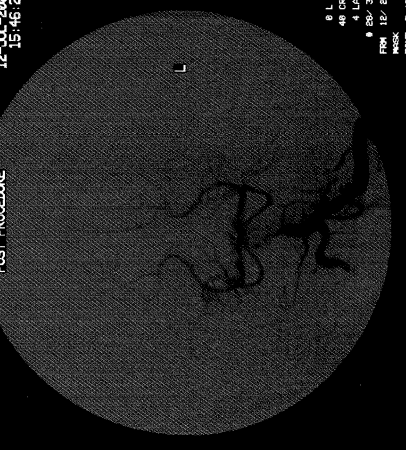
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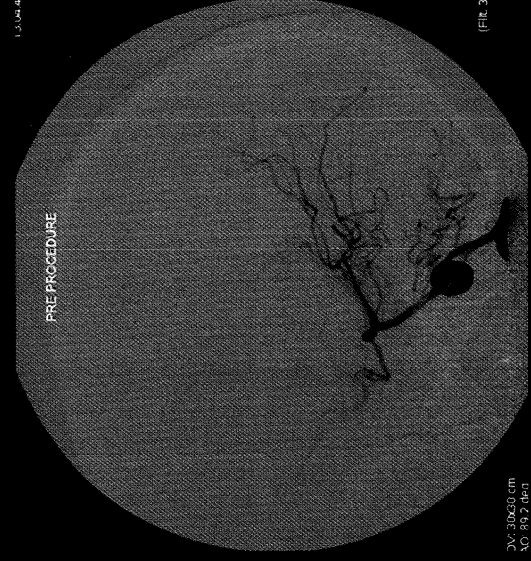
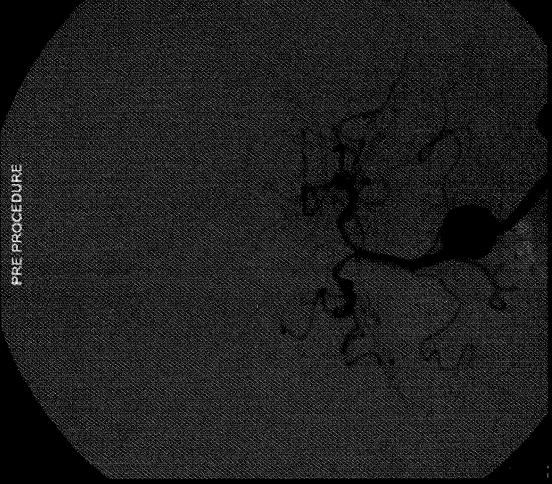
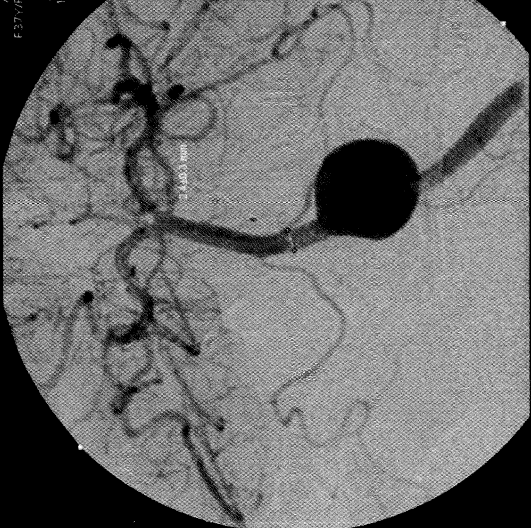
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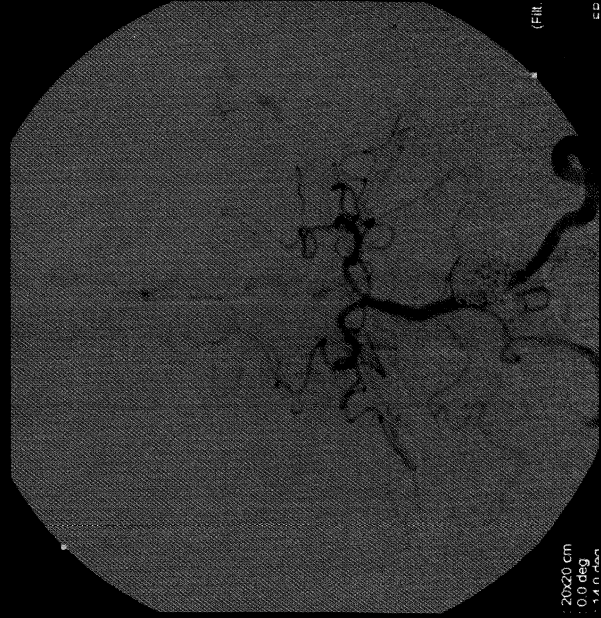
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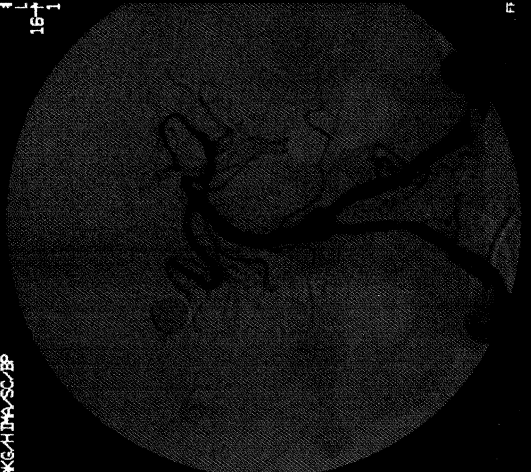
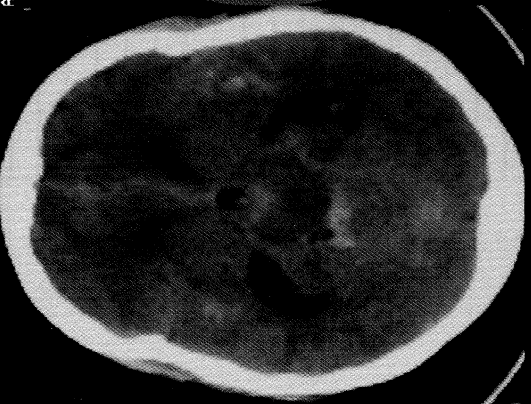
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PATIENT NO.	AGE	SEX	HUNTHESSE GRADE	CT FISHER SCORE	RUPTURED
BASILAR TOP ANEURYSMS:					
1	35	male	2	2	Yes
2	37	male	0	1	no
3	53	male	2	2	Yes
4	48	male	2	2	yes
5	58	female	0	1	no
6	58	female	2	3	Yes
7	46	male	1	2	Yes
8	66	male	0	1	no
9	55	male	2	2	Yes
10	48	female	2	3	Yes
11	43	male	0	1	no
12	41	male	2	2	Yes
13	54	female	3	2	Yes
14	56	male	3	3	Yes
15	51	female	3	3	Yes
BASILAR TRUNK ANEURYSMS:					
16	27	male	0	0	no
17	37	female	2	2	Yes
18	24	male	0	0	no
19	15	male	0	0	no
20	56	female	3	2	Yes
PCA ANEURYSMS					
21	44	male	0	0	no
22	53	male	0	0	no
23	7	female	0	0	no
24	7	male	0	0	no
25	34	female	3	3	Yes
26	29	male	3	2	Yes
INTRACRANIAL VERTEBRAL ARTERY ANEURYSMS					
27	56	male	2	2	Yes
28	52	male	2	2	Yes
29	56	female	2	2	Yes
30	25	female	0	1	no
31	37	female	0	0	no
VERTEBROBASILAR JUNCTION ANEURYSMS					
32	70	female	2	3	Yes
PICA ANEURYSMS					
33	52	male	0	0	no

MASS EFFECT	VASOSPASM	LOCATION	MORPHOLOGY	SIZE	DOMENECK RATIO
no	yes	Basilar top	saccular	9x8.5mm	>2
no	no	Basilar top	saccular	10.1x12.3mm	<2
no	yes	basilar top+SCA origin	saccular	8.5x7.3mm	<2
no	no	basilar top	saccular	3.7 x4mm	>2
no	no	basilar top	saccular	6 x3.2 mm	>2
no	no	basilar top	saccular	6.4 x4mm	<2
no	yes	basilar top	saccular	9.7 x8.5mm	>2
no	no	basilar top with partial thrombosis	saccular	13 x19mm	<2
no	no	basilar top	saccular	10 x10.2mm	<2
no	yes	basilar top	saccular	3.8 x3.5mm	<2
no	no	basilar top	saccular	9 x6mm	<2
no	yes	basilar top	saccular	6 x8mm	>2
no	no	basilar top	saccular	12 x8.9mm	>2
no	no	basilar top+Acom+L cavernous ICA	saccular(basilar)	9.7 x6mm	>2
no	no	basilar top	saccular	8.7 x9.6	>2
yes with pons & midbrain edema	no	partially thrombosed basilar trunk	fusosaccular	3.2 x1.8cm	wide neck
yes on pons	no	basilar trunk aneurysm	fusosaccular	2.8 x1.1cm	wide neck
yes	no	basilar trunk	fusosaccular	1.5 x0.7cm	wide neck
yes	no	basilar trunk	fusosaccular	3.6x1.9cm	wide neck
no	yes	basilar trunk between right SCA&PCA	saccular	7.2 x4.9mm	wide neck
no	no	Left P2-P3 PCA	Fusosaccular	2.2X0.7cm	wide neck
no, left thalamic infarct	no	left P2-P3 PCA	Fusifform	2.5 x0.7cm	wide neck
no	no	left P1PCA	saccular	8.7 x4.8mm	>2
no	no	left P2 PCA partially thrombosed	fusiform	17.9 x17.7mm	wide neck
no	yes	right P2 PCA	Fusifform	7.6 x9.2mm	wide neck
no	no	left P2 PCA partially thrombosed	fusiform	9.2 x6.9mm	wide neck
no	no	V4segment right VA	Fusifform	2.2 x0.8cm	wide neck
no	no	V4 segment right VA	Fusifform	1.6 x0.6cm	wide neck
no	no	V4 segment right VA	Fusifform	1.9 x0.8cm	wide neck
yes	no	V4 segment left VA, partially thrombosed	Fusifform	2.4x1.2cm	wide neck
no	no	V4 segment left VA	saccular	1.2 x1.1cm	wide neck
no	no	vertebrobasilar junction with fenestration of basilar artery	saccular	6.2 x2.9mm	narrow neck
no	no	left PICA origin	saccular	6.6 x5.2mm	>2

SYMPTOMS	TIME BETWEEN DIAGNOSIS&TREATMENT
severeheadache	3 days
headache	8 days
severeheadache	4 days
severeheadache	30days
sudden onset giddiness	12days
severeheadache	19days
severeheadache	6 days
bifrontal headache	45 days
severeheadache	80 days
severeheadache	3 days
gait unsteadiness,vertigo	1month
severe headache	7 days
headache&drowsiness	20 days
giddiness+headache+LOC	residual aneurysm coiling done 5 months after clipping
severe headache+LOC	3 days
recurrent headache &giddiness episodes	1year
headache,vomiting of 2 days	28 days
recurrent headache and rt hemiparesis	3 months
diplopia with recurrent headache	17 days
severe headache,vomiing,alt sensorium+left hemiparesis &diplopia	14 days
recurrent headache since 1 month	1month
numbness,paresthesia 2 weeks duration	14 days
2 episodes of seizures	1 1/2 months
intermittent headache	2 months
severe headache,vomiting,alt sensorium	6 days
severe headache,,vomiting,LOC	14 days
severe headache,vomiting,alt sensorium	12 days
severe headadche	1 month
severe headache	1month
severe headache,vomiting 2 months back	2 months
intermittent headache	10 months
sudden severe headache,vomiting ,LOC	1month
left hemifacial spasm since 2 years	2 years

TREATMENT	PARENT ARTERY OCCLUSION	PERPROCEDURALCOMPLICATIONS
COILS ONLY/STENTASSISTED/BALLOON ASSISTEDCOILING		
coils only	no	nil
stent (leo stent)assisted coiling	no	nil
stent (neuroform stent)assisted coiling	no	nil
coils only	no	nil
coils only	no	nil
balloon (sentry balloon)assisted coiling	no	nil
coils only	no	nil
balloon (sentry balloon)assisted coiling	no	nil
balloon (solistice balloon)assisted coiling	no	nil
coils only	no	nil
balloon(sentry &equinox)assisted coiling	no	nil
coils only	no	nil
coils only	no	nil
balloon(hyperfoam)assisted coiling	no	nil
coils only	no	nil
stent(neuroform)assisted coiling	no	wire tip broke &in rt PCA
stent(leo stent)assisted coiling	no	nil
coils only with occlusion of aneurysm&basilarartery distal toPICA	yes	nil
stent(neuroform)assisted coiling with coil occlusion of left VA	yes(left VA)	Wire tip broke &in rt PCA
coils only	no	nil
coils only	yes	nil
coils only	yes	nil
coils only	no	nil
coils only	no	nil
coils only	yes	nil
coils only	yes	nil
coils only	yes	nil
coils only	yes	nil
coils only	yes	nil
coils only	yes	nil
stent(neuroform) assisted coiling	no	nil
coils only	no	nil
coils only	no	nil

INITIAL OCCLUSION	TREATMENT SESSION
RESIDUAL NECK/INTERSTICES FILLING	
complete occlusion	1
residual aneurysm filling	1
neck remnant+residual aneurysm	
complete occlusion	1
complete occlusion	1
complete occlusion	1
complete occlusion	1
complete occlusion	1
residual neck of 3 x4.2mm	1
complete occlusion	1
small neck remnant	1
small neck remnant	1
small neck remnant	1
complete occlusion	1
small neck remnant	1
residual aneurysm filling	1
residual aneurysm filling	1
residual distal aneurysm part filling	1
complete occlusion	2
residual neck filling	1
complete occlusion	1
complete occlusion	1
complete occlusion	1
residual aneurysm filling	1
complete occlusion	1
complete occlusion	1
complete occlusion	1
complete occlusion	1
complete occlusion	1
minimal residual filling from opp sideVA	1
minimal residual filling of aneurysm	1
minimal residual aneurysmal filling	1
complete occlusion	1

FOLLOWUP			
DURATION/GLASGOW OUTCOME SCORE/MODIFIED RANKIN SCORE/OCCLUSIONOF ANEURYSM/NECK			
3Years	5	1	no residual filling on CEMRA
3Years	5	1	no residual filling on CEMRA
died after 6days due to rebleed with SAH and diffuse cerbral edema			
4 1/2 years	5	1	residual neck of 0.29 x0.22cm on TOFMRA
10 years	5	1	residual neck of 0.34 x0.5cm on TOFMRA
7months	5	1	no residual aneurysm filling on DSA
9 months	5	1	small residual neck0.32 x0.38cm on DSA
died after 2day due to myocardial infarction			
7 1/2 years	5	1	0.65 x0.5cm residual neck on TOFMRA
7months	5	1	no residual aneurysm filling on DSA
6 years	5	1	residual neck of 0.6 x0.5cm on TOFMRA
5 years	5	1	residual neck of 0.4 x0.3cm on CEMRA
6months	5	1	residual neck of 0.3 x.4cm on DSA
2 years	5	1	no residual filling on CEMRA
2 years	5	1	residual neck of 0.53 x0.35cm on TOFMRA
2years	4	3	residual aneurysm lumen 1.2 x0.9cm on CEMRA
7 months	5	1	no residual aneurysm filling on TOFMRA
10 years	5	1	residual aneurysm of 8.8 x7.2mm on DSA
2 1/2 years	5	1	residual aneurysm(3x1cm) on DSA
10 months	5	1	residual neck of 0.36 x0.46mm on CEMRA
10years	5	1	no residual filling on DSA
5 years	5	1	no residual filling on DSA
3 years	5	1	no residual filling on DSA
2 years	5	1	no residual filling on TOFMRA
7 1/2 years	5	1	no residual filling on TOFMRA
4 months	5	1	no residual filling of aneurysm on TOFMRA
5 years	5	1	no residual filling on DSA/TOFMRA
died after 3 days due to cardiac arrest			
1 year	5	1	no residual filling of aneurysm TOFMRA
1 week	5	1	-----
1 year	5	1	clinical assesment,no imaging done
1 year	5	1	no residual filling of aneurysm on DSA

