

ENDOVASCULAR MANAGEMENT OF DURAL ARTERIO VENOUS FISTULAS - RETROSPECTIVE & PROSPECTIVE ANALYSIS



DEPARTMENT OF IMAGING SCIENCES AND
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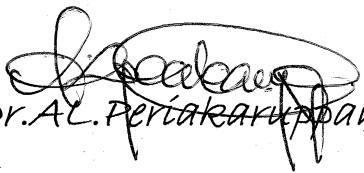


DECLARATION

I, Dr. AL.Periakaruppan hereby declare that I have actually carried out the project "ENDOVASCULAR MANAGEMENT OF DURAL ARTERIO VENOUS FISTULAS -RETROSPECTIVE & PROSPECTIVE ANALYSIS" independently under supervision and guidance in this institute.

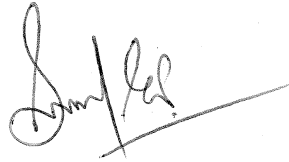
Date - September 2008

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CERTIFICATE

This is to certify that the work contained in this thesis have been carried out by Dr. AL.Periakaruppan in the Department of Imaging Sciences and Interventional Radiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology ,Trivandrum ,during his rotatory postings as per schedule, under my guidance and is to my satisfaction.



Date- 30/8/08

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INTRODUCTION

INTRODUCTION

The field of medicine has seen sea change in the treatment of almost all human ailments over the past few decades. The technological advancements have contributed to the field to a large extent.

The discovery of x-rays by Sir W.C.Roentgen in 1895 revolutionized the field of medicine. However, the crude fluoroscopy was available for clinical use only by the early 1950s. This was followed by the development of angiographic studies, with Seldinger, in 1953, described the use of a hollow-bore needle and guide-wire system for accessing the femoral artery (1). This led to the birth of interventional radiology on January 16, 1964, when Dotter percutaneously dilated a tight, localized stenosis of the superficial femoral artery (2). Over the years, the imaging and fluoroscopy technology has been improved in quality and resolution. With the development of high-end fluoroscopic machines and the various materials available, interventional radiology has today become an inseparable therapeutic modality.

We deal with a wide variety of neurovascular lesions at our institute. DAVFs (DAVF) are one of them. DAVFs are abnormal connections between the dural branches of external carotid artery (ECA), cavernous internal carotid artery (ICA), or vertebral artery (VA) with the dural sinuses or cortical veins (3).

DAVFs are rare vascular abnormalities that can occur anywhere within the intracranial dura mater. Arterial supply is usually from adjacent branches of dural arteries; less frequently from osseous branches, while pial supply is rare. Venous drainage may occur into the dural sinuses, into osteodural veins, retrogradely via leptomeningeal veins. The true incidence of DAVF is unknown (4). However, the reported incidence of intracranial DAVFs is approximately 10-15% of all intracranial vascular abnormalities. Good percentages of DAVF remain clinically silent or involute spontaneously and therefore the true incidence may be much more (5). Present evidence suggests that DAVFs are acquired lesions and present later in life than arteriovenous malformations (AVMs) (6). Digital subtraction angiography (DSA) remains the gold standard for diagnosing these fistulas. Endovascular treatment is presently the first line of treatment available for their management.

Our centre is a major tertiary referral centre for management of all types of neurovascular disorders. Our department of IS& IR has been actively involved in the endovascular management of various neurovascular lesions. We hereby review our experience in the endovascular management of DAVFs over the last 12 years.

DURAL ARTERIO VENOUS FISTULA

DURAL ARTERIO VENOUS FISTULA

Intracranial DAVFs represent 10–15% of all intracranial vascular malformations. DAVFs are rare vascular abnormalities that can occur anywhere within the intracranial dura mater. Patients may be clinically asymptomatic or may experience symptoms ranging from mild symptoms to fatal hemorrhage, depending on the location (e.g., cavernous sinus, transverse-sigmoid sinus, tentorium, superior sagittal sinus, anterior cranial fossa) and venous drainage pattern of the AVF. Although several classification systems have been developed for understanding the type of DAVFs in relation to grade the risks of dural AVFs, those suggested by Cognard et al (4) and Borden et al (7) are the most widely used. DAVFs that drain via the retrograde leptomeningeal cortical venous drainage channel show a significantly high rate of aggressive symptoms.

DAVFs are a heterogeneous group of lesions that were initially (before mid-1970s) considered to be congenital in origin and to be associated with other vascular lesions. However, presently its acquired nature is well established. They can be classified as dural sinus malformations, infantile DAVFs, and adult types of DAVFs (8). Only the dural sinus malformation is truly congenital in origin. Many reports have documented the acquired nature of DAVFs (9). Various etiologies are postulated: e.g., anomalies of venous system, dural sinus thrombosis or occlusion (by surgery or tumor), head trauma, intracranial infection, transcranial surgery, association with pregnancy, delivery and menopause, increased systemic thrombotic activity, cortical vein thrombosis (CVT), role of hormonal changes (use of oral contraceptive and pregnancy) causing increased

angiogenesis, tumors (meningiomas obstructing dural venous outflow), general surgery, otitis, and sinusitis (10).

Vascular diseases associated with dural arteriovenous shunts include cerebral AVM, maxillofacial AVM, hereditary hemorrhagic telangiectasia, bone AVM, cavernomas, and intradural or extradural arterial aneurysms (10). Multiple DAVFs at separate locations in the same patient have also been reported (11).

The common predisposing factor for DAVFs appears to be venous sinus thrombosis (12). The venous hypertension developing after venous thrombosis opens up the microvascular connections within the dura (3). Without intervention, these channels become hypertrophied resulting in direct shunting between the arteries and veins (12). When the fistula grows and becomes more diffuse, it recruits pial supply from parenchymal vessels. This may lead to the angiomatous network of multiple feeding arteries and numerous AV shunts within a partially recanalized sinus that is frequently seen at angiography. The involved dural sinus receives arterialized blood flow that can lead to mechanical obstruction of the sinus and result in retrograde drainage of blood away from the sinus and into the cortical veins. Histopathological analysis of excised DAVFs has revealed thrombosis and angiographic progression of sinus thrombosis to DAVF. In addition, dilatation of the cortical veins may occur, predisposing the patient to intracranial hemorrhage.

PATHOPHYSIOLOGY

Understanding of the pathophysiology of DAVFs came from the school of La Salpetriere, which emphasized the role of the cortical venous drainage of these dural shunts (13). Henceforth, the role of the subarachnoid and subpial venous drainage could be correlated with the type of neurological manifestation and the natural history of dural shunts. The association with venous thrombosis was first described by Djindjian and Merland (1978) and subsequently by Chaudary et al (9).

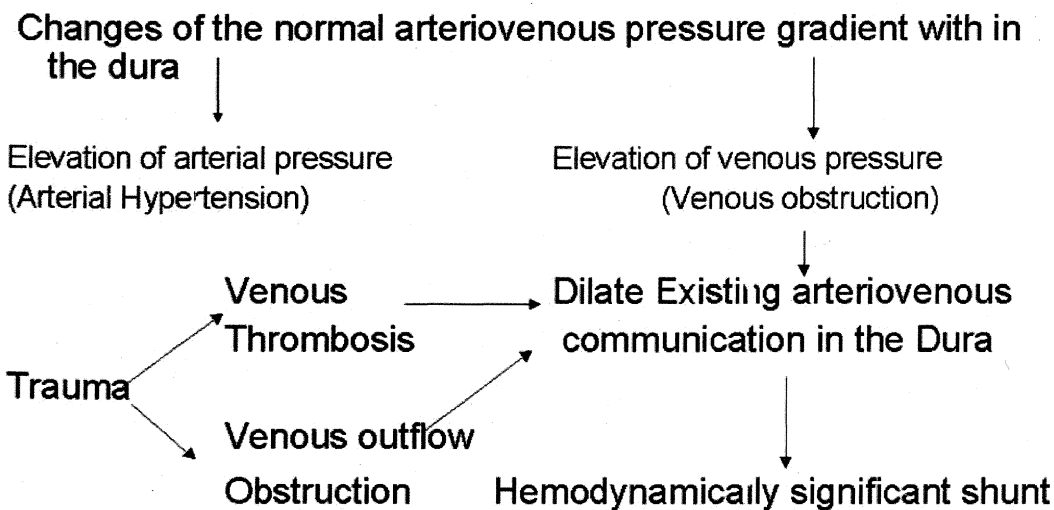
Various etiologies are postulated: e.g., anomalies of venous system, venous thrombosis, head trauma, transcranial surgery, association with pregnancy, delivery and menopause, increased systemic thrombotic activity, cortical vein thrombosis (CVT), role of hormonal changes (use of oral contraceptive and pregnancy) causing increased angiogenesis, tumors (meningiomas obstruct dural venous outflow), general surgery, otitis, and sinusitis (10). The wide variety of these possible etiological factors demonstrates the spectrum of events that can lead to the same result, i.e., the creation of a shunt inside the dural.

These factors by themselves should therefore not be considered as the direct cause of DAVFs, but rather they make up an environment that may be conducive to the development of DAVF, depending on the individual host response to their presence. DAVF in adults are triggered by factors that stimulate angiogenesis. If this angiogenic process is simultaneously associated with loss of some of the venular surface properties, then venous thrombosis is likely to occur. It has been noted that angiogenic growth

Pathophysiology of Intracranial HTN

- Increased pressure in dural sinuses
- Directly related to flow rate of fistula
- Decreased CSF resorption → Increased ICP
- CSF resorption = CSF pressure – SSS pressure

Probable – Cascade of Events



factors such as basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF) are expressed in the DAVFs (14). The absence or presence of thrombosis is associated with non sprouting angiogenesis will determine the clinical presentation and natural history of DAVFs.

OSTEODURAL-VENOUS COMPLEX

At birth, the total cerebral venous drainage is directed towards the posterior sinuses, i.e. a true sinusal system (13). Few months after birth that the so-called cavernous capture of sylvian vein will occur and offer the infant brain an alternate drainage via the cavernous plexus toward the orbit or the pterygoid plexus and into the external jugular venous system. The dura mater along the convexity of the cranial vault has a different origin from the dural along the base of the skull (membranous vs. cartilaginous bone). Septations may occur within the dural sinuses and result in separate venous channels, one of these channels may be used for the brain to drain while the other is sometimes exclusively used for the drainage of the DAVF and may be the specific target for treatment (13).

There are no dural sinuses in the midline at the skull base; these venous plexiform channels are the cavernous plexus (and its drainage in pterigoid plexus), reteroclival venous plexus, the venous plexus along the anterior margin of the foramen magnum, and the spinal venous plexus (13). Transdural veins are portions of the cerebral veins intracranially, before they join a sinus, or an epidural collector. DAVFs at these locations are called as extrasinusal dual lesions (15). A peculiar group of lesions are the

intracranial arteriovenous shunts fed by the dural arteries (that also supply the bone), which are located within the bony structures along the convexity or at the base of the skull: the so called osteodural AVFs.

CLINICAL SYMPTOMATOLOGY

Patients with DAVFs may be completely asymptomatic. Symptoms, when present, may range from mild symptoms to fatal hemorrhage. The symptoms depend on the location and venous and drainage pattern of the DAVF. The clinical symptoms of the majority of DAVFs involving the superior sagittal, lateral, sigmoid, and straight sinuses are often related to the route of venous drainage (8). A petrous region DAVF draining into the transverse or sigmoid sinus commonly produces pulsatile tinnitus, with or without an associated bruit. Such patients may be managed conservatively, depending upon the pattern of venous drainage. Bruit and headache are common symptoms in DAVFs and are usually unrelated to cortical venous drainage. Increased intracranial pressure, papilloedema, and CSF reabsorption abnormalities result in ventricular enlargement. Carotid cavernous DAVFs often present with the classic clinical findings of proptosis, chemosis, and bruit. Treatment of a carotid cavernous (cavernous venous plexus) DAVF is usually undertaken to protect against ocular and visual complications. An interesting feature of cavernous sinus DAVFs is the occurrence of paradoxical symptoms, when ipsilateral cavernous sinus thrombosis causes rerouting of venous drainage to the contra lateral orbital veins.

Symptoms of DAVFs may be characterized further as either nonaggressive (e.g.,

tinnitus) or aggressive (e.g., intracerebral, subarachnoid, or subdural hemorrhage and neurologic deficits) (16, 17). These aggressive features are usually due to venous hypertension, although neurological deficits may be secondary to arterial steal (12). A DAVF demonstrating venous occlusive disease is at a higher risk for complications from the arterialized collateral venous system. Aggressive neurological symptoms are more common in male patients who have aggressive angiographic features like retrograde leptomeningeal venous drainage, variceal or aneurysmal venous structures, and galenic venous drainage (18). Earlier treatment of DAVFs that have these features is important to prevent complications. The risk of conversion from a benign to an aggressive DAVF is small but repeat angiography is indicated if the clinical picture appears to progress (19).

Elevated venous pressure in veins draining the orbit may produce orbital venous congestion, transudation of interstitial fluid into the orbit with resultant proptosis, raised intraocular pressure because of impaired drainage of aqueous humor and secondary glaucoma. Elevated venous pressure and increased intraocular pressure can compromise retinal perfusion and result in severely diminished visual acuity. Diminished arterial pressure to the orbit may also result from a “steal” away from the ophthalmic artery into the fistula through reversal of flow in the supraclinoid carotid artery into the fistula. Edema of orbital contents because of impaired orbital venous drainage can result in mechanical limitation of EOM, and can contribute to the development of diplopia. Diplopia however may be a direct result of ophthalmoplegia from a cavernous sinus syndrome, caused by mass effect on the cavernous sinus (CS) from the fistula, or because of cranial nerve injury by direct trauma (20). Although the sixth cranial nerve is the most

often involved because of its longest course, and any of the cranial nerves can be affected alone or in combination.

The meningeal arteries supply the cranial nerves and ischemia due to an arterial steal phenomenon may contribute to some of the cranial nerve palsies encountered in these lesions. CNS symptoms are related to the venous side of the shunt. Patients showing reversal of flow in the sinus with cortical venous rerouting and venous ectasia are at increased risk of ICH and SAH; and should receive treatment immediately. When this drainage is seen towards the perimedullary venous plexus, patients can present with myelopathic features.

CLASSIFICATION SYSTEM

The initial angioarchitectural classification of DAVFs proposed in 1978 by Djindjian and Merland was based on venous drainage (21); it is recently modified by Cognard et al (4). Table shows two commonly used classification systems. The Borden classification has three subtypes and is user-friendlier (7). The Cognard system is based on venous drainage. It provides more detailed and elaborate information on the presence of normal or abnormal venous drainage and presence or absence of cortical venous recruitment and also takes into account the spinal perimedullary venous drainage (6). Thus, it enables accurate comparison of clinical and radiological parameters. Recently Moret has classified DAVF into sinodural, osteodural, dural arachnoid and dural pial. He has also classified according to location into superficial, deep, cavernous, anterior cranial fossa and congenital giant torcular DAVF.

CLASSIFICATION OF DURAL ARTERIO-VEINOUS FISTULA (Cognard et al).

Type	Borden classification
1	Venous drainage directly into dural venous sinus or meningeal vein
2	Venous drainage into dural venous sinus with CVR
3	Venous drainage directly into subarachnoid veins (CVR only)
	Cognard et al.
I	Venous drainage into dural venous sinus with antegrade flow
Ila	Venous drainage into dural venous sinus with retrograde flow
Ilb	Venous drainage into dural venous sinus with antegrade flow and CVR
II a + B	Venous drainage into dural venous sinus with retrograde flow and CVR
III	Venous drainage directly into subarachnoid veins (CVR only)
IV	Type III with venous ectasias of the draining subarachnoid veins
V	Direct drainage into spinal perimedullary veins
	(CVR indicates cortical venous reflux)

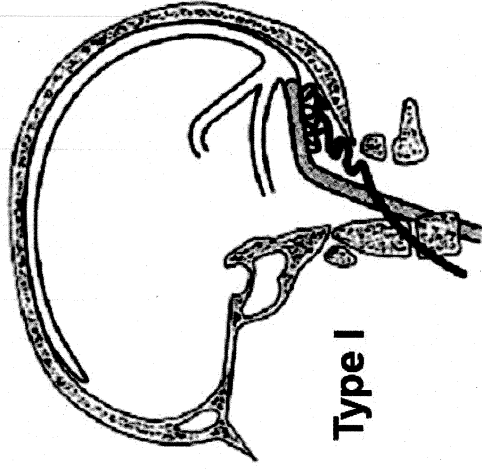
BARROWS CLASSIFICATION OF CAVERNOUS FISTULAS

Type A fistulas is a direct connection between the intracavernous ICA and the CS. They usually are high-flow and high-pressure fistulas. Mostly traumatic

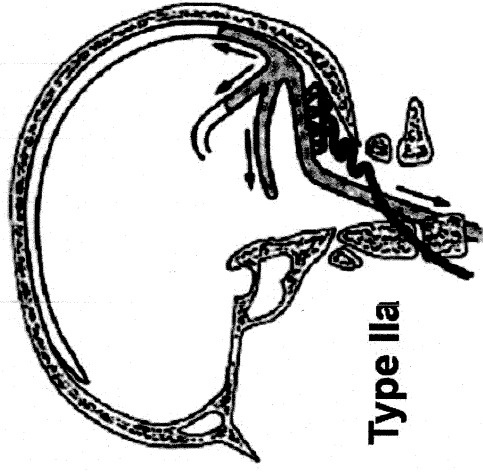
Type B fistulas consist of a dural shunt between intracavernous branches of the ICA and the CS.

Type C fistulas comprises of a dural shunt between meningeal branches of the external carotid artery (ECA) and the CS.

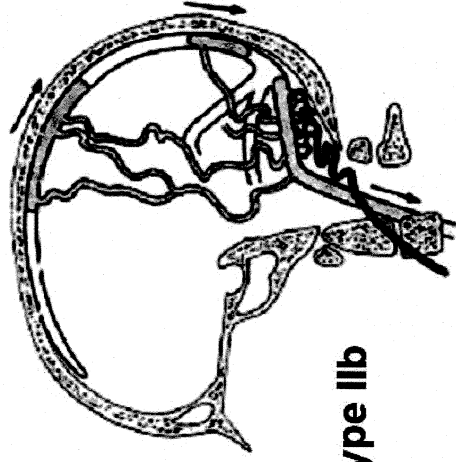
COGNARD CLASSIFICATION



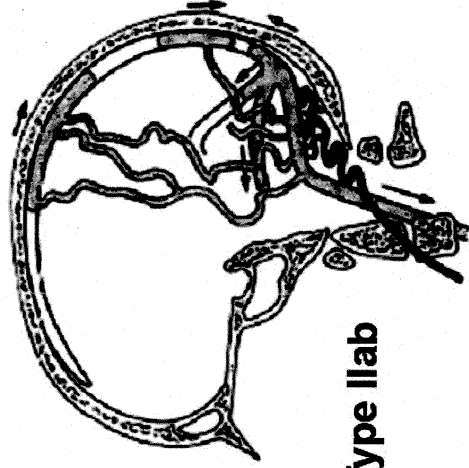
Type I



Type IIa

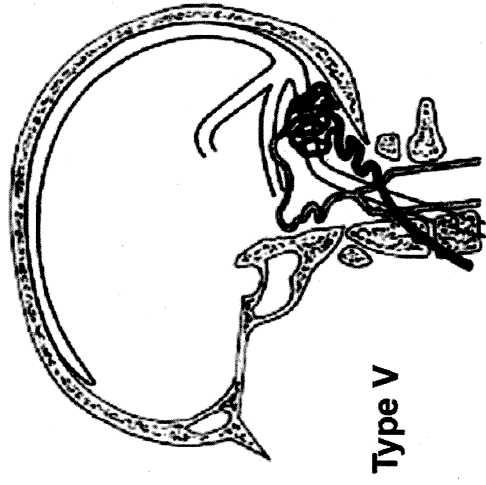
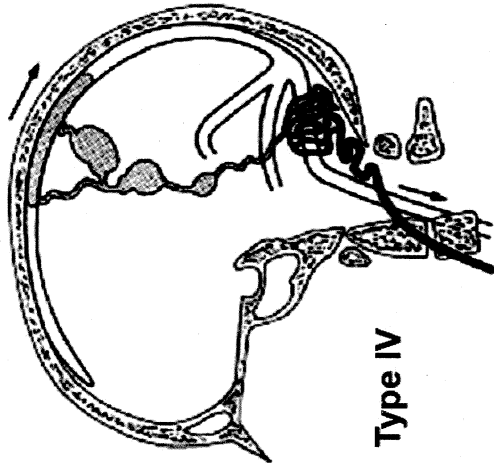
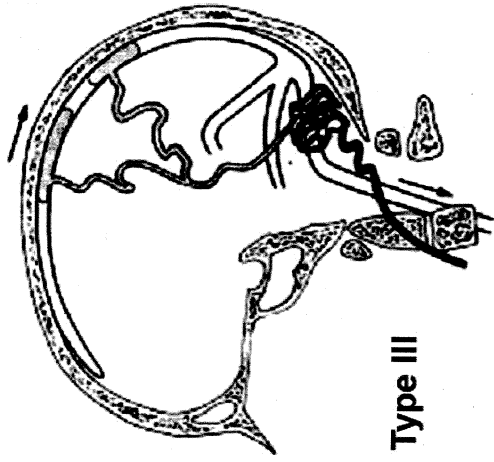


Type IIb



Type IIab

COGNARD CLASSIFICATION



Type C fistulas comprises of a dural shunt between meningeal branches of the external carotid artery (ECA) and the CS.

Type D fistulas are a combination of types B and C, with dural shunts between ICA and ECA branches and the CS.

Types B, C, and D tend to be low-flow and low-pressure fistulas with a slower progression of signs and symptoms.

THE ANGIOGRAPHIC FEATURES OF THE DAVF:

Angiography remains the gold standard for diagnosis and planning of therapy. It entails anatomical and hemodynamic assessment of the fistula. The aim is not only to identify the arterial feeders and the site of the fistula but also to identify the pattern and direction of venous drainage of the fistula and normal cerebral parenchyma. Selective injection of all potential arteries that may contribute to the region of dural pathology needs to be done. The acquisition of images should begin in the early arterial phase and continue into the late venous phase of the study.

A dual arterial injection (competitive angiogram) may be required to study the venous drainage of fistula and normal brain. In this technique first selective injection is done in ICA followed by ECA. Then by using resubtraction and changing of the mask runs, drainage of the fistula and normal brain parenchyma can be superimposed. The angiogram should be analyzed for feeders, type of venous drainage, retrograde flow, occlusion of sinuses, and circulation time. The common angiographic findings seen are

with the distal internal maxillary artery (IMA) via the artery of the foramen rotundum and with the middle meningeal artery via small temporal rami.

VENOUS DRAINAGE OF THE FISTULA

The venous drainage can be into the sinus only. If the fistula is secondary to the sinus thrombosis or if the fistula flow is very high, it can lead to reversed flow in the sinus. When ante grade flow in the sinus is obstructed it can produce reflux into the deep veins, which is called as cortical venous rerouting. Venous ectasia associated with cortical venous rerouting can have aggressive presentation like intracerebral or subarachnoid hemorrhage as initial presentation. Perimedullary venous drainage will have myelopathy as initial presentation. It can extend even lower down into the thoraco lumbar region.

The more anterior the cavernous dural fistula, more prominent is the anterior venous drainage. However, in cases of partial thrombosis or incomplete filling of the CS, anterior venous drainage can be totally absent, even with an anterior fistula. The lack of reflux into the ophthalmic vein is unusual. If noted, the possibility of an underlying aneurysm obstructing the anterior part of the cavernous sinus and thus the exit into the ophthalmic vein should be considered (17).

Posterior venous drainage is often associated with anterior venous drainage in cases

of cavernous DAVFs. The inferior petrosal sinus can be the only posterior venous drainage, but there may also be drainage through the superior petrosal sinus. The more posterior is the fistula, the more prominent is the posterior venous drainage. Contralateral venous drainage through the coronary veins is frequently associated with ipsilateral venous drainage. It is exceptional to find that the only venous drainage is via the contralateral cavernous sinus.

Superior venous drainage through the superficial sylvian vein and collaterals and inferior venous drainage through the pterygoid plexus is sometimes associated with the other types of drainage. The superficial sylvian venous network can be well developed in cases with fistulas and a total steal (17).

It is essential, therefore, to analyze the angiographic anatomy of a given dural fistula in order to plan the appropriate endovascular therapy. The angiographic features also help to identify the patients who need an immediate treatment as opposed to those who can be managed conservatively or electively at a later date.

AIMS

AIMS AND OBJECTIVES

1. To review, **prospectively and retrospectively**, all types of DAVFs that were treated in the Department of Imaging Sciences & Interventional Radiology (IS&IR), Sree Chitra Tirunal Institute of Medical Sciences and Technology (SCTIMST), over a period of 12 years.
2. To analyze different clinical presentations of DAVFs, in relation to various types of DAVFs.
3. To analyze various risk factors in relation to DAVFs.

4. To study the angioarchitecture of various types of DAVFs.
5. To study the details of the procedure and management protocol of different types of DAVFs.
6. To reevaluate and restandardize the diagnostic and therapeutic protocol for future.
7. To analyze the outcome of the endovascular management of DAVFs, in relation to its varied clinical presentation, different routes of approach, various embolic materials used and types of DAVFs treated.
8. To analyze the complication rates in relation to various embolic materials and types of DAVFs treated and find out measures to avoid complication and how to attend them if occurred to get best result.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Yasargill noted that Rizzoli, in 1881, was the first to describe an AVM that involved the dura mater and Sachs reported the first, angiographic description in 1931. Subsequently cranial dural fistulas have been most frequently described at the transverse sinus and cavernous sinus, although they occur at every cranial dural sinus. DAVF are fistulas connecting the branches of dural arteries to dural veins or a venous sinus. Digital subtraction angiography (DSA) remains the gold standard for diagnosing these fistulas. They consist of numerous tiny connections between branches of dural arteries and veins or a venous sinus (22). In the late 1970s, Castaigne and Djindjian proposed an acquired etiology (21). They suggested that DAVFs might develop due to

the opening up of existing micro shunts within the dura and by angiogenesis, leading to the development of new shunts. Various etiologies are postulated: e.g., anomalies of venous system, venous thrombosis, head trauma, transcranial surgery, association with pregnancy, delivery and menopause, increased systemic thrombotic activity, cortical vein thrombosis (CVT), role of hormonal changes (use of oral contraceptive and pregnancy) causing increased angiogenesis, tumors (meningiomas obstructing dural venous outflow), general surgery, otitis, and sinusitis (13).

Conditions associated with DAVFs include stenosis or occlusion of the draining dural sinuses, sinus thrombosis, prothrombin gene mutation, resistance to activated protein (APCR), mutation in factor V gene, and presence of antiphospholipid antibodies. DAVFs in adulthood are an acquired disease and there is no relationship to hereditary or developmental disorders.

The common predisposing factor for DAVFs appears to be venous sinus thrombosis (12). The venous hypertension developing after venous thrombosis opens up the microvascular connections within the dura (3). Without intervention, these channels become hypertrophied resulting in direct shunting between the arteries and veins (3). When the fistula grows and becomes more diffuse, it recruits pial supply from parenchymal vessels. This may lead to the angiomatous network of multiple feeding arteries and numerous AV shunts within a partially recanalized sinus that is frequently seen at angiography. The involved dural sinus receives arterialized blood flow that can lead to mechanical obstruction of the sinus and result in retrograde drainage of blood

away from the sinus and into the cortical veins. Histopathological analysis of excised DAVFs has revealed thrombosis and angiographic progression of sinus thrombosis to DAVF. In addition, dilatation of the cortical veins may occur, predisposing the patient to intracranial hemorrhage.

Another mechanism for DAVF evolution is the release of angiogenic growth factors such as Vascular Endothelial Growth Factor (VEGF) and basic Fibroblast Growth Factor (bFGF), which promotes neovascularization and development of a DAVF. In DAVFs with a patent sinus, cortical venous pathways usually do not develop and hemorrhagic complications with these DAVFs are infrequent (23).

The incidence of DAVF at various locations as reported in literature is as follows: transverse sinus 50%, cavernous sinus 16%, tentorium cerebelli 12%, and superior sagittal sinus, 8% (24).

NATURAL HISTORY

The natural history of DAVFs is poorly documented and mostly reviewed retrospectively. Davis *et al* followed 102 patients with intracranial DAVFs for an average of 33 months, with complete follow up in 91%. In his series, 55 patients had DAVFs without leptomeningeal and cortical venous reflux and, of these, 32 received no treatment. Among the patients who received no treatment, 81% had symptom improvement or complete resolution as compared to 86% of the treated patients with the same angioarchitecture. In the same series, there were 46 patients who had DAVFs with

leptomeningeal or cortical venous reflux and 14 of these declined further treatment. At follow-up, this group had an 11% non-hemorrhagic neurological deficit rate per year and a 20% intracerebral hemorrhage (ICH) rate per year (13). In an updated review of 118 patients with DAVFs and leptomeningeal reflux, the Toronto team demonstrated an annual risk for nonhemorrhagic neurological deficit of 6.9% and a risk of 8.1% for hemorrhage, with an annual mortality rate of 10.4% (13).

Many DAVFs remain stable and do not change on follow-up. Some involute spontaneously; this may be due to thrombosis following DSA. Some DAVFs undergo progressive recruitment of pachymeningeal feeders with the worsening of symptoms. The actual progress of lesions once symptoms develop is unknown. By location, an aggressive course is seen in 75% of anterior fossa lesions, 79% of tentorial lesions, 60% of foramen magnum lesions, and 29% of transverse sinus lesions (13). Factors predisposing to an aggressive course include leptomeningeal (cortical) venous drainage, galenic drainage (deep veins), variceal or aneurysmal venous dilatations, and location at the tentorial incisura. This incidence parallels that of the leptomeningeal retrograde venous drainage at each of these locations. The reported annual morbidity and mortality rates of aggressively presenting DAVFs may vary widely, ranging from 1.8-20% per year (13).

The commonly recognized initial symptoms of a DAVF are tinnitus, headache, proptosis, decreased cognitive function, and neurological deficits associated with intracranial hemorrhage (25). However, recent reports suggest that it is not uncommon for elderly patients with a DAVF to present with disturbance of higher

cognitive function but without other neurological deficits (19). A change in the clinical symptomatology may (the disappearance of the tinnitus) imply a change in the venous drainage pattern and should lead to renewed investigation (28). Spontaneous regression has been reported following hemorrhage (29).

CLASSIFICATION SYSTEM

Various classification methods have been adopted that attempt to explain the significance of the angiographic anatomy; namely, the pattern of venous drainage and the clinical presentation and outcome. The two commonly used classification systems are shown in the introduction of DAVF. With only three sub-types the Borden classification is user friendly (7). However, the Cognard system is more detailed and elaborates on the direction of flow, whether normal (ante grade) or retrograde and the presence or absence of cortical venous recruitment. Such definition enables more accurate comparison of clinical and radiological parameters. In addition, spinal perimedullary venous drainage is specifically recognized (4).

In a retrospective review of 102 DAVFs in 98 patients Davies et al. reported a significant correlation between Borden type and clinical presentation (30). The progression of disease severity with lesion type was tracked in Cognard's cohort of 205 patients. 27 patients had a type IIa fistula. Thirty-seven percent patients in there series had aggressive clinical symptoms manifested as headache, papilloedema or visual disturbance. In the 10 patients with type IIb DAVFs, 3 (30%) followed an aggressive course. 12 of 18 (67%) patients with type II a+b disease showed aggressive symptoms,

including an intracranial hemorrhage in 1 case. 19 (76%) of 25 patients with type III disease had aggressive symptoms, including 10 (40%) presenting with hemorrhage.

Twenty-nine patients had type IV DAVFs with direct venous drainage into a cortical vein with ectasia. Of these, 28 (97%) had aggressive symptoms; of which 19 (66%) had hemorrhage. Six of these patients had symptoms attributable to mass effect from the ectatic vein. The presence of direct cortical venous drainage was therefore a strong predictor of hemorrhage. Of those 12 patients with spinal perimedullary venous drainage, 6 presented with myelopathy.

The majority of cavernous sinus dural fistulas are idiopathic. These idiopathic dural fistulas are usually low-flow. Various implicated conditions for their development include pregnancy, sinusitis, trauma, surgical procedures, and cavernous sinus thrombosis (5, 31). They tend to appear in middle-aged women, and generally present with insidiously progressive glaucoma, proptosis, or a “red eye” – signs and symptoms that are usually less severe than those seen in direct fistulas (31).

Direct cavernous dural fistula rarely resolves spontaneously and requires immediate treatment if there is progressive visual loss, an intolerable bruit, headache, corneal exposure, severe retro-orbital pain, or cortical venous drainage (32). On the contrary, the spontaneous cavernous dural fistula has a tendency to resolve without

treatment. Still a sizeable number of patients with spontaneous cavernous dural fistula will suffer progressive loss of vision, diplopia, or intractable glaucoma and hence warrant interventional therapy (31).

IMAGING

Imaging by CT, CT angiography (CTA), MRI, MRA and catheter angiography play an important role in the investigation of patients with DAVFs. Due to varying nonspecific clinical and imaging features the diagnosis of a DAVF can be delayed or missed. Occasionally a plain film may be useful as it can show grooving within the skull vault due to chronic venous hypertension and compression by middle meningeal vessels.

Before any intervention, a nonenhanced CT must be done to rule out intracranial hemorrhage. Chronic venous congestion produces an area of low density on CT due to edema. Multidetector CTA, because of its rapid acquisition, can now provide high-resolution detail of vascular anatomy and has a temporal advantage over static CT. DAVFs can present as tinnitus and CT scan may help in detecting ear abnormalities such as aberrant vascular anatomy or glomus tumors. Linear bony defects formed by enlarged emissary veins can indicate the presence of a fistula (32). MRI in DAVFs shows dilated cortical veins without a parenchymal nidus. It can also provide clues about DAVF in the presence of veno-occlusive disease. Other MRI imaging appearances include thickened dural leaflet; hypertrophied pachymeningeal arteries; dilated, tortuous, and variceal

venous channels; and a thrombosed or stenosed dural venous sinus. The arterial supply to the DAVF is however not demonstrated on MR images.

PET-based regional cerebral blood flow (rCBF) studies indicate that decreased rCBF may be important in the pathogenesis of the neurological symptoms. In patients with normal cortical venous drainage, values for rCBF, regional cerebral metabolic rate of oxygen (rCMRO), and regional oxygen extraction fraction (rOEF) were normal. Reduced rCBF and mildly or markedly increased rOEF is seen in patients with clinical symptoms and cortical venous drainage (19).

DIGITAL SUBTRACTION ANGIOGRAPHY

Angiography remains the gold standard for diagnosis and planning of therapy. In addition to diagnosis, anatomical and hemodynamic assessment of the fistula needs to be performed. The aim is not only to identify the arterial feeders and the site of the fistula but also to identify the pattern and direction of venous drainage of the fistula and normal cerebral parenchyma. Selective injection of all potential arteries that may contribute to the region of dural pathology needs to be done.

A dual arterial injection may be required to demonstrate the feeders and shunts in some cases. This dual arterial injection is called as competitive angiogram, which is selective injection of ICA followed by ECA. With road mapping the drainage of the fistula and normal brain parenchyma can be assessed. The angiogram should be analyzed

for feeders, type of venous drainage, retrograde flow, occlusion of sinuses, and circulation time. The normal appearance of vein after arterial injection occurs after 6 to 7 seconds. The acquisition of images should begin in the early arterial phase and continue into the late venous phase of the study. The angiogram should be analyzed for feeders, type of venous drainage, retrograde flow, occlusion of sinuses, and circulation time. The common angiographic findings seen are feeders from pial and dural branches, AV shunting, venous ectasia, stenosis, calcifications, impaired drainage of cerebral parenchyma with delayed venous drainage, and cortical venous collaterals. Prior to intervention, the venous phase of the cortical veins that drain the DAVFs and rerouting of drainage to adjacent territories, demonstrating a pseudophlebitic pattern (33). Aneurysms can be associated with DAVFs and may be on the feeding vessels, either extradural or subarachnoid in location. (13)

CORTICAL REROUTING

Aggressive symptoms are shown by those DAVFs, which drain retrograde by leptomeningeal cortical venous channels. Houser *et al*, (3) point out that the sinus need not even be thrombosed to cause retrograde venous drainage into cortical veins. Partial filling of sinuses by the high-pressure arterial blood can impede ante grade venous drainage. Incidence of hemorrhage has been reported between 35% and 42% (4, 30). A high frequency of hemorrhage is reported in DAVFs involving the anterior cranial fossa and tentorium. These DAVFs almost always drain into intradural veins (34). Apart from hemorrhage, retrograde venous flow accompanied by increased venous pressure can lead to chronic passive congestion and venous infarcts, which are a major cause of focal

neurologic deficits in DAVFs. Hydrocephalus is another complication of DAVF. It is due to decreased absorption of cerebrospinal fluid due to increased venous pressure or mechanical compression of the aqueduct by mesencephalic veins.

AGGRESSIVE FACTORS

Willinski et al has suggested aggressive course will be seen in 75% of anterior cranial fossa lesion, 79% of tentorial lesions, 60% of foramen magnum lesions and 29% of transverse sinus lesions (30). The incidence corresponds to that of leptomeningeal retrograde venous drainage at each of these locations. Awad et al pointed out the aggressive factors as leptomeningeal cortical venous drainage, galenic deep venous drainage, variceal or aneurismal dilatations and lesions located at the tentorial incisura.

TREATMENT

General treatment approaches for the treatment of DAVFs include the following:

1. Conservative treatment
2. Neuroradiological endovascular intervention, which comprises of:
 - i) Particulate embolization
 - ii) Glue or onyx injection through arterial or venous route
 - iii) Coiling with or without glue or onyx injection
 - iv) Venous stenting
3. Radiation therapy

4. Surgery
5. Combination of above, i.e., RT + intervention or intervention + surgery

1. CONSERVATIVE TREATMENT & Carotid Compression

Conservative treatment involves mainly symptomatic treatment and supportive measures. Spontaneous regression of dural DAVFs has been reported (11). Low flow type IIa and I dural fistulas are rarely life threatening. They being low flow arteriovenous shunts may thrombose spontaneously in time (28).

Because of the high rate of spontaneous regression between 10 to 60% according to literature, in most cases, a "wait and see" strategy may be appropriate. This spontaneous cure can also be observed in bilateral cavernous dural fistulas, as described by Voigt et al (29).

Seeger et al believed that a spontaneous cure of dural cavernous fistula in most cases is due to partial or complete thrombosis of the cavernous sinus and/or its tributaries (35). The indications for treatment of DAVFs that drain directly into a dural venous sinus with normal ante grade flow in the sinus is limited (13). The natural history of this type of DAVFs is now known to be benign and the chances of stability or spontaneous symptom resolution are high.

The persistence of the physiological venous flow from the cavernous sinus to the petrosal sinuses or a thrombosis upstream to the dural fistula may be factors favoring

spontaneous occlusion (36). A high frequency (85%) of thrombosis of both the ipsilateral inferior and superior petrosal sinuses associated with cavernous dural fistulas has been noted. However, whether petrosal sinus thrombosis is a cause or a consequence of the cavernous dural fistulas is as yet unknown (36). With manual jugular and carotid compression, spontaneous fistulas may have up to a 30% rate of resolution (16).

Once the fistula is diagnosed, an angiogram needs to be done. After evaluation of the findings, further treatment can be planned. Various authors have described their experience in treatment of these fistulas.

Vinuela et al described the management of 20 spontaneous CCFs. Seventeen were unilateral & three bilateral, these dural fistulas were treated with PVA and IBCA. Two patients underwent post embolization surgical procedures, 8 patients received no therapy. In two, spontaneous obliteration of the fistula occurred. Of the 9 cavernous AVM embolized with particles or IBCA, complete embolization was achieved in 7 cases and partial embolization followed by surgery in 2 cases. In one patient right hemiplegia with aphasia resulted from reflux of IBCA emboli through the artery of foramen rotundum to the MCA (28).

However, the pitfalls of the transvenous approach include converting the sinus into an aneurysm by blocking all venous drainage without occluding the fistula. Occluding the

posterior venous drainage results in diversion of flow into the ophthalmic veins and worsening of vision loss or ocular symptoms. Similarly, occluding the anterior drainage may increase cortical or petrosal venous drainage and cause cerebral or brainstem ischemia.

2. ENDOVASCULAR NEURORADIOLOGICAL INTERVENTION

TRANSARTERIAL ROUTE

i. PARTICULATE EMBOLIZATION: Embolization of external carotid branches with particles are easily performed and can reduce shunt flow. Transarterial particulate embolization typically is not curative. Total obliteration is difficult to achieve with this method because some feeding arteries cannot be catheterized and because of the recruitment of blood supply from collateral arteries (37). Therefore, it is generally used to relieve symptoms or in combination with other procedures such as irradiation, surgery, or transvenous embolization (TVE).

ii. n-Butyl-2Cyanoacrylate or ONYX EMBOLIZATION: Deposition of glue into the collecting vein or fistula through the transarterial route may also result in cure, but it is less controllable than coil embolization and thus may present a higher risk. Transarterial embolization (TAE) with n-Butyl Cyanacrylate is regularly used to treat complex DAVFs if percutaneous transvenous catheterization is not possible (38).

The development of the permanent liquid agent Onyx for embolization of AVMs drastically changed the management strategies of AVMs. Onyx can also be used to treat dural fistulas. Using a stent placed across the orifice of the fistula may prove safe to

occlude the feeders with this novel agent.

Onyx is a new nonadhesive liquid embolic agent. It is supplied in ready-to-use vials and is a mixture of EVOH (Ethyl vinyl alcohol copolymer), DMSO (Dimethyl sulfoxide), and Tantalum. Onyx, after coming in contact with blood, gets precipitated and occludes the vessels but is nonadherent to the vessel wall. This non-adhesive property of onyx eliminates the risk of gluing of the microcatheter to the vessels. This also allows a slow and prolonged single injection of the embolic agent with concomitant check angiograms. Good positioning of the microcatheter and slow injection can completely fill the fistula and enable a complete cure.

Currently onyx is available in two concentrations (FDA-approved) for presurgical cerebral AVM embolization (39). Depending on the need, onyx or glue in mild to moderate concentration (20, 25, or 33%) may be injected.

iii. COILING VIA TRANSVENOUS ROUTE: Coils are now regularly used for curative purposes. Many studies have reported their usefulness. Complete occlusion may occur in 80-100% of cases (40). An angiographically nonvisualized inferior petrosal sinus (IPS) can be successfully catheterized in 30-50% of patients (23). When this is thrombosed, the transvenous access may be tried out via the superior petrosal sinus (SPS) facial vein, angular vein, pterygoid sinus, and superior ophthalmic vein (SOV) (41). Inadequate embolization leads to a worsening of symptoms.

In some of these cases, occlusion of venous draining pathways may be attempted. However, the problem in occluding the draining venous pathway is the high compliance of the vein or sinus, which can allow its dilatation, and subsequent migration of emboli. The SOV dilates anteriorly thereby increasing the risk of migration of emboli placed posteriorly in this structure (42).

The placement of coils into the CS (intraoperatively) to produce the closure of a cavernous fistula was first reported by Hosobuchi (43). The use of detachable coils before onyx or glue injection slows and decreases flow in the fistula and provides secure anchoring to the onyx or glue cast.

The traditional approach through the transvenous route to the CS is via the IPS. The IPS route has certain advantages - a) It is less time consuming especially when it is draining the fistula. b)-The anatomical route is straight and does not cause significant friction inside the catheter. c) - It allows the stable positioning of the guiding catheter inside the IJV, which facilitates catheter navigation to the ipsilateral or even contralateral cavernous sinus. Even if thrombosed, it can be used as a route for embolization (44).

In Benndorf cases the angiogram did not show opacification of the IPS, indicating that it neither drained the arteriovenous fistula nor the cerebral venous outflow (44). The authors state that it should be clearly distinguished if non-opacification of the IPS occurs

during the early arterial phase (draining the fistula) or in the late venous (draining the brain) or in both phases. Cases belonging to the second group (not draining the fistula but draining the brain) have a better chance of successful catheterization (44).

If the IPS does not serve as a venous outflow at all, then it is highly likely that this sinus has undergone subtotal or complete thrombosis. However this does not preclude the existence of anatomic connections and therefore the possibility of reaching the fistula site with a suitable microcatheter. A large volume jugular bulb phlebogram may be useful in visualizing a tiny vascular structure as a remnant of the thrombosed IPS that can then be successfully catheterized. The authors also state that in 31% of cases, the IPS cannot be catheterized due to its plexiform architecture and lack of connections with the IJV. Nevertheless it is not possible to predict if a catheter can successfully be navigated through the IPS in a given case (44).

Halbach first described 2 cases in which the IPS was not demonstrated by arteriography but successful catheter navigation through the non-opacified IPS was possible (5). Yamashita and colleagues were able to catheterize the IPS in 2 patients whose sinuses were not demonstrated in angiography (45).

Apart from the IPS route, the SOV route is also well established. Derang et al (46) described the treatment of cavernous dural fistulas via the retrograde SOV route. According to them indications for embolising cavernous dural fistulas though the

SOV are -

1. The SOV is the main draining vein, obviously dilated & arterialized. Symptom duration is over 3 months (47, 48).
- 2 Direct CCF, which is difficult, dangerous, and frustrating to embolise via the arterial route, and is prone to recur after embolization via arterial route (49).
- 3 Cavernous dural fistulas that are not spontaneously cured after six months.

In cases of spontaneous cavernous dural fistulas, the indications for urgent treatment include visual decline, which can progress rapidly, and if it is severe (to the point of light perception only) it is often irreversible, even with prompt closure of the fistula. Any patient with angiographic findings of a cavernous sinus varix should have emergent treatment to eliminate the risk of subarachnoid hemorrhage. Patients with angiographic evidence of a pseudoaneurysm have a grave prognosis and should also receive immediate treatment. Differentiating the two, the onset of the pseudoaneurysm is usually coincidental with the trauma, as opposed to the delayed onset of a varix, which develops after the occlusion of venous outflow pathways (50).

DAVFs of the superior sagittal sinus are technically challenging to treat definitively from the arterial side because of the distal location of the nidus with respect to the achievable position of the tip of the microcatheter in the external carotid artery branches. Sacrifice of the superior sagittal sinus is usually not a good choice unless

limited to the anterior third of the sinus (13). To attain a cure for reterograde leptomeningeal or cortical reflux, the venous approach is most likely to succeed, unless access to the sinus is restricted because of coexisting thrombosis.

Spontaneous cavernous dural fistulas have been reported to close with carotid – jugular compression. In the author's series, combined carotid and jugular compression resulted in complete cure in 7/23 patients and improvement in one additional patient. There were no complications in these patients. Patients in whom carotid –jugular compression failed or who demonstrated CVR, or visual decline, were treated with endovascular embolization (50).

Once clinical cure of the symptoms has occurred, suggesting closure of the fistula, a check angiogram need not be done. The authors only performed follow-up angiography if patients had persistent symptoms or new symptoms, or if the control angiogram obtained after embolization demonstrated subtotal occlusion (50).

The transvenous route has few concerns. Although arterializations of venous structures can occur in long-standing shunts, the draining veins and dural sinuses are still thin-walled and can be perforated by catheter and guide wire manipulations. With increased pressure and flow of the arterialized blood in these structures, small perforations can result in rapidly fatal hemorrhage. Hence, caution needs to be exercised

when approaching via the venous route. He stated that if the posterior drainage is closed, a contralateral inferior petrosal sinus or superior ophthalmic vein approach is used. If the superior ophthalmic approach fails and the vein is occluded, fistula flow may be diverted into cortical veins, increasing the risk of hemorrhagic complications (51).

At times, there might be no transarterial or transvenous access. In such cases, a percutaneous puncture of the SOV can be performed deep in the orbit, permitting venous occlusion without complications (46). The advantage of the SOV route is that it preserves the arterial supply, while taking less time. The embolization is done only in the venous system, reducing the opportunity for occlusion and vasospasm of ipsilateral arteries (46).

In a retrospective analysis of 27 patients with dural cavernous fistulas by Theaudin, twenty patients received endovascular treatment via a transvenous route (n=16) or via a transarterial route (n= 4). Complete occlusion of the fistula was obtained in 14/16 patients treated by transvenous approach and in 1 out of four patients treated via transarterial approach. Seven patients did not receive any endovascular treatment. Three had spontaneous obliteration and 4 had minor clinical symptoms and no leptomeningeal venous drainage. 16 pts had early clinical improvement after endovascular treatment. One had a cerebral hemorrhage after transvenous embolization of DCCF with leptomeningeal drainage. On follow up, all patients treated by the transarterial route remained symptomatic while 10/14 patients treated via transvenous route was asymptomatic (36).

As regards the extent occlusion of a dural fistula and cortical venous rerouting, subtotal occlusion is often sufficient for clinical improvement and subsequent angiographic and clinical cure is common (50).

Because of the dangers inherent to vessel manipulation and puncture in these patients, it is best to use noninvasive imaging methods whenever possible, such as CT and MR angiography, and to decide upon the need for treatment based upon the severity of a patient's symptoms.

iv. **STENT PLACEMENT**: Some authors have tried to close the shunts in the DAVFs with the help of stents (52). Significant advances in stent and catheter design now make it possible in many instances to deploy either balloon-expandable or self-expanding stents in the narrowed portions of the sinuses. With these devices, it is possible to reconstruct a severely stenosed sinus. Kieran et al have described the use of stents to treat dural fistulas and obtain closure of the feeders (53).

Kieran et al, have postulated that it is possible to recanalize a chronically occluded dural venous sinus and complete closure of multiple DAVF feeders can be achieved by the deployment of stents (53). Long-term results from the use of stents in the treatment of DAVFs are not yet known. Ante grade sinus flow can be established with the help of the radial force in the stent, which can close the shunts in the sinus wall (52).

3. RADIATION THERAPY

Good results have been reported from the use of stereotactic radiotherapy for the treatment of DAVFs, with complete occlusion being reported in 44-87% of cases (54, 55). This method, although reported to be effective in several case reports or small series, carries an unacceptable delay of 1-3 years in the cure of DAVFs with cortical venous reflux (CVR) and therefore is not recommended as a primary therapeutic measure for these lesions (56). The advantages of radiotherapy include decreased invasiveness of the procedure and fewer short-term complications; the major disadvantage is the latency of 6-12 months after irradiation before there are tangible benefits. The combined use of stereotactic radiotherapy and TAE with particles is in practice and it enhances the effectiveness of both the techniques. Combined treatment gives good results and reduces the risk of worsening symptoms during the follow-up period (57, 58).

4. SURGERY

Endovascular management has become a first-line treatment for dural DAVFs. However, some DAVFs of the anterior cranial fossa can often be treated more easily and safely with surgical disconnection of the venous drainage (57). Combination with endovascular procedures is required in difficult cases where sinus isolation and resection are needed.

The authors described surgical approach for cases in which transarterial and transvenous route both failed. A standard pterional craniotomy with a Dolenc approach is sufficient to unlock the cavernous sinus and provide access to the fistula (59).

MATERIALS AND METHODS

MATERIALS AND METHODS

The design of this study carried out in the department of IS & IR, at SCTIMST, Trivandrum was retrospective as well as prospective. For the purpose of this study, patients who had undergone endovascular management for DAVF in the department of IS & IR, SCTIMST, dating from January 1996 to 30th August 2008. There were 42 cases in the retrospective group and 31 patients in the prospective one.

The clinical data of the patients was obtained by reviewing their case sheets obtained from the Medical Records Department & the imaging data was obtained from the DSA lab archive. From these, the data regarding clinical presentation, angiographic characteristics, details of the embolization procedure, post procedural status and the

follow up was collected. The post procedure follow-ups were carried out at 1 month, 6 months, 1 year and yearly thereafter. For the purpose of this study, the patients were also followed up with questionnaires.

A total of 73 cases of DAVF were treated. All patients underwent a complete neurological evaluation prior to the procedure. The patients had a baseline imaging done – either a CT or MRI of the brain. Subsequently, the patients underwent a complete six vessel diagnostic cerebral angiogram. All the studies were performed on Advantx digital subtraction angiography unit by (GE Milwaukee, USA). A wide variety of catheters and embolic material was used for the procedures as was best suited for the individual patient depending on the angioarchitecture.

The procedure was done under local (ECA feeders) or general anesthesia (ICA or VA feeders). Premedication (Inj. Pethidine 50mg i.m & Inj. Phenergan 25mg i.m for adults , and as per body weight for children) was give before the local procedures. Post procedure these patients were managed in the wards. For procedures carried out under general anesthesia, the patient was monitored in the neurological intensive care unit.

After discharge, the patients were followed up with clinical evaluation for improvement in their symptoms. Follow up angiograms were performed in majority of the patients.

PATIENT EVALUATION

A complete evaluation of the patient evaluated prospectively was carried out as per the proforma attached.

DIGITAL SUBTRACTION ANGIOGRAPHY

Angiography remains the gold standard for establishing the complete diagnosis. The angiographic work up should include bilateral vertebral, internal and external carotid artery injections. Cases where there has been prior treatment, particularly if it involved the sacrifice of a major feeder, the dural fistulas may recruit supply from one of the cervical arteries – either the ascending cervical arising from the thyrocervical trunk or the deep cervical arising from the costocervical trunk. This is because the dural fistulas may recruit supply from one of these arteries.

However, the angiographic evaluation should be tailored to each case depending on the findings at each step. In most cases, the initial angiographic work up should be sufficient to both establish a diagnosis and plan therapy.

MATERIAL USED FOR ANGIOGRAM AND EMBOLIZATION

1.	Sheath – Radiofocus, Cordis, Impulse	6, 7 French size
2.	Diagnostic catheters – Vertebral glide (Terumo), Right coronary (Cordis), Mani cerebral (Cordis), Simmons (Cordis)	5, 4 French size
3.	Guide wire – Terumo exchange length, Terumo standard,	150 cm – Standard 260 cms - Exchange
4.	Guiding catheters – Vistabrite, Launcher (Cordis)	6, 7 French size
5.	Microcatheters – Excelsion 1018, Tracker, Spinnaker 1.5, 1.8, Ultraflow, Marathon.	1.5 to 1.8 French
6.	Microguidewires – Transcend, Agility, Mirage.	.010, .014
7.	Embolic materials – Poly vinyl alcohol (150 to 1000 micron), N-Butyl Cyanacrylate, Gelfoam, Onyx, Coils – GDC, MTI	Coils varying size Glue varying percentage

Technique of embolisation:

The procedures were performed under local / general anesthesia. A guiding catheter was placed in the main feeding arteries (cervical ICA/ CCA in majority of the cases). In ECA feeders, guiding catheter was placed in distal CCA or proximal ECA and then feeders were selectively cannulated and embolized using poly vinyl alcohol (PVA) particles, ethanol or glue. In ICA feeders, guiding catheter was placed in vertical portion

of petrous ICA and then feeders were selectively cannulated with microcatheter, positioned near the fistula and were embolized with N-Butyl cyanacrylate or ONYX. In VA feeders, guiding catheter was placed in V1 segment and then feeders were cannulated and embolized using NBCA or ONYX. In some cases, the fistula was accessed via the venous route. A femoral vein puncture was followed by placement of the guiding catheter into the main venous trunk – internal jugular vein (IJV) for inferior petrosal sinus (IPS) approach and facial vein for superior ophthalmic vein (SOV) approach. The microcatheter was then negotiated into the sinus and coils were placed to occlude the fistula. The coils were released after confirming their position in the sac. In cases where the arterial or venous approach failed to achieve significant reduction of the fistula, either cut down of the superior ophthalmic vein (in cases of cavernous DAVFs) or burr hole (in case of torcula DAVF) was done to further obliterate the fistula. A complete check angiogram was performed after all embolization procedures.

Post procedural management:-

The patient was shifted to the ward or the neurological ICU for 24 hours after the procedure as required. Patient was given antiedema measures with dexona (6mg, TDS) and mannitol (75 ml TDS), (for 3 to 4 days) in cases with venous hypertension. ECA feeder embolization patients received analgesics (Tramazac 50 mg) for pain relief.

External compression

In patients having cavernous DAVFs without cortical venous drainage, ipsilateral carotid artery compression can result in cure in some patients. The patient is instructed to compress the carotid artery with the contralateral hand for a period of 10- 30 sec, several times per hour, in the sitting position. Patients are warned to release the compression immediately at the onset of any symptoms of cerebral ischemia, such as weakness, sensory changes, blindness, or altered mental status. The contralateral hand is used for this purpose so that if cerebral ischemia develops, that hand will become weak and will drop automatically, thereby releasing the compression. Maximal effect is achieved within 4-6 weeks. Patients with cortical venous drainage were excluded from compression therapy because combined carotid artery and jugular compression may increase cerebral venous pressure and could theoretically lead to venous hypertension, rupture.

ANALYSIS OF CASES

A total of 73 patients with DAVF were analyzed. DAVF were classified according to classification by Cognard et al. Of these, Type I fistula was noted in 3 patients (4%), type IIa in 28 (39%), type IIb in 7 (10%), type IIab in 19 (26%), type III in 6 (8%), type IV in 9 (12%) and type V in one patient (1%).

TYPE I

Type I DAVF was noted in 3 patients, all of them were males. The age range was from 30 years to 39 years (mean age 34.5 yrs). Location of the fistula was in the cavernous venous plexus in two patients (66.7%) and in the transverse sinus in one patient (33.3%).

TYPE IIa

Type IIa DAVF was noted in 28 patients of which 13 (46.4%) were males and 15 (53.6%) were females. The age range was from 20 years to 79 years (mean age 49.5

years). Location of the fistula was in the cavernous venous plexus in twenty-six patients (92.8%) and in the transverse sinus in two (7.1%).

TYPE IIb

Type IIb DAVF was noted in 7 patients of which 4 (57.1%) were males and 3 (42.9%) were females. The age range was from 20 years to 79 years (mean age 49.5 years). Location of the fistula was in the cavernous venous plexus in one patient (14.2%) and in the transverse sinus in six (85.8%).

TYPE IIab

Type IIab DAVF was noted in 19 patients of which 11 (57.8%) were males and 8 (42.2%) were females. The age range was from 20 years to 79 years (mean age 49.5 years). Location of the fistula was in the transverse sinus in 8 patients (42.1%), cavernous venous plexus in 5 (26.3%), torcula in 3 (15.7%), and sigmoid and straight sinuses in one patient (5.3%). One patient (5.3%) had fistula at two locations one in the transverse sinus and another in the superior sagittal sinus.

TYPE III

Type III DAVF was noted in 6 patients of whom 5 (83.3%) were males and 1 (16.7%) was a female. The age range was from 20 years to 49 years (mean age 34.5 years). Location of the fistula was in the transverse sinus in two patients (33.3%), superior sagittal sinus in two patients (33.3%), and one patient (16.7%) in the cavernous venous plexus. One patient (16.7%) had fistula at two locations one in the transverse sinus and another in the superior sagittal sinus.

TYPE IV

Type IV DAVF was noted in 9 patients of which 8 (88.9%) were males and one (11.1%) was a female. The age range was from 10 years to 59 years (mean age 34.5 years). Location of the fistula was in the transverse sinus in 4 patients (44.4%), torcula in 2 (22.2%), and cavernous venous plexus in 2 (22.2%) patients. One patient (11.1%) had fistula at the sigmoid sinus.

TYPE V

Type V DAVF was noted in one male patient. The patient was sixty-eight years old. Location of the fistula was in the transverse sinus with venous drainage into the perimedullary venous plexus of the cervical spinal cord.

RESULTS

TYPE & RESULT Crosstabulation

							Total
			1	2	3	4	1
TYPE 1	Count		3	0	0	0	3
	% within TYPE		100.0%	.0%	.0%	.0%	100.0%
2	Count		20	5	3	0	28
	% within TYPE		71.4%	17.9%	10.7%	.0%	100.0%
3	Count		7	0	0	0	7
	% within TYPE		100.0%	.0%	.0%	.0%	100.0%
4	Count		14	2	0	3	19
	% within TYPE		73.7%	10.5%	.0%	15.8%	100.0%
5	Count		3	2	1	0	6
	% within TYPE		50.0%	33.3%	16.7%	.0%	100.0%
6	Count		5	0	2	2	9
	% within TYPE		55.6%	.0%	22.2%	22.2%	100.0%
7	Count		0	0	0	1	1
	% within TYPE		.0%	.0%	.0%	100.0%	100.0%
Total	Count		52	9	6	6	73
	% within TYPE		71.2%	12.3%	8.2%	8.2%	100.0%

TYPE & FOLLOW UP CLINICAL Crosstabulation

			FOLLOW UP CLINICAL			Total
			1	2	3	1
TYPE 1	Count		3	0	0	3
	% within TYPE		100.0%	.0%	.0%	100.0%
2	Count		24	1	2	27
	% within TYPE		88.9%	3.7%	7.4%	100.0%
3	Count		7	0	0	7
	% within TYPE		100.0%	.0%	.0%	100.0%
4	Count		14	1	0	15
	% within TYPE		93.3%	6.7%	.0%	100.0%
5	Count		4	0	1	5
	% within TYPE		80.0%	.0%	20.0%	100.0%
6	Count		5	0	1	6
	% within TYPE		83.3%	.0%	16.7%	100.0%
Total	Count		57	2	4	63
	% within TYPE		90.5%	3.2%	6.3%	100.0%

TYPE & COMPLICATION Crosstabulation

			COMPLICATION		Total
			0	1	0
TYPE 1	Count		3	0	3
	% within TYPE		100.0%	.0%	100.0%
2	Count		24	4	28
	% within TYPE		85.7%	14.3%	100.0%
3	Count		7	0	7
	% within TYPE		100.0%	.0%	100.0%
4	Count		16	3	19
	% within TYPE		84.2%	15.8%	100.0%
5	Count		3	3	6
	% within TYPE		50.0%	50.0%	100.0%
6	Count		6	3	9
	% within TYPE		66.7%	33.3%	100.0%
7	Count		0	1	1
	% within TYPE		.0%	100.0%	100.0%
Total	Count		59	14	73
	% within TYPE		80.8%	19.2%	100.0%

TYPE & LOCATION Crosstabulation

		LOCATION							Total
		1	2	3	4	5	6	7	1
TYPE	1 Count	0	0	1	2	0	0	0	3
	% within TYPE	.0%	.0%	33.3%	66.7%	.0%	.0%	.0%	100.0%
	2 Count	0	0	2	0	0	26	0	28
	% within TYPE	.0%	.0%	7.1%	.0%	.0%	92.9%	.0%	100.0%
	3 Count	0	0	6	0	0	1	0	7
	% within TYPE	.0%	.0%	85.7%	.0%	.0%	14.3%	.0%	100.0%
	4 Count	0	3	8	1	1	5	1	19
	% within TYPE	.0%	15.8%	42.1%	5.3%	5.3%	26.3%	5.3%	100.0%
	5 Count	2	0	2	0	0	1	1	6
	% within TYPE	33.3%	.0%	33.3%	.0%	.0%	16.7%	16.7%	100.0%
	6 Count	0	2	4	1	0	2	0	9
	% within TYPE	.0%	22.2%	44.4%	11.1%	.0%	22.2%	.0%	100.0%
	7 Count	0	0	1	0	0	0	0	1
	% within TYPE	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	100.0%
Total		2	5	24	4	1	35	2	73
		2.7%	6.8%	32.9%	5.5%	1.4%	47.9%	2.7%	100.0%

TYPE & IMAGING Crosstabulation

			IMAGING									Total
			0	1	2	3	4	5	6	7	8	0
TYPE	1	Count	0	0	0	0	0	0	0	2	1	3
		% within TYPE	.0%	.0%	.0%	.0%	.0%	.0%	.0%	66.7%	33.3%	100.0%
	2	Count	2	1	0	0	25	0	0	0	0	28
		% within TYPE	7.1%	3.6%	.0%	.0%	89.3%	.0%	.0%	.0%	.0%	100.0%
	3	Count	1	1	0	0	1	0	0	1	3	7
		% within TYPE	14.3%	14.3%	.0%	.0%	14.3%	.0%	.0%	14.3%	42.9%	100.0%
	4	Count	2	0	1	2	5	2	2	1	4	19
		% within TYPE	10.5%	.0%	5.3%	10.5%	26.3%	10.5%	10.5%	5.3%	21.1%	100.0%
	5	Count	0	5	0	0	1	0	0	0	0	6
		% within TYPE	.0%	83.3%	.0%	.0%	16.7%	.0%	.0%	.0%	.0%	100.0%
	6	Count	0	6	1	0	1	0	0	1	0	9
		% within TYPE	.0%	66.7%	11.1%	0	11.1%	0	0	11.1%	.0%	100.0%
	7	Count	0	0	1	0	0	0	0	0	0	1
		% within TYPE	.0%	.0%	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
Total			5	5	3	3	34	4	3	7	9	73
			6.8%	6.8%	4.1%	4.1%	46.6%	5.5%	4.1%	9.6%	12.3%	100.0%

TYPE & HEADACHE Crosstabulation

			HEADACHE		Total
			0	1	0
TYPE	1	Count	1	2	3
		% within TYPE	33.3%	66.7%	100.0%
	2	Count	11	17	28
		% within TYPE	39.3%	60.7%	100.0%
	3	Count	2	5	7
		% within TYPE	28.6%	71.4%	100.0%
	4	Count	6	13	19
		% within TYPE	31.6%	68.4%	100.0%
	5	Count	2	4	6
		% within TYPE	33.3%	66.7%	100.0%
	6	Count	3	6	9
		% within TYPE	33.3%	66.7%	100.0%
	7	Count	0	1	1
		% within TYPE	.0%	100.0%	100.0%
Total		Count	25	48	73
		% within TYPE	34.2%	65.8%	100.0%

TYPE & CRANIAL NERVE PALSY Crosstabulation

			CRANIAL NERVE PALSY		Total
			0	1	0
TYPE	1	Count	3	0	3
		% within TYPE	100.0%	.0%	100.0%
	2	Count	5	23	28
		% within TYPE	17.9%	82.1%	100.0%
	3	Count	6	1	7
		% within TYPE	85.7%	14.3%	100.0%
	4	Count	10	9	19
		% within TYPE	52.6%	47.4%	100.0%
	5	Count	5	1	6
		% within TYPE	83.3%	16.7%	100.0%
	6	Count	5	4	9
		% within TYPE	55.6%	44.4%	100.0%
	7	Count	1	0	1
		% within TYPE	100.0%	.0%	100.0%
Total		Count	35	38	73
		% within TYPE	47.9%	52.1%	100.0%

TYPE & PAPILLOEDEMA Crosstabulation

			PAPILLOEDEMA		Total
			0	1	0
TYPE 1	Count		3	0	3
	% within TYPE		100.0%	.0%	100.0%
2	Count		18	10	28
	% within TYPE		64.3%	35.7%	100.0%
3	Count		5	2	7
	% within TYPE		71.4%	28.6%	100.0%
4	Count		10	9	19
	% within TYPE		52.6%	47.4%	100.0%
5	Count		2	4	6
	% within TYPE		33.3%	66.7%	100.0%
6	Count		6	3	9
	% within TYPE		66.7%	33.3%	100.0%
7	Count		0	1	1
	% within TYPE		.0%	100.0%	100.0%
Total	Count		44	29	73
	% within TYPE		60.3%	39.7%	100.0%

TYPE & BRUIT Crosstabulation

			BRUIT		Total
			0	1	0
TYPE 1	Count		2	1	3
	% within TYPE		66.7%	33.3%	100.0%
2	Count		23	5	28
	% within TYPE		82.1%	17.9%	100.0%
3	Count		6	1	7
	% within TYPE		85.7%	14.3%	100.0%
4	Count		11	8	19
	% within TYPE		57.9%	42.1%	100.0%
5	Count		5	1	6
	% within TYPE		83.3%	16.7%	100.0%
6	Count		8	1	9
	% within TYPE		88.9%	11.1%	100.0%
7	Count		1	0	1
	% within TYPE		100.0%	.0%	100.0%
Total	Count		56	17	73
	% within TYPE		76.7%	23.3%	100.0%

TYPE & DM Crosstabulation

			DM		Total
			0	1	0
TYPE 1	Count		3	0	3
	% within TYPE		100.0%	.0%	100.0%
2	Count		17	11	28
	% within TYPE		60.7%	39.3%	100.0%
3	Count		4	3	7
	% within TYPE		57.1%	42.9%	100.0%
4	Count		16	3	19
	% within TYPE		84.2%	15.8%	100.0%
5	Count		6	0	6
	% within TYPE		100.0%	.0%	100.0%
6	Count		8	1	9
	% within TYPE		88.9%	11.1%	100.0%
7	Count		1	0	1
	% within TYPE		100.0%	.0%	100.0%
Total	Count		55	18	73
	% within TYPE		75.3%	24.7%	100.0%

TYPE & HT Crosstabulation

			HT		Total
			0	1	0
TYPE 1	Count		3	0	3
	% within TYPE		100.0%	.0%	100.0%
2	Count		19	9	28
	% within TYPE		67.9%	32.1%	100.0%
3	Count		7	0	7
	% within TYPE		100.0%	.0%	100.0%
4	Count		16	3	19
	% within TYPE		84.2%	15.8%	100.0%
5	Count		6	0	6
	% within TYPE		100.0%	.0%	100.0%
6	Count		8	1	9
	% within TYPE		88.9%	11.1%	100.0%
7	Count		0	1	1
	% within TYPE		.0%	100.0%	100.0%
Total	Count		59	14	73
	% within TYPE		80.8%	19.2%	100.0%

ROUTE & COMPLICATION Crosstabulation

			COMPLICATION		Total
			0	1	0
ROUTE	0	Count	7	0	7
		% within ROUTE	100.0%	.0%	100.0%
	1	Count	18	10	28
		% within ROUTE	64.3%	35.7%	100.0%
	2	Count	11	1	12
		% within ROUTE	91.7%	8.3%	100.0%
	3	Count	0	1	1
		% within ROUTE	.0%	100.0%	100.0%
	5	Count	22	2	24
		% within ROUTE	91.7%	8.3%	100.0%
Total	6	Count	1	0	1
		% within ROUTE	100.0%	.0%	100.0%
		Count	59	14	73
		% within ROUTE	80.8%	19.2%	100.0%

ROUTE & RESULT Crosstabulation

			RESULT				Total
			1	2	3	4	1
ROUTE	0	Count	4	3	0	0	7
		% within ROUTE	57.1%	42.9%	.0%	.0%	100.0%
	1	Count	16	5	5	2	28
		% within ROUTE	57.1%	17.9%	17.9%	7.1%	100.0%
	2	Count	10	0	1	1	12
		% within ROUTE	83.3%	.0%	8.3%	8.3%	100.0%
	3	Count	0	0	0	1	1
		% within ROUTE	.0%	.0%	.0%	100.0%	100.0%
	5	Count	22	0	0	2	24
		% within ROUTE	91.7%	.0%	.0%	8.3%	100.0%
Total	6	Count	0	1	0	0	1
		% within ROUTE	.0%	100.0%	.0%	.0%	100.0%
		Count	52	9	6	6	73
		% within ROUTE	71.2%	12.3%	8.2%	8.2%	100.0%

EMBOLIC AGENT & COMPLICATION Crosstabulation

			COMPLICATION		Total
			0	1	0
EMBOLIC AGENT	1	Count	13	3	16
		% within EMBOLIC AGENT	81.3%	18.8%	100.0%
	2	Count	11	1	12
		% within EMBOLIC AGENT	91.7%	8.3%	100.0%
	3	Count	1	2	3
		% within EMBOLIC AGENT	33.3%	66.7%	100.0%
	4	Count	2	0	2
		% within EMBOLIC AGENT	100.0%	.0%	100.0%
	5	Count	0	3	3
		% within EMBOLIC AGENT	.0%	100.0%	100.0%
	6	Count	7	0	7
		% within EMBOLIC AGENT	100.0%	.0%	100.0%
	7	Count	24	2	26
		% within EMBOLIC AGENT	92.3%	7.7%	100.0%
Total		Count	58	11	69
		% within EMBOLIC AGENT	84.1%	15.9%	100.0%

EMBOLIC AGENT & RESULT Crosstabulation

			RESULT				Total
			1	2	3	4	1
EMBOLIC AGENT	1	Count	11	2	3	0	16
		% within EMBOLIC AGENT	68.8%	12.5%	18.8%	.0%	100.0%
	2	Count	10	0	1	1	12
		% within EMBOLIC AGENT	83.3%	.0%	8.3%	8.3%	100.0%
	3	Count	1	1	1	0	3
		% within EMBOLIC AGENT	33.3%	33.3%	33.3%	.0%	100.0%
	4	Count	2	0	0	0	2
		% within EMBOLIC AGENT	100.0%	.0%	.0%	.0%	100.0%
	5	Count	0	0	1	2	3
		% within EMBOLIC AGENT	.0%	.0%	33.3%	66.7%	100.0%
	6	Count	4	3	0	0	7
		% within EMBOLIC AGENT	57.1%	42.9%	.0%	.0%	100.0%
	7	Count	24	0	0	2	26
		% within EMBOLIC AGENT	92.3%	.0%	.0%	7.7%	100.0%
Total		Count	52	6	6	5	69
		% within EMBOLIC AGENT	75.4%	8.7%	8.7%	7.2%	100.0%

NUMBER OF EMBOLISATION & RESULT Crosstabulation

			RESULT				Total
			1	2	3	4	1
NUMBER OF EMBOLISATION	1	Count	37	8	6	4	55
		% within NUMBER OF EMBOLISATION	67.3%	14.5%	10.9%	7.3%	100.0%
	2	Count	10	1	0	0	11
		% within NUMBER OF EMBOLISATION	90.9%	9.1%	.0%	.0%	100.0%
	3	Count	5	0	0	1	6
		% within NUMBER OF EMBOLISATION	83.3%	.0%	.0%	16.7%	100.0%
	5	Count	0	0	0	1	1
		% within NUMBER OF EMBOLISATION	.0%	.0%	.0%	100.0%	100.0%
	Total	Count	52	9	6	6	73
		% within NUMBER OF EMBOLISATION	71.2%	12.3%	8.2%	8.2%	100.0%

LOCATION & CRANIAL NERVE PALSY Crosstabulation

			CRANIAL NERVE PALSY		Total
			0	1	0
LOCATION	1	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
	2	Count	4	1	5
		% within LOCATION	80.0%	20.0%	100.0%
	3	Count	18	6	24
		% within LOCATION	75.0%	25.0%	100.0%
	4	Count	3	1	4
		% within LOCATION	75.0%	25.0%	100.0%
	5	Count	1	0	1
		% within LOCATION	100.0%	.0%	100.0%
	6	Count	5	30	35
		% within LOCATION	14.3%	85.7%	100.0%
	7	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
Total		Count	35	38	73
		% within LOCATION	47.9%	52.1%	100.0%

LOCATION & COMPLICATION Crosstabulation

			COMPLICATION		Total
			0	1	0
LOCATION	1	Count	0	2	2
		% within LOCATION	.0%	100.0%	100.0%
	2	Count	3	2	5
		% within LOCATION	60.0%	40.0%	100.0%
	3	Count	20	4	24
		% within LOCATION	83.3%	16.7%	100.0%
	4	Count	3	1	4
		% within LOCATION	75.0%	25.0%	100.0%
	5	Count	1	0	1
		% within LOCATION	100.0%	.0%	100.0%
	6	Count	30	5	35
		% within LOCATION	85.7%	14.3%	100.0%
	7	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
Total		Count	59	14	73
		% within LOCATION	80.8%	19.2%	100.0%

LOCATION & RESULT Crosstabulation

			RESULT				Total
			1	2	3	4	1
LOCATION	1	Count	0	1	1	0	2
		% within LOCATION	.0%	50.0%	50.0%	.0%	100.0%
	2	Count	3	0	0	2	5
		% within LOCATION	60.0%	.0%	.0%	40.0%	100.0%
	3	Count	19	1	1	3	24
		% within LOCATION	79.2%	4.2%	4.2%	12.5%	100.0%
	4	Count	2	0	1	1	4
		% within LOCATION	50.0%	.0%	25.0%	25.0%	100.0%
	5	Count	1	0	0	0	1
		% within LOCATION	100.0%	.0%	.0%	.0%	100.0%
	6	Count	25	7	3	0	35
		% within LOCATION	71.4%	20.0%	8.6%	.0%	100.0%
	7	Count	2	0	0	0	2
		% within LOCATION	100.0%	.0%	.0%	.0%	100.0%
Total		Count	52	9	6	6	73
		% within LOCATION	71.2%	12.3%	8.2%	8.2%	100.0%

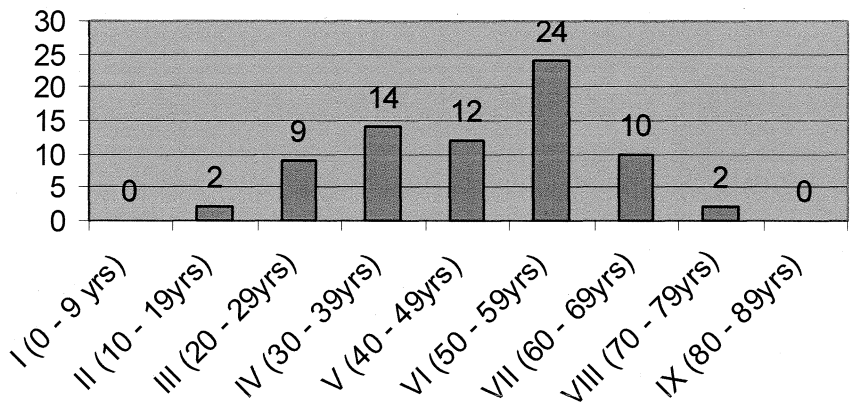
LOCATION & DM Crosstabulation

			DM		Total
			0	1	0
LOCATION	1	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
		% within DM	3.6%	.0%	2.7%
	2	Count	4	1	5
		% within LOCATION	80.0%	20.0%	100.0%
		% within DM	7.3%	5.6%	6.8%
	3	Count	19	5	24
		% within LOCATION	79.2%	20.8%	100.0%
		% within DM	34.5%	27.8%	32.9%
	4	Count	4	0	4
		% within LOCATION	100.0%	.0%	100.0%
		% within DM	7.3%	.0%	5.5%
	5	Count	1	0	1
		% within LOCATION	100.0%	.0%	100.0%
		% within DM	1.8%	.0%	1.4%
	6	Count	23	12	35
		% within LOCATION	65.7%	34.3%	100.0%
		% within DM	41.8%	66.7%	47.9%
	7	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
		% within DM	3.6%	.0%	2.7%
Total		Count	55	18	73
		% within LOCATION	75.3%	24.7%	100.0%
		% within DM	100.0%	100.0%	100.0%

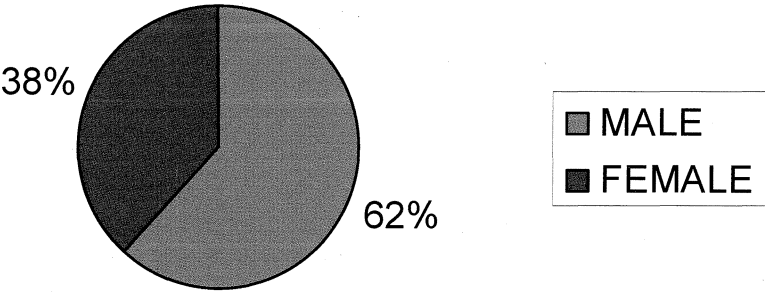
LOCATION & HT Crosstabulation

			HT		Total
			0	1	0
LOCATION	1	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
		% within HT	3.4%	.0%	2.7%
	2	Count	5	0	5
		% within LOCATION	100.0%	.0%	100.0%
		% within HT	8.5%	.0%	6.8%
	3	Count	18	6	24
		% within LOCATION	75.0%	25.0%	100.0%
		% within HT	30.5%	42.9%	32.9%
	4	Count	4	0	4
		% within LOCATION	100.0%	.0%	100.0%
		% within HT	6.8%	.0%	5.5%
	5	Count	1	0	1
		% within LOCATION	100.0%	.0%	100.0%
		% within HT	1.7%	.0%	1.4%
	6	Count	27	8	35
		% within LOCATION	77.1%	22.9%	100.0%
		% within HT	45.8%	57.1%	47.9%
	7	Count	2	0	2
		% within LOCATION	100.0%	.0%	100.0%
		% within HT	3.4%	.0%	2.7%
Total		Count	59	14	73
		% within LOCATION	80.8%	19.2%	100.0%
		% within HT	100.0%	100.0%	100.0%

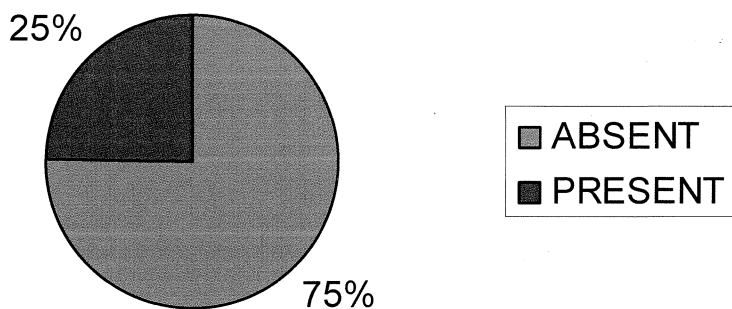
AGE DISTRIBUTION



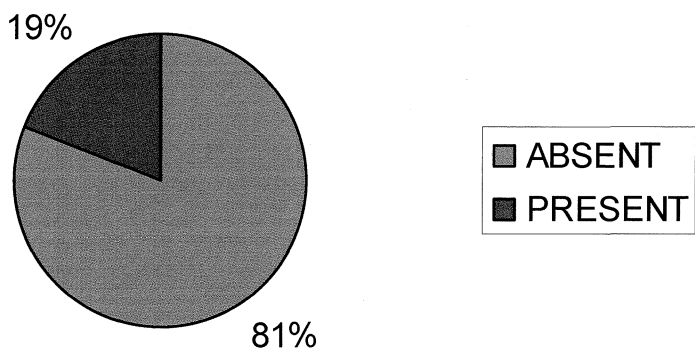
SEX DISTRIBUTION



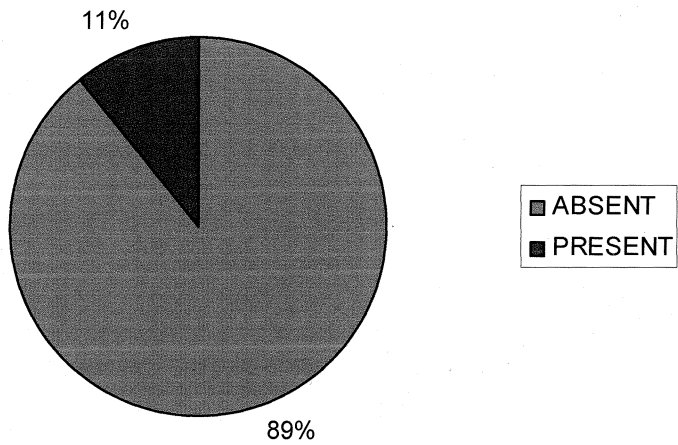
RISK FACTOR - DIABETES MELLITUS



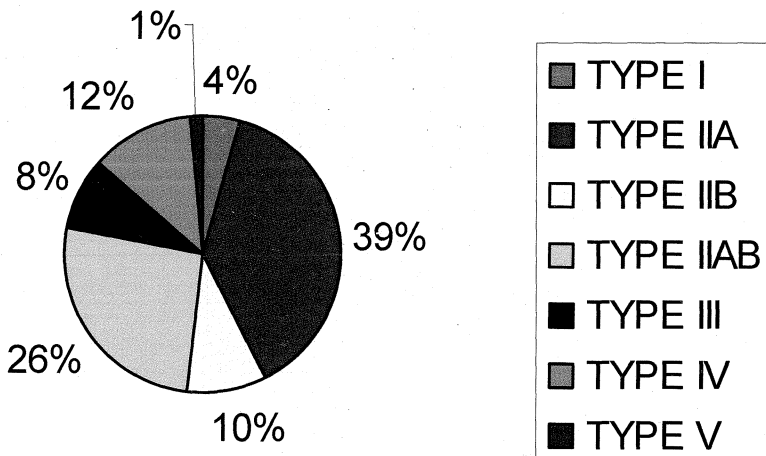
RISK FACTOR - HYPERTENSION



MISCELLANEOUS RISK FACTORS



TYPE OF DAVF (Cognard et al)



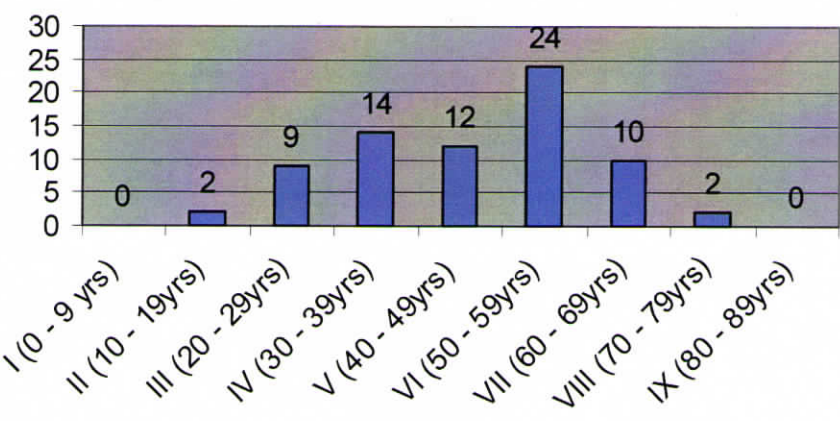
RESULTS

We analyzed 73 patients of DAVF. Of these, Type I fistula was noted in 3 patients (4%), type IIa in 28 (39%), type IIb in 7 (9.5%), type IIab in 19 (26%), type III in 6 (8%), type IV in 9 (12%) and type V in one patient (1%). The results of each type are presented separately.

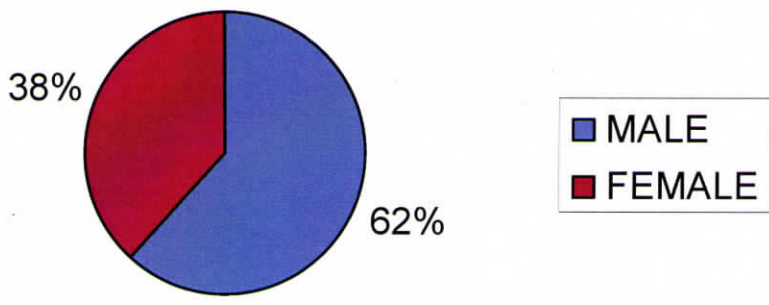
TYPE I

Type I DAVF was noted in 3 patients, all of them were males. The age range of patients was 30 - 39 years (mean age 34.5 yrs). Location of the fistula was in the

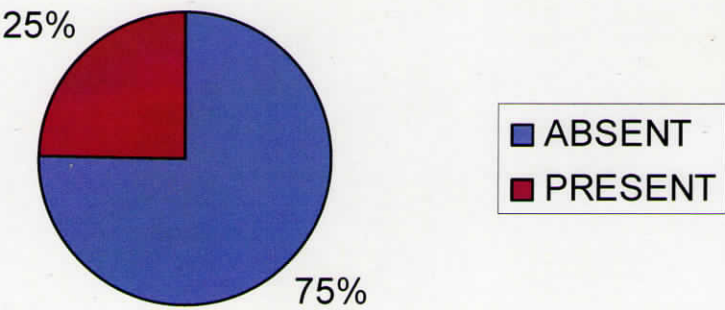
AGE DISTRIBUTION



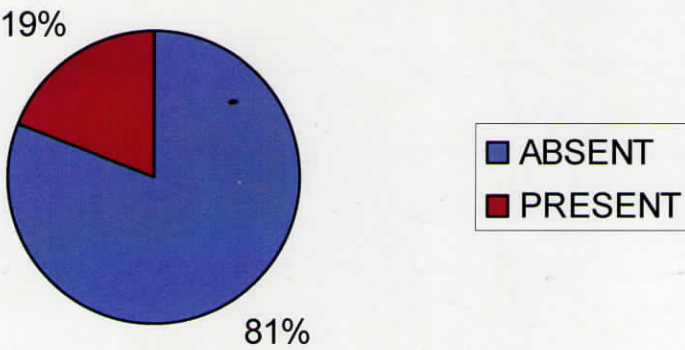
SEX DISTRIBUTION



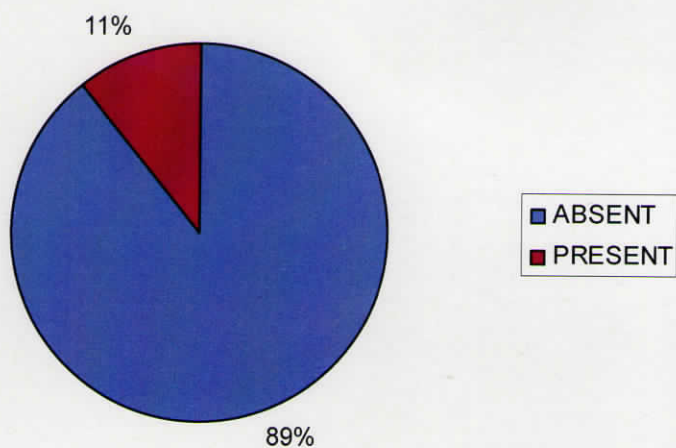
RISK FACTOR - DIABETES MELLITUS



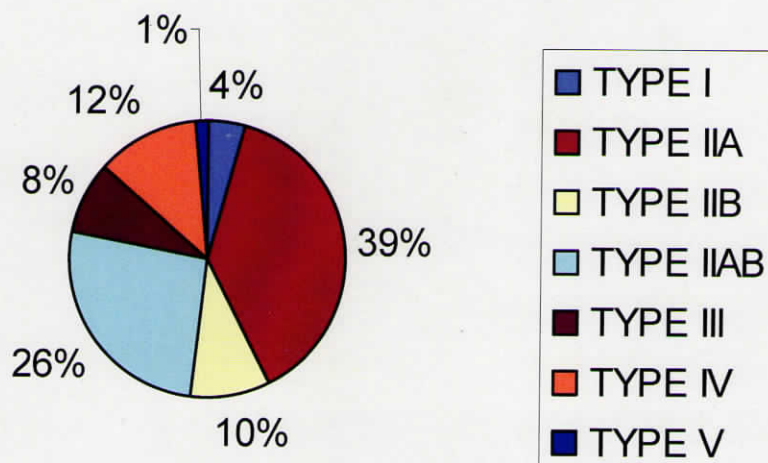
RISK FACTOR - HYPERTENSION



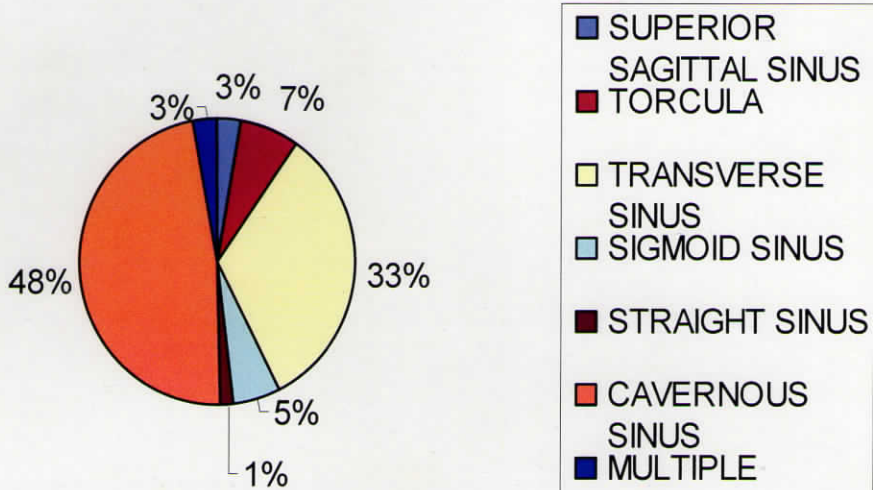
MISCELLANEOUS RISK FACTORS



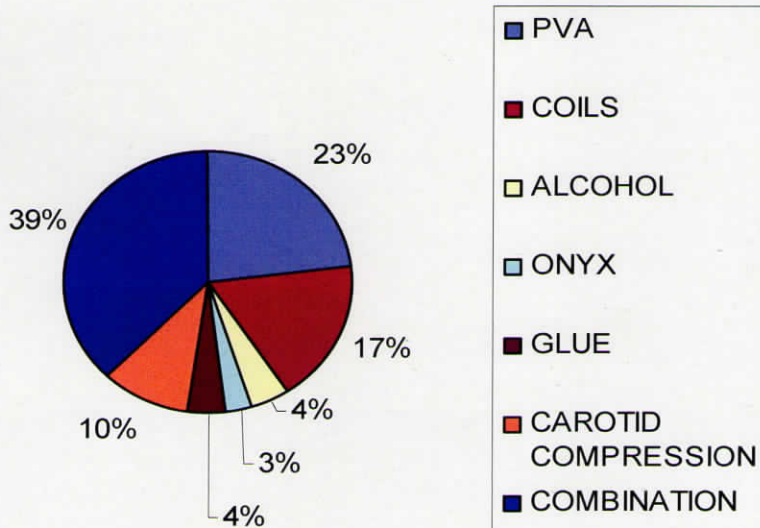
TYPE OF DAVF (Cognard et al)



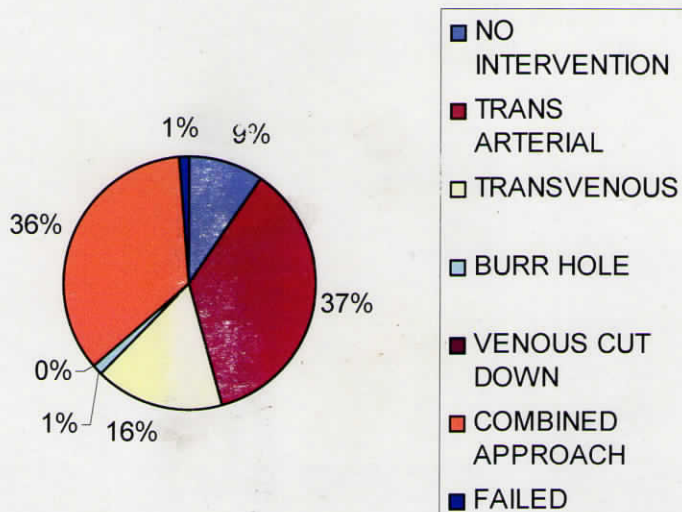
LOCATION OF FISTULA



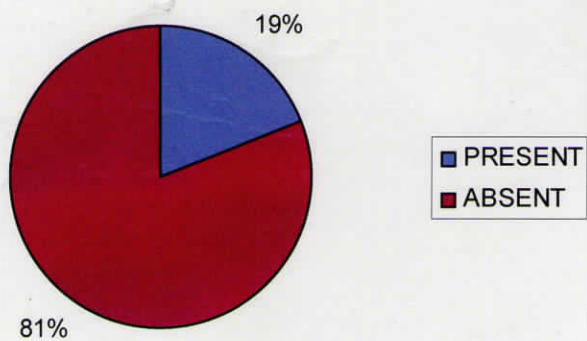
VARIOUS EMBOLIC MATERIALS



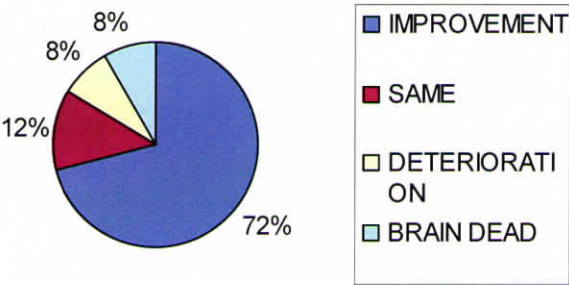
ROUTE OF EMBOLIZATION



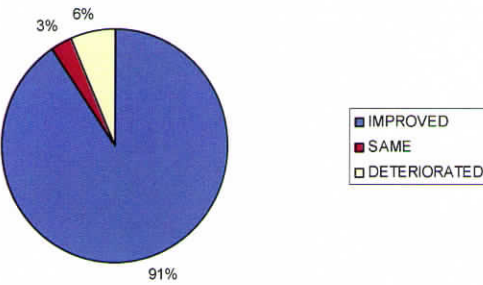
COMPLICATIONS



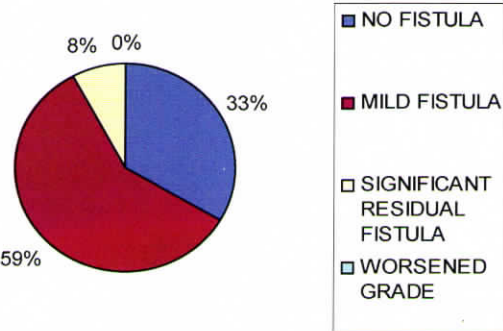
IMMEDIATE RESULTS



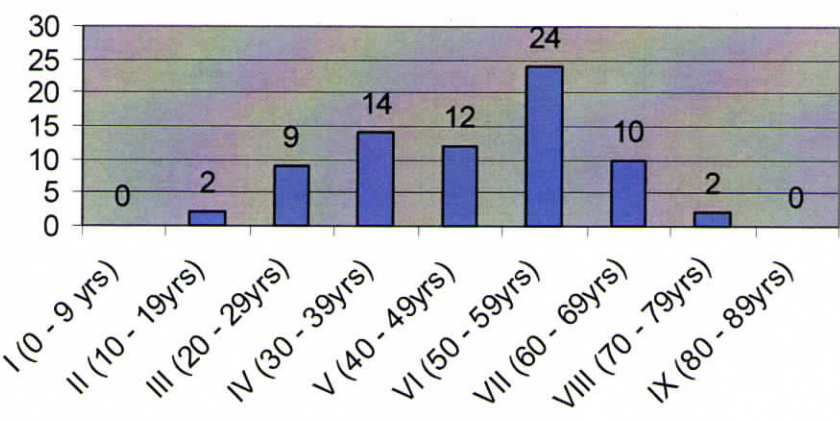
FOLLOW UP RESULTS - CLINICAL



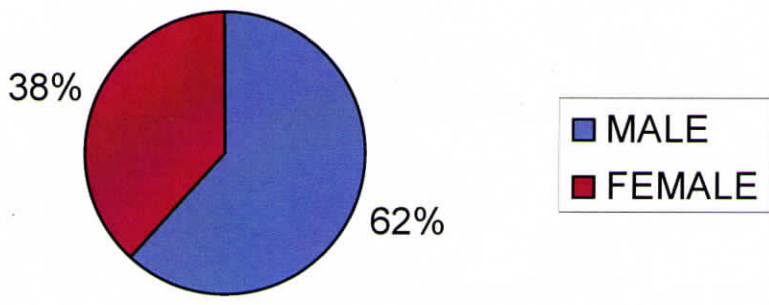
FOLLOW UP ANGIOGRAM - RESULTS



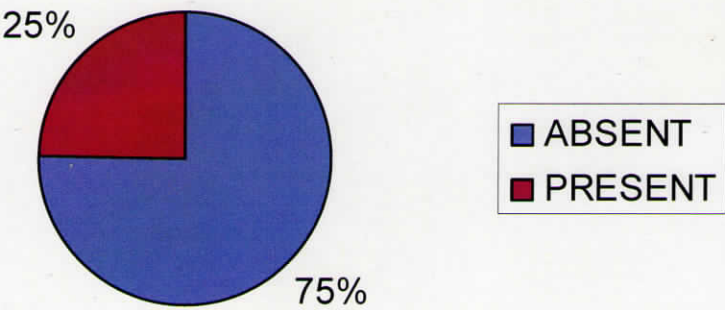
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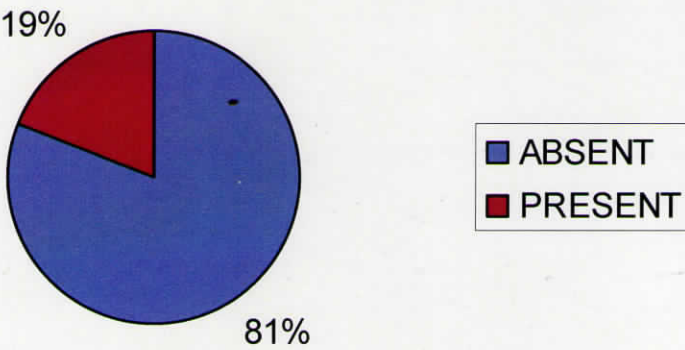
SEX DISTRIBUTION



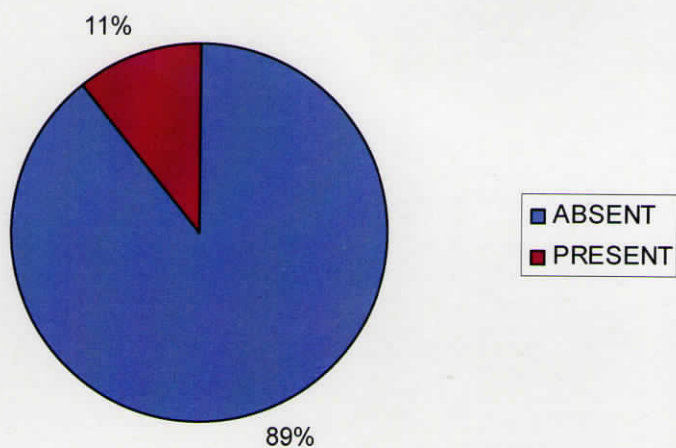
RISK FACTOR - DIABETES MELLITUS



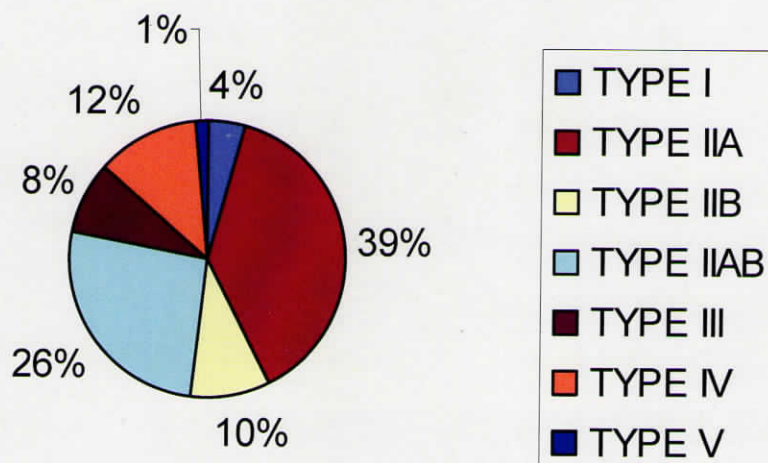
RISK FACTOR - HYPERTENSION



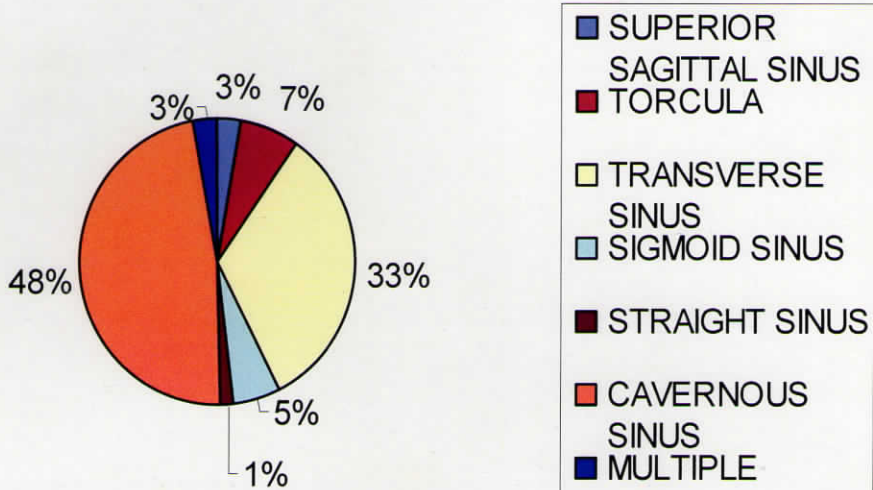
MISCELLANEOUS RISK FACTORS



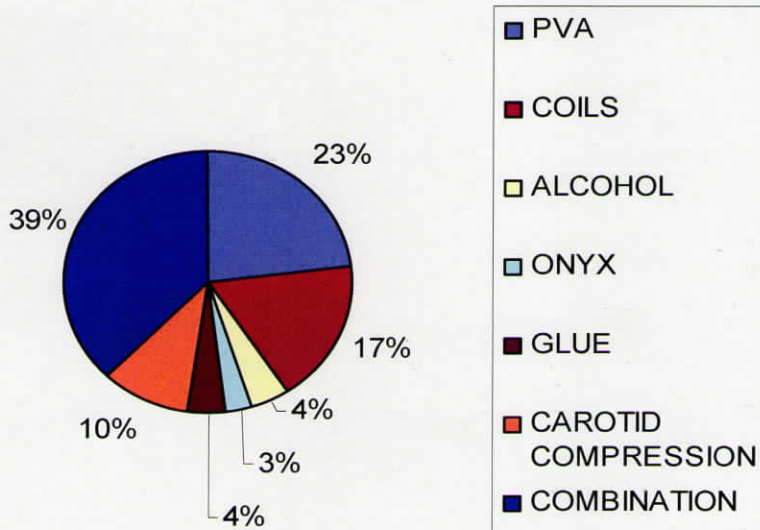
TYPE OF DAVF (Cognard et al)



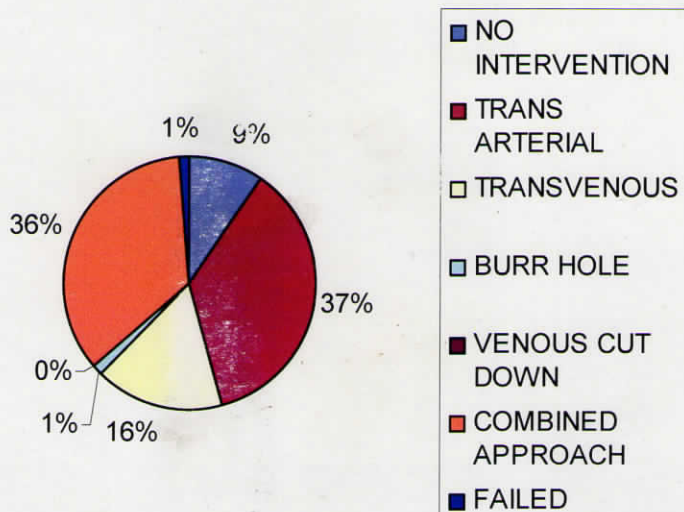
LOCATION OF FISTULA



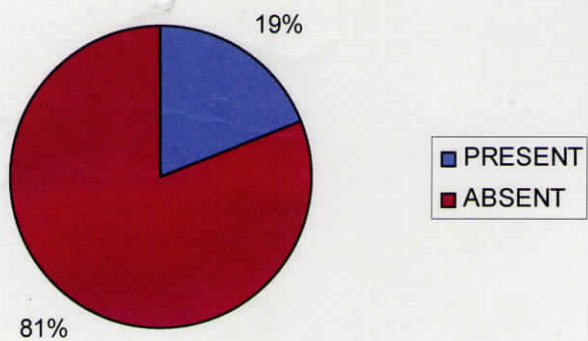
VARIOUS EMBOLIC MATERIALS



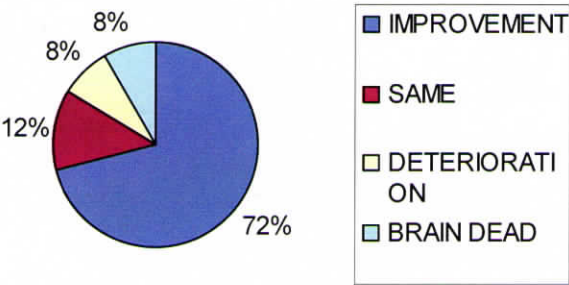
ROUTE OF EMBOLIZATION



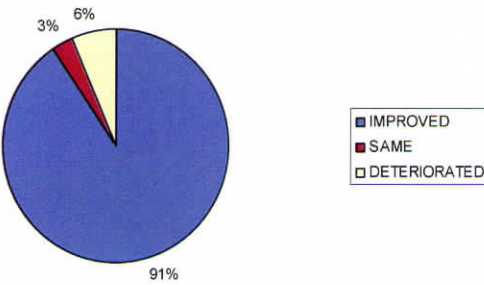
COMPLICATIONS



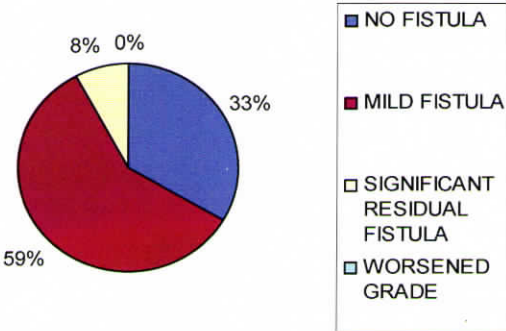
IMMEDIATE RESULTS



FOLLOW UP RESULTS - CLINICAL



FOLLOW UP ANGIOGRAM - RESULTS



cavernous venous plexus in two patients (66.7%) and in the transverse sinus in one patient (33.3%).

Of these three cases, two presented with headache, one with tinnitus and bruit. This group was managed conservatively and had significant (100%) improvement in symptoms. None of the patients in this group had visual symptoms or focal neurological deficit. No patient presented with hemorrhage on presentation or follow up. After angiography in these three patients, they were managed conservatively with carotid compression, clinical follow up and check angiogram. There was no worsening of symptoms. Twelve months follow up angiogram showed persistence of low flow fistula.

TYPE IIa

Type IIa DAVF was noted in 28 patients out of which 13 (46.4%) were males and 15 (53.6%) were females. The age range was from 20 - 79 years (mean age 49.5 years). Location of the fistula was in the cavernous venous plexus in 26 (92.8%) and in the transverse sinus in two patients (7.1%).

Of these 28 patients, 96% (26 patients) presented with diplopia, proptosis and visual field defect. Sixty one percent (17 patients) presented with headache, 89 % (25 patients) had chemosis, 36% (10 patients) had papilloedema and 82% (23 patients) had cranial nerve palsy on examination. Nearly 71% (20 patients) showed significant

improvement in symptoms on follow up. In three patients with cavernous DAVFs there was worsening of the vision with cortical venous rerouting inspite of PVA particle embolization of the external carotid feeders. Two patients showed reduction of the visual symptoms in the follow up period, with minimal persistence of the tiny dural feeders from ICA. One patient lost follow up. This group showed about 70% (19 patients) improvement in symptoms noted after second sitting of embolization.

TYPE IIb

Type IIb DAVF was noted in 7 patients of which 4 (57.1%) were males and 3 (42.9%) were females. The age range was from 20 - 79 years (mean age 49.5 years). Location of the fistula was in the cavernous venous plexus in 1 (14.2%) and in the transverse sinus in 6 patients (85.8%).

Only one patient (14%) in this group presented with subarachnoid hemorrhage. Three patients (43%) presented with narrowing of the transverse sinus and increased intracranial tension features. Two cases (28.5%) presented with focal neurological deficits. All the patients in this group showed complete resolution of the symptoms after the embolization. In this six patients (86%) was managed with combination of embolic materials. There were no complications noted following the procedure.

TYPE IIab

Type IIab DAVF was noted in 19 patients of which 11 (57.8%) were males and 8 (42.2%) were females. The age range was from 20 years to 79 years (mean age 49.5 years). Location of the fistula was in the transverse sinus in 8 patients (42.1%), cavernous venous plexus in 5 patients (26.3%), torcula in 3 patients (15.7%), and in the sigmoid and straight sinuses one patient (5.3%). One patient (5.3%) had fistula at two locations one in the transverse sinus and other in the superior sagittal sinus.

Fifty four percent (10 patients) presented with diplopia, proptosis and visual field defect, 69% (13 patients) with headache, 15% (3 patients) with seizures and focal neurological deficits, 27% (5 patients) with chemosis, 42% (8 patients) with papilloedema and 38% (7 patients) with cranial nerve palsy and bruit on examination. Seventy-three percent (13 patients) showed significant improvement in the symptoms. Of the two patients who failed to improve in the immediate post procedure period, one (5%) gradually improved on the follow up. Nearly 93% (18 patients) showed significant improvement in the symptoms on follow up. In one patient (5%) having transverse sinus DAVF, there was worsening of the cortical venous rerouting even after PVA & glue embolization of the external carotid feeders. Deterioration was noted because of progressive venous hypertension, waiting for next sitting of embolization. Three patients died because of intracerebral hemorrhage and mass effects. This occurred because of migration of glue into the venous side.

TYPE III

Type III DAVF was noted in 6 patients of which 5 (83.3%) were males and one (16.7%) was a female. The age range was from 20 years to 49 years (mean age 34.5 years). Location of the fistula was in the transverse sinus in two (33.3%), superior sagittal sinus in two (33.3%), and one patient (16.7%) in the cavernous venous plexus. One patient (16.7%) had fistula at two locations one in the transverse sinus and other in the superior sagittal sinus.

Of these 6 patients, 33% (2 patients) presented with diplopia, proptosis, visual field defect and tinnitus, 67% (4 patients) with headache and 16% (1 patient) with seizures and focal neurological deficits. None of the patients had chemosis, 67% (4 patients) had papilloedema and 16% (1 patient) had cranial nerve palsy and bruit on examination. Papilloedema and cranial nerve palsy was due to raised intracranial tension, abducent nerve palsy being a false localizing sign.

Nearly 50% (3 patients) showed significant improvement in the presenting symptoms on follow up. In two patients (33%), there was no change in the presenting symptoms noted after embolization. One patient (16%) with transverse sinus DAVF, who was having normal mental functions before the procedure developed dementia following embolization. Follow up angiogram showed residual fistula which had recruited new dural feeders from ICA and had developed venous reflux with venous ectasia. None of

the patients died in this group. Patient who has developed venous rerouting is planned for next sitting of embolization.

TYPE IV

Type IV DAVF was noted in 9 patients of which 8 (88.9%) were males and one (11.1%) was a female. The age range was from 10 years to 59 years (mean age 34.5 years). Location of the fistula was in the transverse sinus in four (44.4%), torcula in two (22.2%), and cavernous venous plexus in two patients (22.2%). One patient (11.1%) had fistula at the sigmoid sinus.

Of these 9 patients, 56% (5 patients) presented with diplopia, proptosis, visual field defect and tinnitus, 67% (6 patients) with headache, 22% (2 patients) with focal neurological deficits, 33% (3 patients) with chemosis; papilloedema and 44% (4 patients) with cranial nerve palsy on examination. Papilloedema and cranial nerve palsy was because of increased intracranial tension, abducent nerve palsy being a false localizing sign.

Nearly 67% (6 patients) showed significant obliteration of the fistula on immediate post procedure angiogram and reduction of the symptoms. On follow up, one patient (11%) had worsening of the headache and visual deterioration. In two patients (22%), there was no change in the symptoms after embolization with persistence of

venous ectasia and cortical rerouting. Both of them were planned for next sitting of embolization.

Follow up angiogram in the patient with worsened symptoms showed residual fistula which had recruited many new dural feeders from ICA and had developed venous reflex. He was lost on follow up. One patient (11%) with persisting venous ectasia and cortical rerouting presented with headache and subarachnoid hemorrhage two weeks later. Two patients (22%) with type IV fistula died; one patient developed subarachnoid. This could be attributing to rupture of venous ectasia located in the parietal region. In other patient glue migrated into the venous side, during second sitting of embolization producing deep venous obstruction and intracerebral hemorrhage, following which the patient died.

TYPE V

We had only one male patient (1.3%) of 68 years age with this type of DAVF. Location of the fistula was in the transverse sinus with venous drainage into the perimedullary venous plexus of the cervical spinal cord.

This patient presented with headache and quadriparesis, and on imaging showed subarachnoid hemorrhage. He underwent DSA for subarachnoid hemorrhage, which showed type V DAVF in transverse sinus draining into perimedullary venous plexus and

also refluxing into posterior fossa cortical veins. Normal posterior fossa structures were draining through contralateral transverse sinus and ponto-mesencephalic veins. Transarterial cannulation of posterior meningeal feeder of vertebral artery was tried but failed. So fistula was treated by packing of the transverse sinus with coils. After embolization, patient failed to improve and died because of subarachnoid hemorrhage and hemodynamic instability.

CLASSIFICATION ACCORDING TO LOCATION

Fistulas were also classified based on location and which sinus is involved. Thirty five patients had fistula involving the cavernous venous plexus (48%). Twenty-four had fistula involving the transverse sinus (33%). Five patients had fistula-involving torcula (7%); 4 had it in the sigmoid sinus (5%), 2 in the superior sagittal sinus (3%) and one (1.3%) in the straight sinus.

Sixty three percent (22 patients) of fistulas involving the cavernous venous plexus presented with headache. Ninety seven percent (34 patients) of fistulas involving the cavernous venous plexus presented with diplopia, proptosis, chemosis, and 86 % (30 patients) cranial nerve palsy and visual field defects. Cavernous DAVF was associated with diabetes mellitus in nearly 68% of cases (24 patients).

Seventy one percent (17 patients) of fistulas involving the transverse sinus presented with headache. Forty two percent (10 patients) of fistulas involving the transverse sinus presented with diplopia and visual field defects. Hypertension was associated with transverse sinus fistula in nearly 43% (10 patients).

Seizures were common when the fistula was involving more than one sinus. Focal neurological deficits were observed in patients with location of the fistula in the torcula. Miscellaneous risk factors like hormonal contraceptives and history of trauma were associated with transverse sinus and cavernous DAVFs more than other sites.

Over all demographics of our analysis showed majority of the patients were in the age group of 30 to 60 years. Sixty-two percent (45 patients) were males and 38% (28 patients) was females. Only 25% (18 patients) of the patients had risk factor of diabetes mellitus. Hypertension was present in only 19% of cases (14 patients). Miscellaneous risk factors like oral contraceptives, trauma or post partal period (one patient was 5 weeks post partum and other was 13 weeks post partum) was present in 11% of cases (8 patients).

Results were better when fistulas were located in the transverse sinus, cavernous venous plexus and torcula region. Transverse sinus had 79% (19 patients) rate of obliteration of fistula with significant reduction of symptoms. The success rate was 71% (25 patients) when the fistula was located in the cavernous venous plexus.

Complication rate was more with fistula located in the anterior cranial fossa and involving the superior sagittal sinus (50%, one patient) with cortical venous reflex. When the fistula was in the transverse sinus or cavernous venous plexus, complication rates are 14 to 16%. When fistula was located at multiple sites, staged treatment and treatment of aggressive factors resulted in decreased complication rate in this group.

When analyzing the various embolic agents, combination of embolic agents had produced the best results with nearly 92% patients showing significant improvement in symptoms. Transvenous coiling had also produced good results with 83% patients showing improvement in symptoms. Palliative treatment with poly vinyl alcohol particles had produced reduction of symptoms in nearly 68% of our cases. Complications encountered with glue were mainly due to crossing of glue to the venous side. N-Butyl Cyanoacrylate is a versatile agent, which indicates that expertise is required for utilizing it effectively. After availability of onyx, these complications are not encountered with onyx. With analysis of risk factors for aggressive presentation like hemorrhage with type of DAVF, showed type III and IV DAVF had a statistically significant association with hemorrhagic presentation ($p=0.03$). Of the 15 patients in type III and IV DAVF, 12 had hemorrhagic presentation either in the form of intracerebral or subarachnoid hemorrhage, which accounts for nearly 80% of cases.

Results were better, when embolization was done in staged manner. Our study showed better results with two sittings of embolization. Cranial nerve palsy was more

frequent when fistula was located in the cavernous venous plexus. Complication rate was more when fistula was in the anterior cranial fossa and involving superior sagittal sinus with cortical venous reflux (100%). We had two patients in this group; one developed MCA stem occlusion (Due to thromboemboli from the guiding catheter) and other developed intracerebral hemorrhage and developed hemiplegia. This patient was lost on follow up. MCA stem occlusion opened up after urokinase infusion. Patient had transient monoparesis, which improved with physiotherapy.

IMMEDIATE & FOLLOW UP RESULTS

Nearly 72% (52 patients) showed near complete closure of the fistula in the immediate post procedure angiogram. Majority of the patients who showed improvement were having a fistula of lower grade. (Less than Type III according to Cognard et al classification). Forty four patients (60%) had fistula located in the cavernous venous plexus and transverse sinus showed improvement. Eight percent (5 patients) showed deterioration compared to the preprocedure angiogram, either in the form of cortical venous reflux or venous ectasia or recruitment of more dural feeders from ICA and retrograde flow in the sinuses. Two patients in this group had lost follow up. Two are waiting for next sitting of embolization. One patient improved after second sitting of embolization with coils through transvenous route. Six patients died following the procedure due to intracerebral hemorrhage, subarachnoid hemorrhage or hydrocephalus producing pressure effects.

In the follow up period, 63 patients (86%) were followed up clinically. Ninety one percent (57 patients) showed significant improvement of the symptoms compared to the 72% (46 patients) in the immediate post procedure angiogram. However, the follow up angiogram after 1 year in majority of the cases showed recruitment of tiny feeders from ICA decreasing the anatomical cure to 59% (37 patients) from 72% (46 patients) in the immediate post procedure period. Overall, complication rate in our study was 19% (14 patients). In four patients (5.5%) complications was transient, which improve in the follow up period and able to perform all activities of daily living independently. We had a mortality rate of 8.3% (6 patients). Permanent complication in form of focal neurological deficit and dementia was encountered in four patients (5.5%). In two patients it was due to hemorrhage and in one it was due to infarct. Progressive dementia noticed may be due to progressive venous hypertension.

Results were better when fistula is located in the cavernous venous plexus or in the transverse sinus. Similarly fistula of the lower grade up to IIb had favorable outcome compared to the higher grade. Patients were followed up and assessed with Modified Rankin scale (66). Scale is given in the table below. Our results by using this scale were 78% (57 patients). All these patients ≤ 2 in this scale. Only 12 patients (22%) were of ≥ 3 with dependent life style. We had a mortality rate of 8.3% (6 patients) according to this scale.

MODIFIED RANKIN SCALE

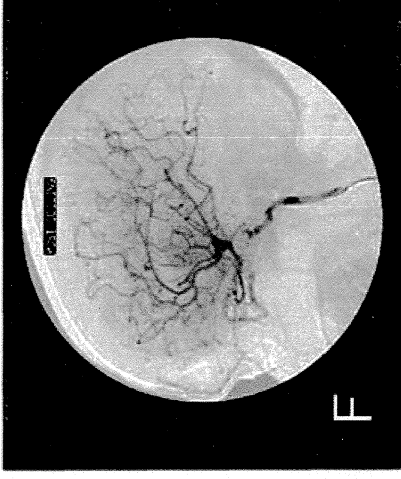
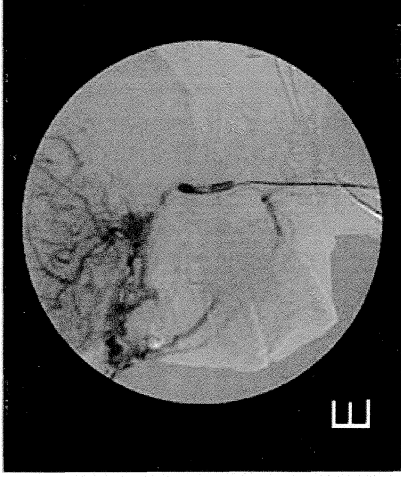
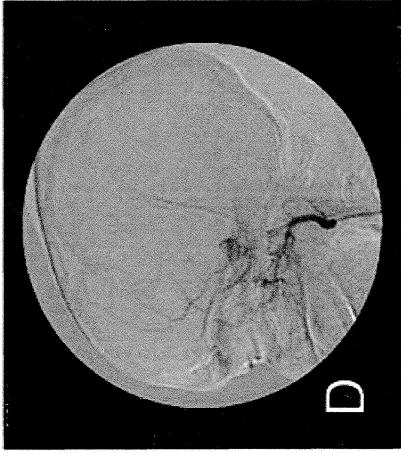
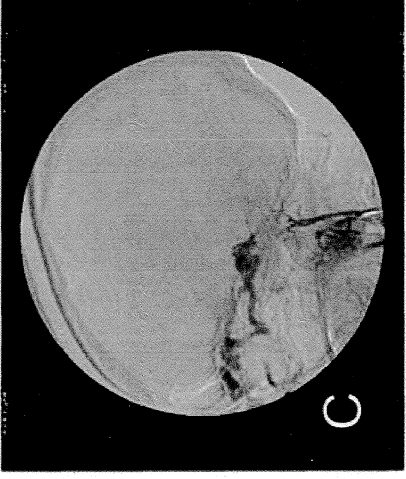
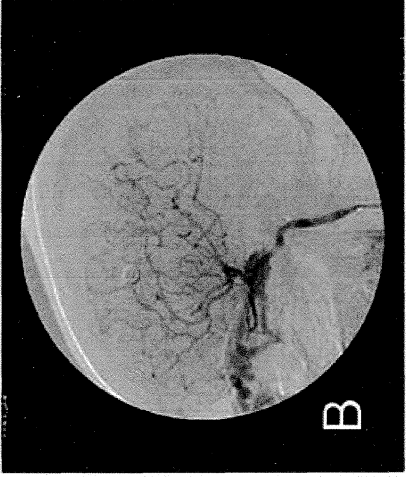
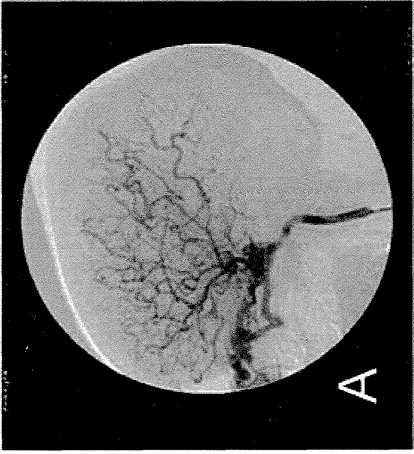
CORE	DESCRIPTION
0	No symptoms at all
1	No significant disability despite symptoms; able to carry out all usual duties and activities
2	Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance
3	Moderate disability; requiring some help, but 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

OUR STUDY RESULTS WITH MODIFIED RANKIN SCALE

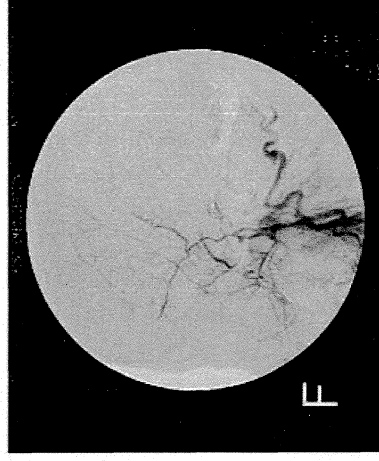
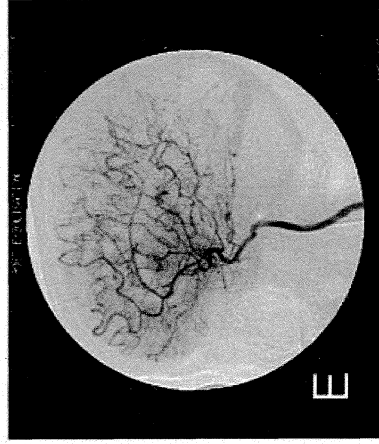
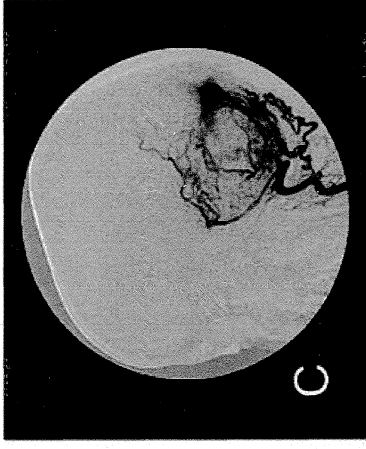
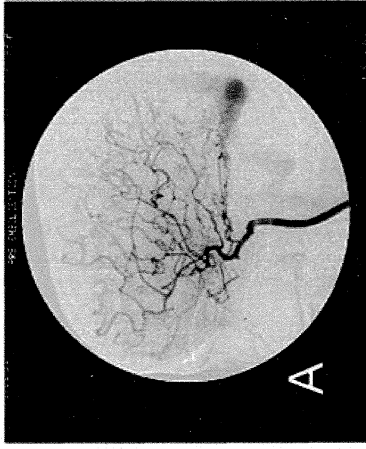
SCORE	NUMBER OF PATIENTS
0	19
1	36
2	2
3	2
4	3
5	1
6	6

ILLUSTRATIVE CASES

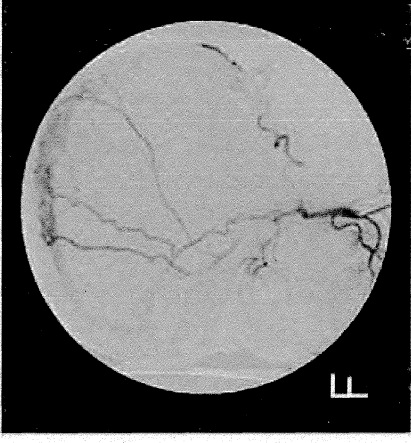
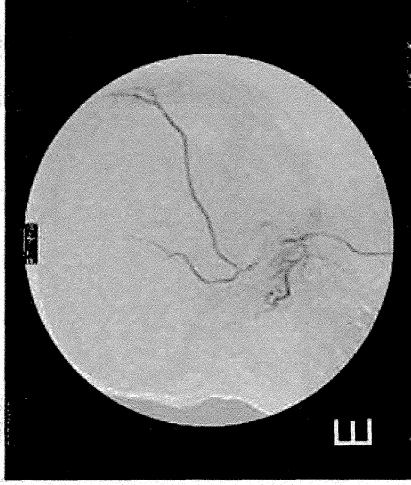
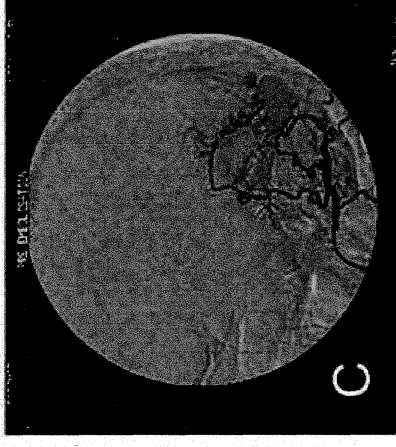
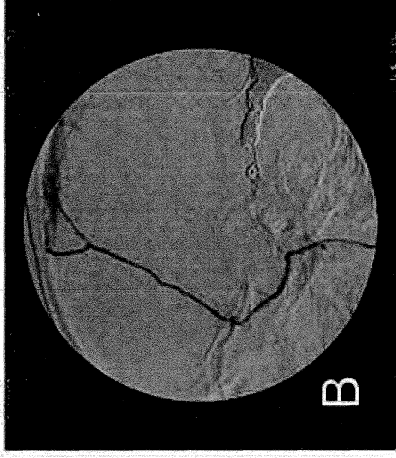
CASE NO : 1, DAVF TYPE IIA



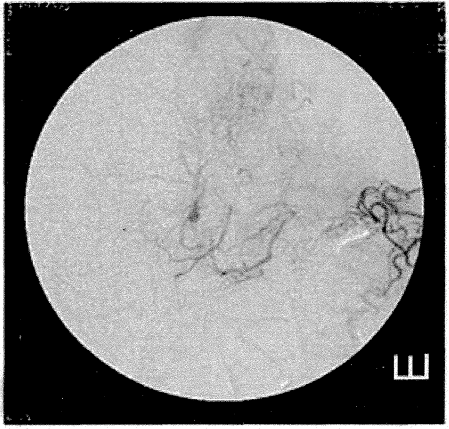
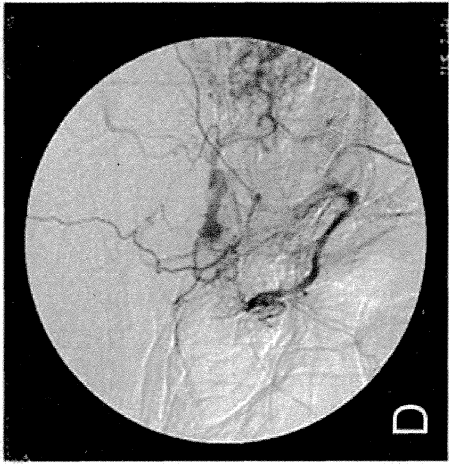
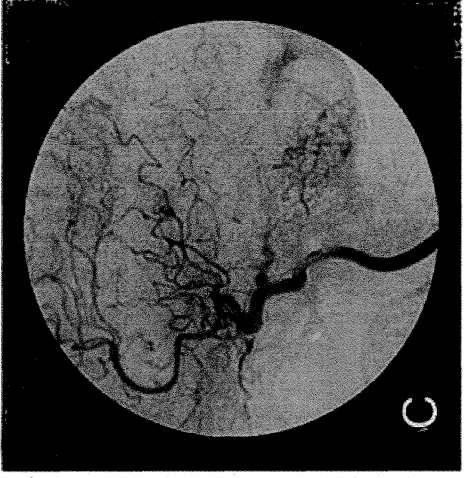
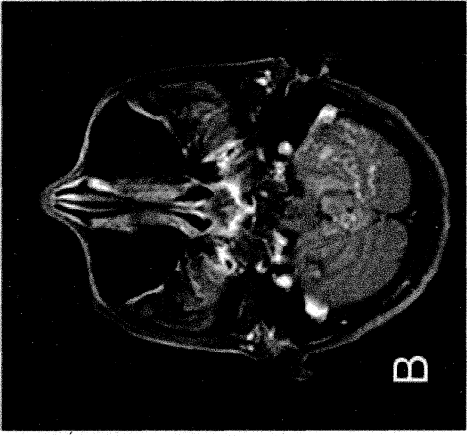
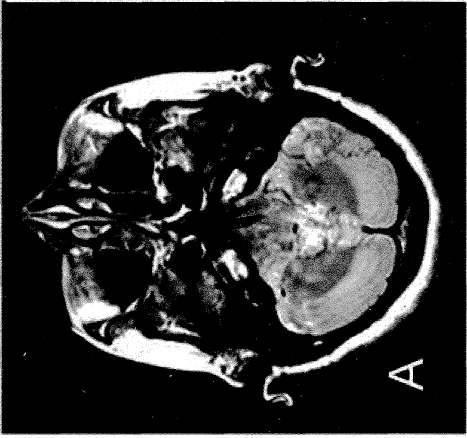
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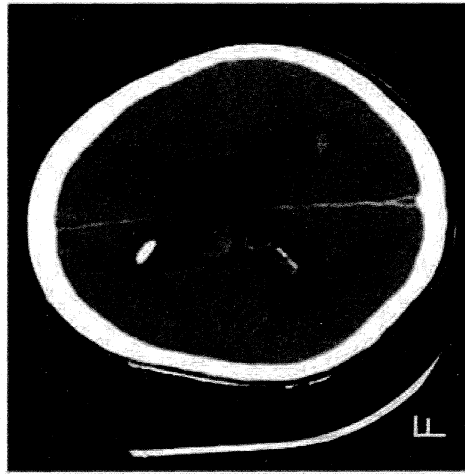
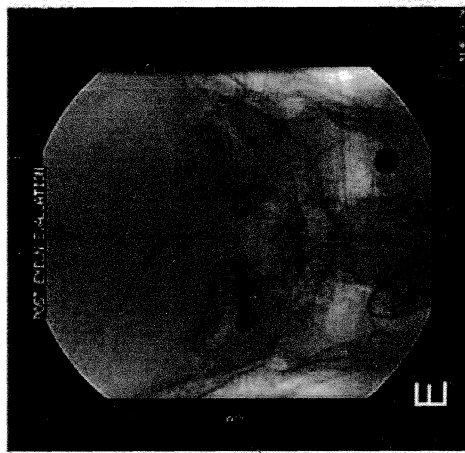
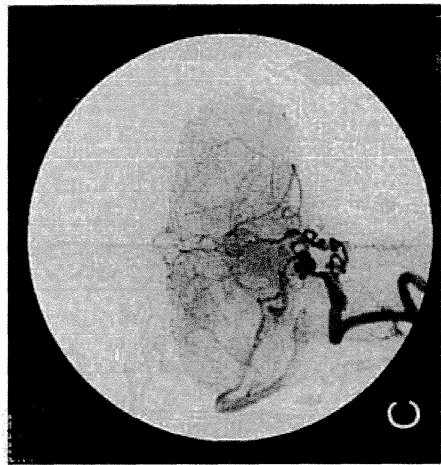
CASE NO : 2, DAVF TYPE IIA+B (2nd & 3rd sitting)



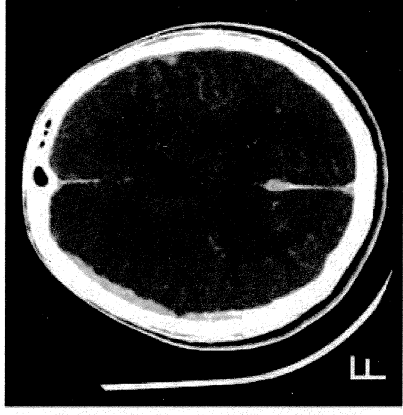
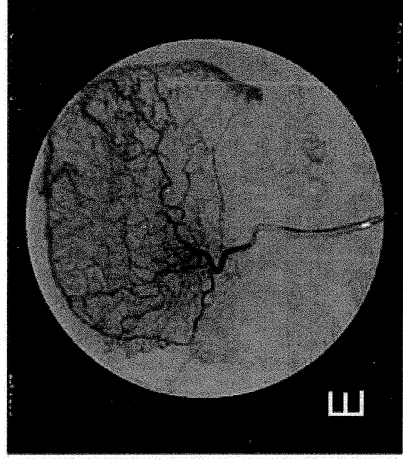
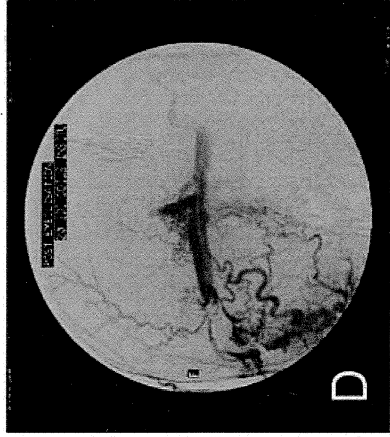
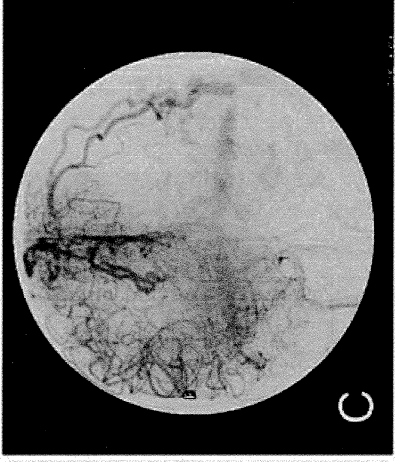
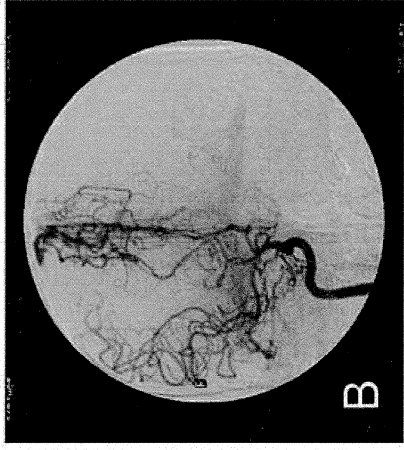
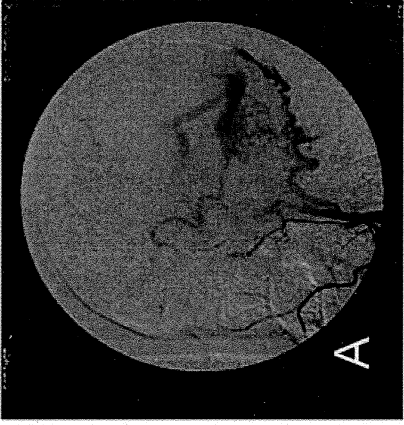
CASE NO : 3, DAVF TYPE III



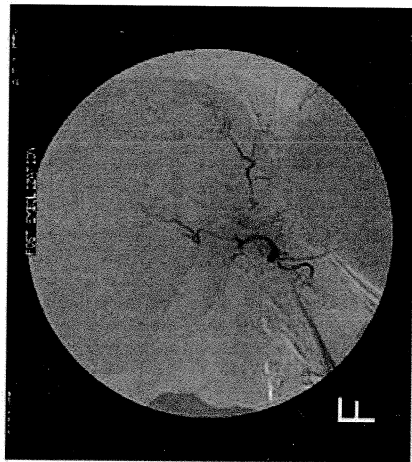
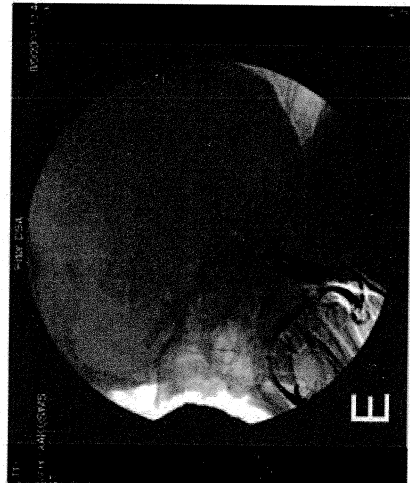
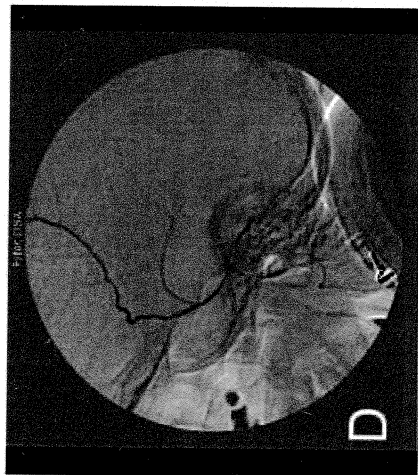
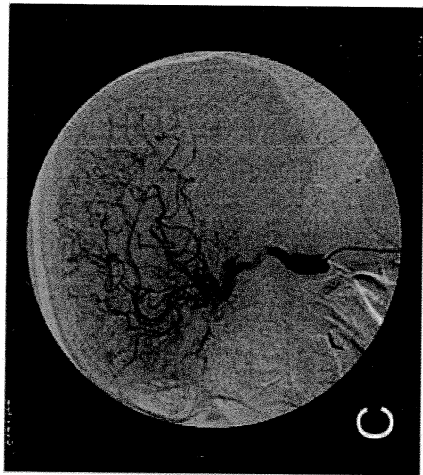
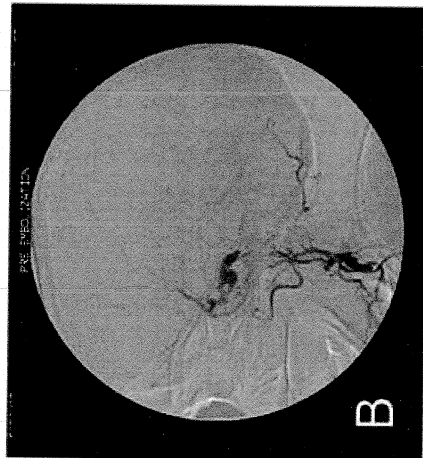
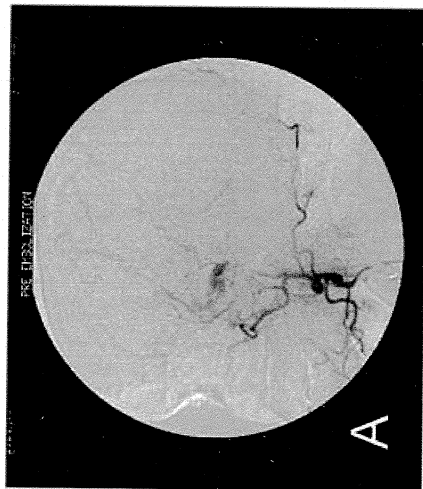
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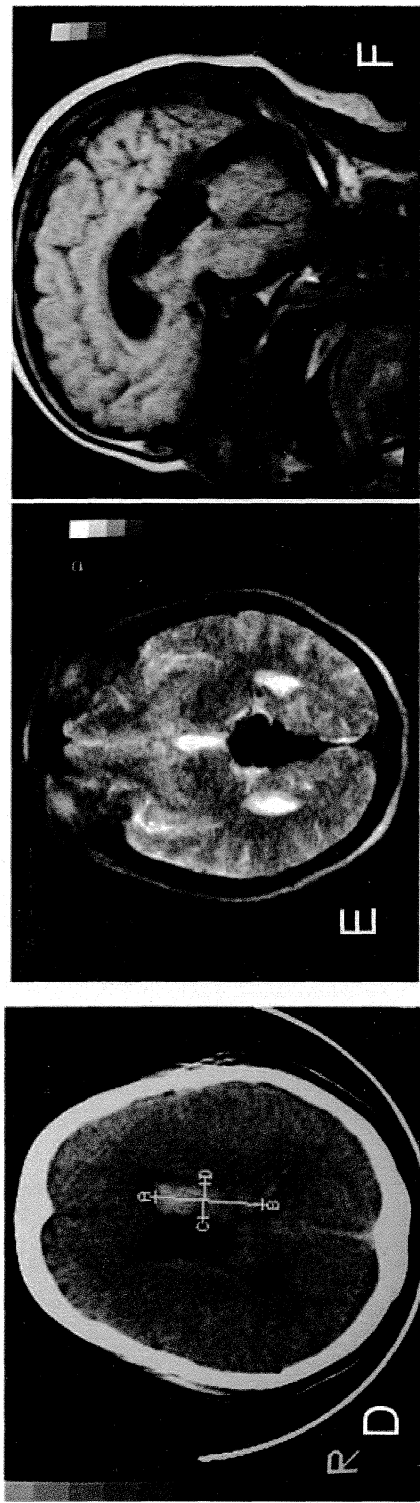
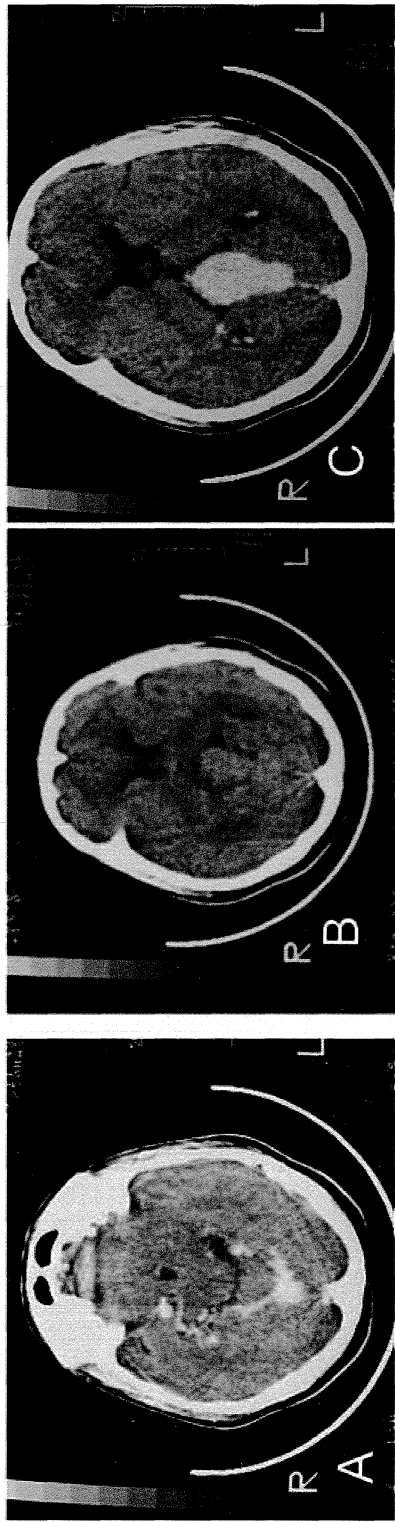
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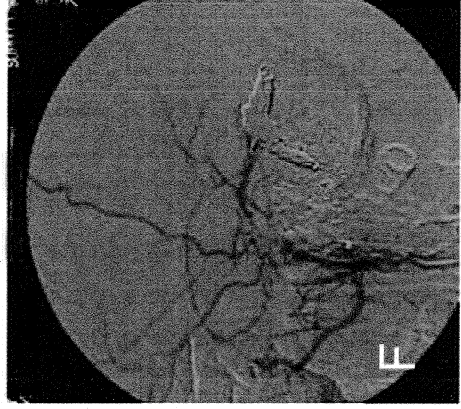
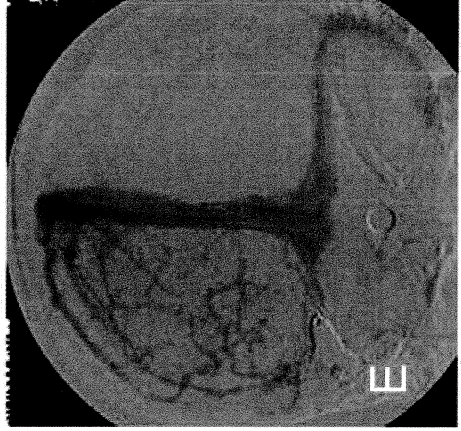
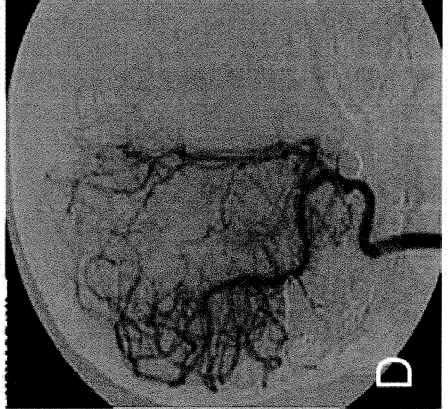
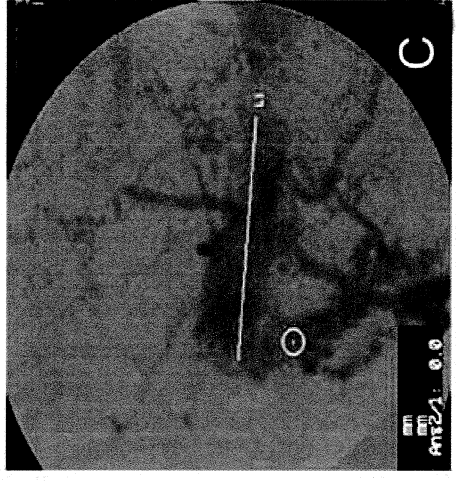
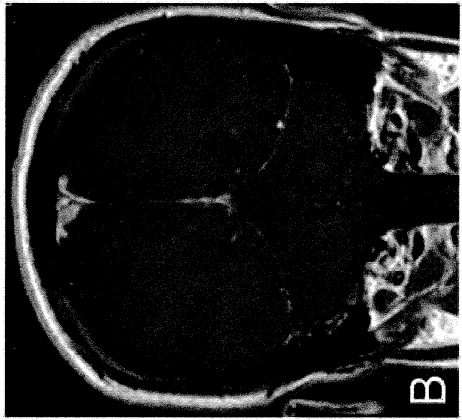
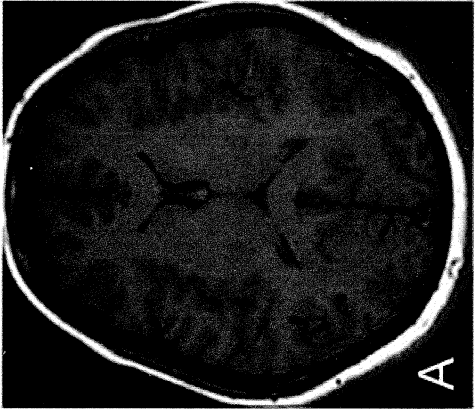


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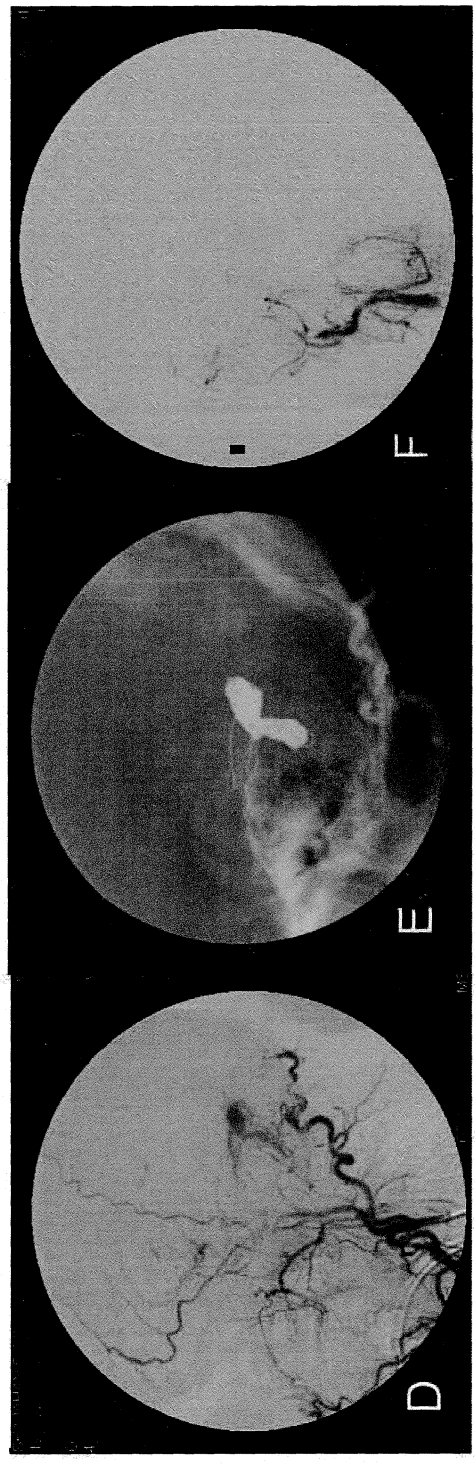
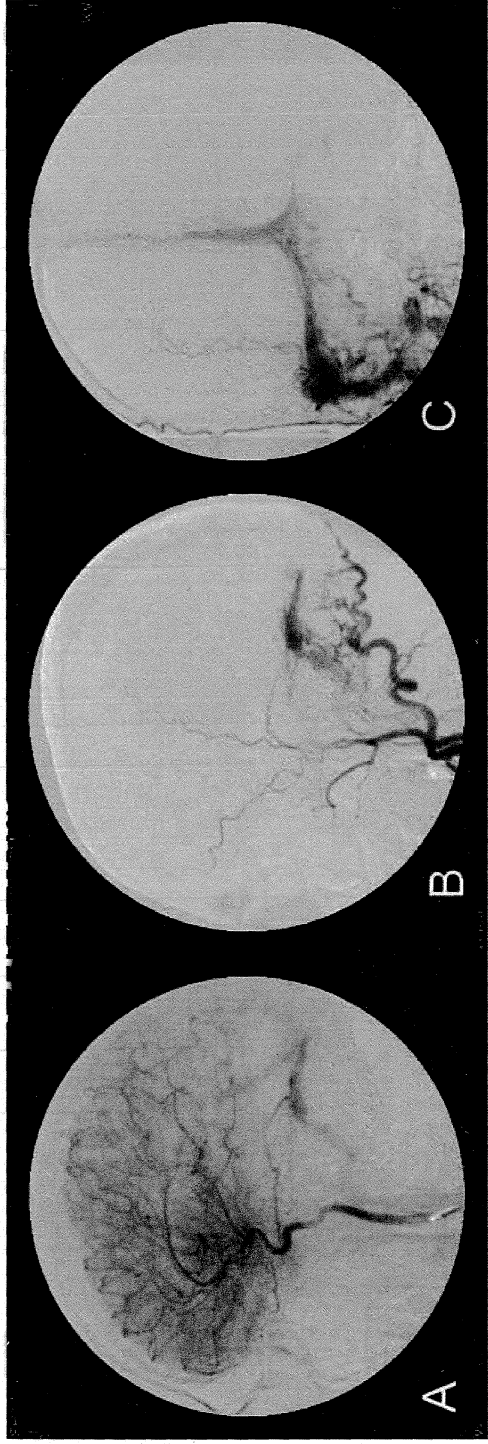


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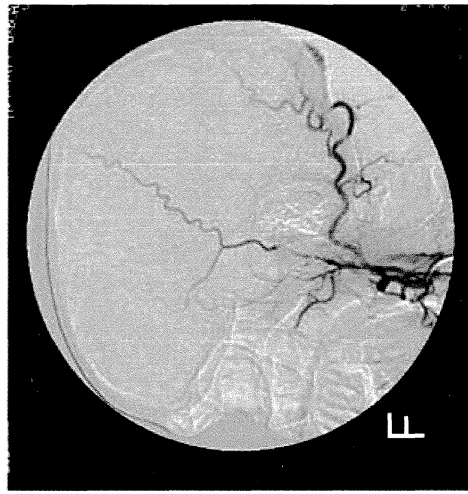
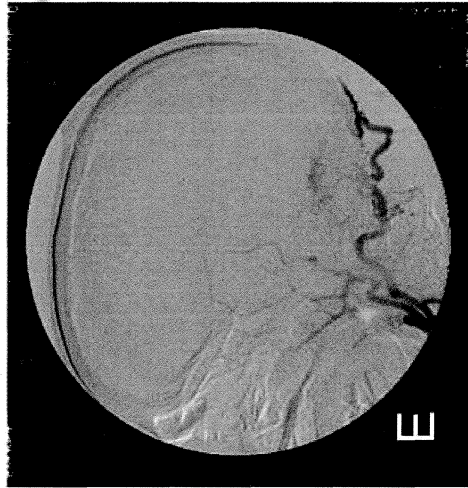
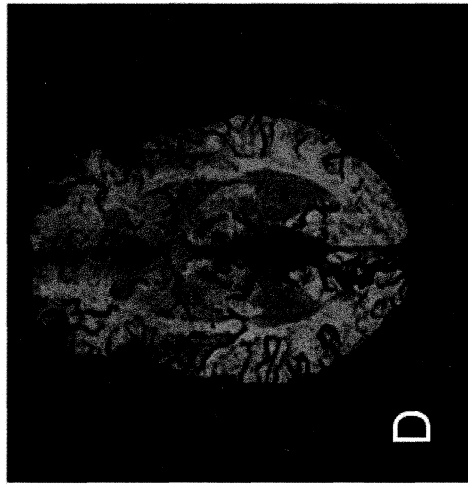
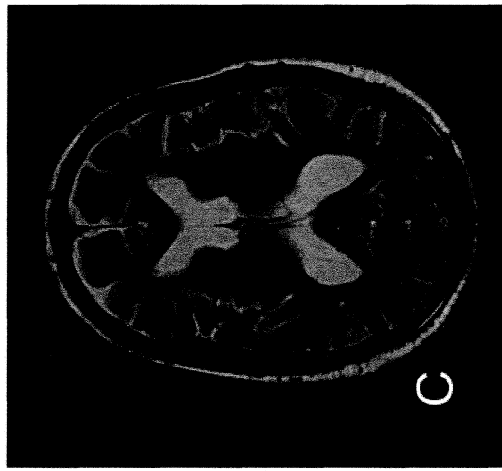
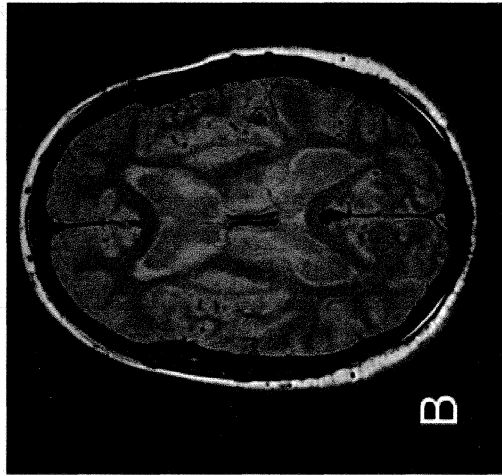
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CASE NO : 9, DAVF TYPE IIA+B

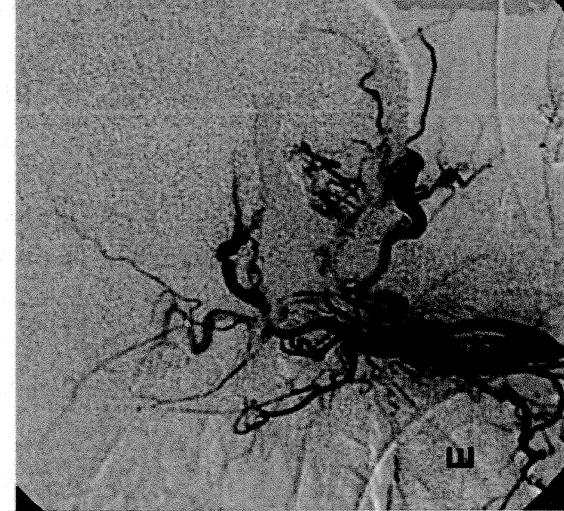
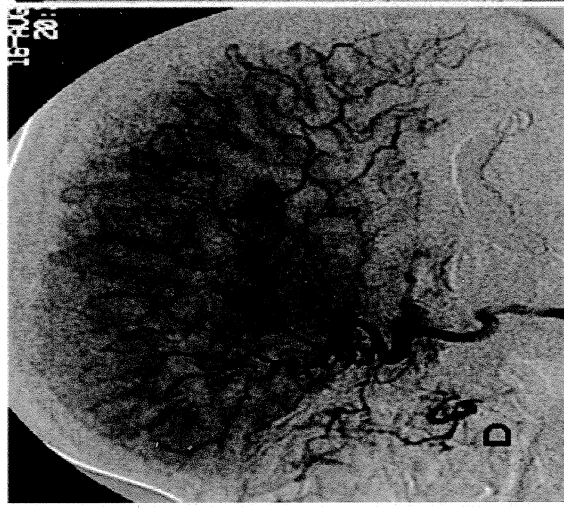
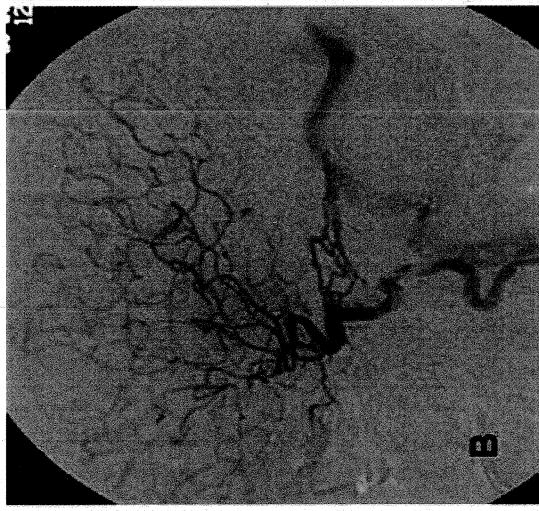
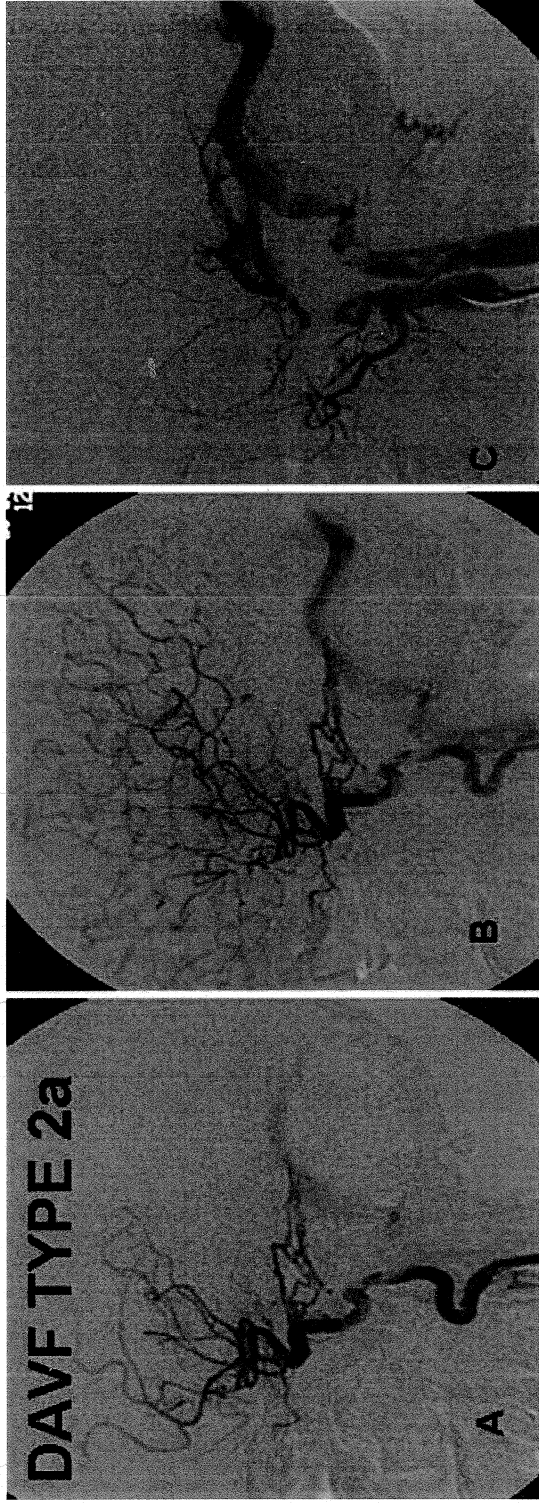


CASE NO : 10, DAVF TYPE IIB



CASE NO : 11, DAVF TYPE IIA

DAVF TYPE 2a



DISCUSSION

DISCUSSION

DAVFs are a heterogeneous group of vascular abnormalities that can occur anywhere within the intracranial dura mater. They represent 10%–15% of all intracranial vascular malformations. It may be asymptomatic or may experience symptoms ranging from mild symptoms to fatal hemorrhage, depending on the location (eg, cavernous venous plexus, transverse-sigmoid sinus, tentorium, superior sagittal sinus, anterior fossa) and venous drainage pattern of the AVF. Several classification systems have been developed to grade the risks of DAVFs, those devised by Cognard et al (4) and Borden et al (7) are the most widely used. DAVFs can be classified into (Type I) Venous drainage into dural venous sinus with ante grade flow; (Type IIa) Venous drainage into dural venous sinus with retrograde flow; (Type IIb) Venous drainage into dural venous sinus with ante grade flow and CVR; (Type IIa+b) Venous drainage into dural venous sinus

with retrograde flow and CVR; (Type III) Venous drainage directly into subarachnoid veins (CVR only); (Type IV) Type III with venous ectasia of the draining subarachnoid veins and (Type V) Direct drainage into spinal perimedullary veins (4).

Majority of our patients are of older age group, correlating with acquired nature as stated in some of literatures (60). Although the exact pathogenesis is still debated, most authors now agree regarding the acquired nature of DAVFs (7). The epidemiological features of the clinical manifestations and the many reported cases with previously normal angiograms support this theory (61).

Twenty percent of our patients, who were of younger age group, were also having associated risk factors such as sinus thrombosis induced by post-partum state, hormonal contraceptives, trauma or surgery. These are some of the causes postulated in the literature for the development of dural arteriovenous shunts (13, 61). Venous occlusion also seems to explain the dynamic behavior of some DAVFs.

Halbach et al described 30 cases of dural fistulas of the cavernous sinus out of a total of 158 cases of CCF. Of these 30, 22 were females and eight were males .The patient's age ranged from 22 to 75 years with a mean of 47 years (21). Théaudin et al described 27 cases of cavernous dural fistulas. There were 18 females and 9 males with age ranging from 32 to 80 years (36).

Common neurological manifestations in our study were headache, proptosis, diplopia and visual field defects, which were present in 65 to 66% of patients. In Borden et al and Davies et al series most common neurological manifestation was pulse-synchronous bruit (7, 30). In our study group only 24 % (17 patients) had pulse-synchronous bruit at presentation. This might be due to the increased number of cavernous DAVFs in our study, which is usually of low grade when it occurs spontaneously in the elderly age group.

On analyzing association between headache and the location of fistula, our series found increased correlation of patients presenting with headache, with the fistula is located in the superior sagittal sinus or anterior cranial fossa (100%), or involving the straight sinus (100%). This indicates that the fistula at these sites is more prone for aggressive presentation like hemorrhage and focal neurological deficits.

On analyzing the association of proptosis, diplopia and visual field defects with the location of fistula, our series showed increased percentage of patients presenting with these symptoms when fistula is located in the cavernous venous plexus (97%). This is similar to the results of Eskridge et al; out of twenty-seven patients in their series, more than 90% had symptoms of proptosis, diplopia and visual field defects (47).

Halbach et al described cavernous dural fistulas in 30 patients. In their patients 23 (77%) had proptosis, 14(47%) had bruit, 15 patients (50%) had diplopia from ophthalmoplegia and 8 (27%) had diminishing visual acuity (21). Theaudin et al studied 27 patients with cavernous DAVFs. The most common clinical symptoms were diplopia in 12 (45%), conjunctival injection in 11(41%), chemosis in 10(37%), proptosis in 10(37%) and visual deterioration in 8 (30%) patients (36).

When the clinical diagnosis of dural fistula is suspected, adjunctive MR imaging or CT scanning is of little value, but maybe useful in establishing the location of fistula and identifying aggressive presentations. CT is especially useful to identify hemorrhage before interventions, so that aggressive factors can be looked for. The CT / MR show the white matter hyper intensities, dilated vessels in the scalp, prominent flow voids adjacent to the sinuses, dilated SOV, proptosis & bulging of the cavernous sinus. Dilated superficial veins, which represent an ominous sign of cortical venous drainage, can also be visualized. Orbital Doppler may also demonstrate dilated SOV in addition to a reversal in the direction of the blood flow in SOV.

With the imaging clues, a working diagnosis is well established. However, angiography remains the gold standard for establishing the complete diagnosis. The angiographic work up should include bilateral vertebral, internal and external carotid artery injections.

Cases which have been treated previously, particularly those which involved the sacrificing a major feeder, the DAVFs may recruit supply from other ECA branches or rarely from costocervical and thyrocervical trunk through ascending pharyngeal arteries. These vessels also need to be studied in such situations. Each case however is unique in its own sense and angiographic evaluation should be tailored accordingly. Ideally, the initial angiographic work up should be sufficient to both establish a diagnosis and plan therapy.

Fourteen percent of our patients (10) presented with altered mental status, this is due to the increased venous hypertension. In our study around 16 – 20% (13 patients) presented with altered sensorium. It is generally believed that in DAVF patients intracranial hemorrhages or other aggressive CNS symptoms (including focal neurological deficits, seizures, intracranial hypertension, and altered mental status) are attributable to leptomeningeal venous drainage or passive venous hypertension (62).

We believe that dilated leptomeningeal or medullary vessels may represent retrograde leptomeningeal venous drainage (RLVDs) or congested medullary veins and are related to venous hypertension. The medullary veins seen on MR imaging may be seen as pseudophlebitic pattern on conventional angiography (which is seen during the venous phase), and is related to an aggressive presentation and white matter T2 hyperintensity (36). T2 white matter hyperintensity seems to be a relatively late-presenting feature because all the patients with this finding also show dilated vessels and

prominent vascular enhancements on MR imaging, while the vice versa is not true. Advanced imaging sequences like susceptibility weighted imaging will help in identifying these abnormalities. It can show increased prominence of veins adjacent to the fistula or thrombosed sinus.

ANGIOGRAPHIC CONSIDERATIONS PRIOR TO TREATMENT

The objectives of the initial angiogram are to:-

- Establish a diagnosis.
- Differentiate the type of dural arteriovenous fistula (DAVF).
- Identify all feeding arteries and site of the fistula.
- Fully identify the venous drainage of the fistula as well as the normal brain parenchyma.
- Identify and confirm the patency of outflow pathways of the sinus.
- Identify high-risk features such as cortical venous drainage, venous ectasia and venous hypertension.

Venous drainage of the fistula

Three critical points should be looked for. First is the presence of cortical venous drainage, which increases the risk of hemorrhage. Second is whether the venous drainage of the fistula is compromising the normal brain parenchymal venous drainage, thus causing intracranial venous hypertension. Third is to see whether the retrograde venous access to the fistula site is possible or not, for transvenous embolization when indicated.

A total of 73 patients with DAVF were analyzed. DAVF were classified according to classification by Cognard et al. Of these, Type I fistula was noted in 3 patients (4%), type IIa in 28 (39%), type IIb in 7 (10%), type IIab in 19 (26%), type III in 6 (8%), type IV in 9 (12%) and type V in 1 patient (1%) (Represented in the color pie charts).

CURRENT APPROACHES TO TREATMENT

Carotid compression

DAVF treatment includes different techniques: non invasive techniques are oral therapy with low dose aspirin and repeated manual compression of the ipsilateral occipital artery behind the mastoid or carotid jugular compression with increasing frequency and duration (4). In our series we advised this technique in seven patients with type I and IIa and followed up them clinically. All patients showed significant improvement in the symptoms and four of them in the follow up DSA showed obliteration of the fistula. We did not use aspirin, in view of its antiplatelet action.

We had total 31 patients with low flow type I and IIa fistula of which, seven had shown improvement with carotid compression. Some studies have also reported a spontaneous regression or a rare conversion to aggressive type, in a benign DAVF, a DAVF with RLVD, with aggressive neurological symptoms which necessitates a prompt treatment (53).

Transarterial embolization

The efficacy and safety of the permanent solidifying agent ONYX are well established in arterio-venous malformation (AVM). Their uses in dural fistulas have been described, with increased focus on preserving sinus flow. N-Butyl cyanacrylate can be used with safety precaution of occluding only the feeding arteries without occluding the normal venous drainage.

In general, the transarterial particle embolization is done in nearly all cases of DAVFs (whether they involve the cavernous venous plexus or transverse/sigmoid sinuses) helps to slow the flow through the fistula, and hence to prepare it for transvenous coil embolization (14).

A simpler procedure such as particle embolization is preferred for symptomatic relief in the patients presenting with disturbing bruit. The possibility of progression from

a low-risk type DAVF to a more aggressive one, although rarely reported (14), warrants clinical follow-up of uncured patients. High-flow transverse-sigmoid DAVFs with reflux and retrograde flow were associated with potential neurological complications in a recent series (4). For palliative symptom relief, arterial embolization is better (13). With the availability of newer agents like ONYX permanent cure can be achieved through arterial route.

Transvenous embolization

Transvenous coil occlusion of the DAVF is indicated in the cases where transarterial route has failed to obliterate the fistula or when there is recurrence of fistula. The prerequisite is that the sinus, which is to be obliterated, should not be draining the normal brain parenchyma. For this technique to be effective, the occlusion must be at the point of the fistula. If the occlusion is performed any further downstream, it might simply redirect the venous drainage and can increase the risk of hemorrhage.

In our study also transvenous approach had the maximum cure rate for obliteration of the fistula, considering an individual approach. We had a symptom reduction in 83% (61 patients), only slightly less than combined approach, which achieved a cure in 67 patients (92%). In the series published by Halbach et al, 31 cavernous sinuses were successfully embolized in 28 patients. Complete angiographic obliteration of the fistulae was achieved immediately in 25 patients. Residual shunting

was observed in the other 3, who had drainage through the pterygoid plexus (2 patients) or the inferior petrosal sinus (4) after the procedure. Headache and vomiting were the most common symptoms after the embolization (63).

One of our patients had a posttraumatic cavernous DAVF for which she underwent combined arterial and venous route embolization (PVA particle embolization from ECA feeders and cavernous venous plexus coiling via venous route) at the first sitting with complete closure of the fistula. However the symptoms (and fistula) recurred after one month and the patient underwent second sitting of transvenous coiling with complete obliteration of the fistula. A follow up angiogram at two years showed no recurrence of the fistula.

One of the drawbacks of venous embolization is the risk of changing the venous drainage of the fistula from a benign one to a more aggressive one with subarachnoid vein involvement (7). As stated by Borden et al, venous rerouting can leads to hemorrhagic complication. Post embolization 5 of our patients develops hemorrhagic complication. (Two of our patients had intraventricular bleed which was difficult to explain as venous occlusion give rise to parenchymal hematoma or SAH). So the best indication for venous embolization is the patient in which the involved sinus is already compromised and no longer contributes to the drainage of normal brain parenchymal tissue (14).

Caution is recommended when contemplating occlusion of a sinus that is freely communicating with normal venous structures. The use of detachable balloons for

occlusion of transverse sinus has also been recommended (64). But in none of our cases we used balloon to occlude the sinus. Urtasun et al (14) reported a higher percentage of immediate occlusion. This is probably due to the more frequent use of glue in combination with coils in their series. In our series, glue was used in combination with coils through venous route in 3 cases only. In these cases thromogenicity of coils was thought to be insufficient to close the fistula.

Our follow up results of 33 cases of cavernous DAVF are comparable with those of Halbach et al (21) who reported clinical cure rates of 81% in cavernous DAVFs and cure or marked improvement in 55% and 35% of patients with transverse and sigmoid DAVFs, respectively. Ninety-one percent of our patients showed improvement in the follow up period after endovascular therapy. In the immediate period only 72% patients showed improvement. This could be attributed to some of the transient procedural complications encountered in our cases. This could also be attributed to slow recovery of the secondary changes already done by the fistula.

To treat retrograde leptomeningeal and cortical reflux, the venous approach will more likely result in a predictable cure (13). Super selective disconnection or obliteration of the refluxing vein is preferred over sacrifice of the dural sinus although this is often necessary to achieve a cure.

Angioplasty and stenting of the stenosed or occluded torcula and transverse sinus, with packing of the fistula on the opposite transverse sinus with coils was done in two of

our cases. One of these patients showed significant improvement of the headache and visual disturbance. The other patient presented 6 month later with deep vein thrombosis for which he was started on anticoagulants. With these stent devices, now it is possible to reconstruct a severely stenosed sinus. Kieran et al have described the use of stents to treat DAVFs and had obtained closure of the feeders (53). Some authors have tried to close the shunts in the dural arteriovenous fistulas with the help of stents (52). Long-term results from the use of stents in the treatment of DAVFs are not yet known. Studies with significant number of cases need to be done, before coming to final conclusions. The advantage of using the stent is that an ante grade flow can be established in the stenosed or occluded sinus with the help of the radial force in the stent.

SURGERY

In our series none of the patient underwent surgical resection. Two of our patients underwent external ventricular drainage for hydrocephalus due to SAH, which had happened one to two days post procedure. Surgical techniques include feeder artery ligation, surgical excision of the fistula plus involved sinus and surgical disconnection of the sinus involved. Feeder artery ligation technique has been abandoned because of its ineffectiveness (21). Surgical excision of the fistula plus involved sinus is often associated with massive intraoperative bleeding (65). Surgical disconnection of the involved sinus consists of disconnection of the sinus from veins carrying high-pressure arterialized blood. DAVFs of the anterior cranial fossa can be safely treated by surgery (13).

COMPLICATIONS

We encountered various complications during the endovascular management of our patients. Complications were noted in total 14 procedures (patients). Out of these 6 patients died. Two of these patients encountered intracerebral hemorrhage because of persisting cortical venous reflux in spite of embolization with glue and coils. In the third patient, during packing of sinus, one coil went into the cortical vein, which might have perforated the vein causing subdural and subarachnoid hemorrhage. In fourth patient, glue migrated to the venous side and occluded the normal venous drainage, causing hemorrhagic venous infarct. Subarachnoid hemorrhage developed after coiling of the fistula through venous route in the fifth patient. Obstructive hydrocephalus was not responding to external ventricular drainage and patient died on the fourth day. Sixth patient had the fistula at the junction of torcula and straight sinus, which was embolized with coils and glue through venous side. Patient developed intraventricular hemorrhage and died after few days because of migration of glue into the deep veins.

Patients were followed up and assessed with Modified Rankin scale (66). Our results is given in the table below. Our results by using this scale were 78% (57 patients). All these patients ≤ 2 in this scale. Only 12 patients (22%) were of ≥ 3 with permanent complications had a dependent life style. We had a mortality rate of 8.3% (6 patients) according to this scale. Transient complications were noted in 21 patients (29%). These

complications were like diplopia, mild headache and minimal visual field defects. All these will come in less than 2 according to this modified rankin scale.

OUR STUDY RESULTS WITH MODIFIED RANKIN SCALE

SCORE	NUMBER OF PATIENTS
0	19
1	36
2	2
3	2
4	3
5	1
6	6

Three patients developed infarcts following particle embolization of the external carotid feeders. This happened mainly due to opening of dangerous anastamosis between external and internal carotid arteries, which was not visible on the initial angiogram. Once there was significant reduction in the shunt, particle had gone through the dangerous anastamosis to the internal carotid territory. This indicates that periodic check angiogram should be done during particle embolization of external carotid feeders and in all angiogram opening of dangerous anastamosis should be looked for.

Embolic infarcts occurred in the ACA and MCA territory. First patient had infarcts in the ACA territory and the motor cortex & the second had watershed infarcts in bilateral ACA-MCA territory. Third patient had large MCA territory infarct following which he developed dense hemiplegia. Out of these 3 infarcts two were small without any permanent neurological deficits (transient monoparesis improved with physiotherapy

in the first case); one was large to produce hemiplegia, which improved with physiotherapy, but still dependent on others for activities of daily living.

Two patients with cavernous DAVF developed increase in diplopia after transvenous packing, due to pressure effects on the cranial nerves in the lateral wall. Steroids improved the compression in one patient; but failed to do so in the other. Cortical venous rerouting developed in four patients. Two of these developed rerouting following transarterial embolization with glue while in one it occurred after packing of the sinus. In two patients transvenous packing of the sinus obliterated the fistula. One transverse sinus fistula was obliterated with embolizing the arterial feeder with onyx. One patient was lost follow up.

Two patients, who underwent particle embolization of the ECA feeders, had dissection of the IMA during the procedure. However no neurological deficits resulted. In one patient, after embolization with onyx microcatheter was stuck. So it was cut at the groin and left intra arterially.

Halbach encountered three transient and one permanent complication. In one patient liquid adhesive glued a catheter tip into the distal IMA. One patient had a transient increase in proptosis from occlusion of venous outflow of the orbit .One patient had not undergone selective angiography and suffered a transient decrease in vision when

the unrecognized meningolacrimal artery was embolized with PVA particles. The fourth patient suffered a stroke when thrombus was dislodged from catheter after removal from a direct carotid puncture. On follow up patient showed residual mild hemiparesis (21). In their series, permanent complications occurred in 4% of patients with cavernous lesions, whereas permanent complications occurred in 5% and transient complications in 15% of patients with transverse-sigmoid fistulas.

Our transient complications rate was about 19 to 20% (14 patients), which is more than reported by Halbach et al (21). This could be attributed to some technical difficulties and early days of performance. Dissection occurred during manipulation of micro catheters and wire in the external carotid branches. Permanent complication rate is also about 9%, which is more than Halbach et al series. This may be because in some patients after each bolus of particle embolization check angiogram was not done. This may have lead to missing the opening of dangerous anastamosis and inadvertent non-target embolization leading to infarcts. In few patients glue had migrated in to the venous side, leading to hemorrhagic venous infarcts.

Urtasun et al (14) reported anatomical cures in 80% and clinical cure in 90% of patients with transverse-sigmoid sinus DAVFs. Our series had also similar clinical cure rates of 91% and relatively less anatomical cure rate of 73%. This could be due to the inclusion of all the fistulas, including those with venous ectasia and with perimedullary

venous drainage in our series. Daniel et al (64) reported an 87.5% anatomical cure rate and a 96% clinical cure rate.

In our series we had managed seven patients (9.6%) of type I and IIa fistula with carotid compression. All of them showed significant reduction of symptoms on follow up. In the series of indirect CCF by Halbach et al, carotid compression therapy resulted in complete closure of fistula in 30% of the patients (50). In the series of 27 patients with cavernous DAVFs reported by Theaudin et al, one patient with leptomeningeal drainage developed cerebral hemorrhage after transvenous embolization of the fistula (36).

On follow up, 57 (90%) patients showed reduction of the symptoms. Out of 7 patients who had been advised carotid compression, the fistula got closed in four patients. Persistence of the fistula was noted in two patients who came for follow up. Both these patients were diabetic and had poorly controlled diabetes at the time of recurrence. Both of them were advised strict diabetic control and to continue with carotid compression. . It should be noted that patients who were treated with only carotid compression showed recurrence of their fistula when their diabetes flared up. This was an important observation. Hence, we advice all patients with cavernous DAVF and concurrent diabetes to have a strict control on their blood sugar level. Failing this, the cavernous DAVFs may recur. There was no morbidities/mortality in these patients. In the series by Halbach et al, carotid compression therapy resulted in complete closure of fistula in 30% of the patients treated (50).

Low flow dural fistulas type I and IIa in the cavernous region have a relatively high incidence of spontaneous closure. Barrows (11) encountered spontaneous closure of cavernous dural fistula in five of 14 patients (36%). This spontaneous cure can also be observed in bilateral spontaneous cavernous fistulas as described by Voigt et al (29). Seeger et al believed that a spontaneous cure of dural cavernous fistula in most cases is due to partial or complete thrombosis of the cavernous sinus and/or its tributaries (35).

One patient, who had undergone particle embolization of the ECA feeders with near total obliteration of the fistula, developed a recurrence after 2 months. This patient then underwent venous route embolization. The right IPS was not demonstrated. The left IPS was catheterized but the cavernous venous plexus could not be accessed due to absence of intercavernous communication (fistula was on right side). The SOV could not be accessed via the facial vein route and hence it was decided to proceed after angular vein cut down. Under all aseptic precautions, in the presence of an ophthalmologist, the angular vein was cut down and the SOV accessed. The CS was then packed with 12 coils with complete closure of the fistula.

Miller et al have described the treatment of 12 cavernous fistulas via the SOV approach. All their patients were successfully treated either by advancement of detachable balloon catheter to the CS with subsequent inflation and detachment

(11patients) or by introduction of multiple coils into the fistula (1 patient). All patients had complete fistula closure and resolution of symptoms and signs after occlusion of the CCF (49). Derang et al have described the SOV approach and have successfully treated 21 cases of cavernous fistulas (46).

In two patients burr hole was made over the torcula, since the fistula was recurring after multiple sittings of embolization through arterial and venous route. Through this burr hole initially coils and then some amount of glue was injected to obliterate the fistula. Out of these two patients, fistula was successfully obliterated in one while the other developed intracerebral hemorrhage.

LIMITATIONS OF THE STUDY

Number of patients in certain grades was very less, because of which we are not able to get statistically significant correlation between the results and the type of fistula. All patients with risk factors for the aggressive presentation are not given the same treatment protocol, because of ever growing new embolic agents like onyx. It was available in our institute only since 2006, but we started using it in DAVF cases only from 2007. Some patients with high flow fistulas did not undergo earlier arterial embolization and directly went for transvenous coiling. Some of our patients, in whom fistula was only partially treated, had lost follow up.

We need to remember that even though endovascular techniques are evolving and embolization is an appealing way to deal with this disease, Type I DAVF is a benign entity (7). Thus, the aggressiveness of the treatment needs to be tailored according to the patient's symptomatology and to the angiographic pattern that correlates with the prognosis. Whenever cortical venous drainage or venous hypertension is present in a DAVF, radiographically demonstrated anatomic cure is the goal of treatment. A combination of arterial and transvenous embolization as well as surgery, if necessary, is then indicated to prevent intracranial hemorrhage and raised intracranial pressure.

CONCLUSION

CONCLUSIONS

- 1) Dural arteriovenous fistulas are one of the vascular conditions where endovascular management has become the first line of treatment of choice. It has to be classified according to the commonly used classifications.
- 2) The features of aggressive presentation like hemorrhage, focal neurological deficit, deep venous drainage, cortical rerouting and venous ectasia should be looked for in all cases. Proptosis, redness, chemosis, diplopia, diminution of vision are the commonest symptoms and should alert the physician of a possibility of venous hypertensive changes. The symptomatology may be subtle in type I and IIa. A timely clinical evaluation followed by angiogram can direct the early management of these lesions. It is essential to

identify high-risk patients to provide immediate management with a view to preserve the vision as best as possible.

- 3) Non-invasive modalities should be the first line imaging, as it can help in predicting the location of the fistula and hemorrhage. The goal of endovascular management should be elimination of the angiographic risk factors like cortical venous reflux; venous ectasia, obtaining ante grade flow in the sinus and decreasing venous hypertension.
- 4) First transarterial route should be tried to obliterate the fistula. For this onyx is very good embolic agent. However instead of onyx glue can also be used. In cases where transarterial route fails to obliterate the fistula, transvenous coiling of the sinus should be considered to occlude the fistula. However, sinus occlusion should be planned only when it is not draining any normal brain parenchyma. Sinus occlusion can be carried out with coils with or without glue. The choice of types of the coils to be used depends on the angiographic features in the given case. Other methods like PVA particle embolization of arterial feeders, angioplasty and stenting of occluded or stenosed sinus can be carried out when above methods fail.
- 5) Given the present day expertise, technology, machines and available materials endovascular management of dural arteriovenous fistulas has extremely good results.

Endovascular treatment should be considered as the first choice in the treatment of DAVF.

- 6) The mortality or morbidity associated with endovascular management is very low in the present day. Previously encountered morbidities were due to in expertise and technical limitations. Combination of different embolic materials will have the best results. A detailed clinical evaluation, imaging work up, angiographic analysis and timely follow up for all patients with DAVFs is recommended.

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ABBREVIATIONS

CS	: Cavernous Sinus
TS	: Transverse Sinus
SSS	: Superior Sagittal Sinus
St S	: Straight Sinus
SS	: Sigmoid Sinus
CVR	: Cortical venous reflux
DCCF	: Dural Carotico Cavernous Fistula
DM	: Diabetes Mellitus
ECA	: External Carotid artery
EDS	: Ehler's Danlos Syndrome
HT	: Hypertension
ICA	: Internal Carotid Artery
ICH	: Intracranial Hemorrhage
IJV	: Internal Jugular Vein
ICV	: Internal Cerebral Vein
IMA	: Internal Maxillary Artery
IOV	: Inferior Ophthalmic vein
IPS	: Inferior Petrosal Sinus
MMA	: Middle Meningeal Artery
OA	: Occipital Artery
APA	: Ascending pharyngeal Artery
SOV	: Superior Ophthalmic vein
VA	: Vertebral Artery
NBCA	: n Butyl Cyanacrylate
PVA	: Poly vinyl alcohol
CT	: Computed tomography
MRI	: Magnetic Resonance Imaging
DSA	: Digital Subtraction Angiography

DEPARTMENT OF IMAGING SCIENCES AND INTERVENTIONAL RADIOLOGY

**SREE CHITRA TIRUNAL INSTITUTE OF MEDICAL SCIENCES AND TECHNOLOGY
TRIVANDRUM, KERALA**

PROFORMA FOR EVALUATION & MANAGEMENT OF DURAL ARTERIOVENOUS FISTULAS

I) PATIENT DETAILS

Name:

Age/Sex:

Hospital No:

DOA:

DOD:

D/O PROCEDURE:

Occupation:

Address:

Phone:

E-Mail:

II) PRESENTING COMPLAINTS:

- a) Headache:
- b) Diplopia, redness, Proptosis:
- c) Diminished vision:
- d) LOC / Altered sensorium/Focal neurological deficits:
- e) Seizures:
- f) Dementia
- g) Bruit:

B. H/o Trauma:

C. Personal history:

HT / DM / MI / CAD / Stroke:

H/O recent delivery, oral contraceptive use or Hypercoagulable state:

Br Asthma / TB:

History of Allergies:

Smoking / Alcohol:

H/o prior hospitalization:

D. Family History:

Similar medical illness in family members:

E. Prior treatment history:

III) EXAMINATION:

A. GENERAL EXAMINATION:

Cutaneous stigmata:

Features suggestive of connective tissue disorder:

B. NEUROLOGICAL EXAMINATION:

Higher mental functions:

Cranial nerve deficits:

Motor deficits:

Sensory deficits:

Meningeal signs:

Cerebellar signs:

C. LOCAL EXAMINATION :

Signs:

Dilated episcleral vessels:

Diminished vision:

Elevated intra ocular pressure:

Chemosis:

Papilledema:

Third nerve palsy:

Sixth nerve palsy:

Other cranial nerve palsies:

Bruit:

D. OPTHALMOLOGICAL EXAMINATION:

Proptosis: U/L: B/L:

Visual acuity:

Visual field charting:

Pupils:

Intra ocular tension:

Fundus:

E. OTHER SYSTEM EXAMINATION :

Respiratory system:

Cardiovascular system:

Abdomen:

IV) INVESTIGATIONS:

Hb:	TC/DC:
ESR:	Platelets:
PT :	aPTT:
Bl Urea:	Sr. Creatinine:
LFT:	Lipid Profile:
Bl. Group:	HIV:
HBs Ag:	HCV:
VDRL:	Others:

V) IMAGING:

A. CT:

Date:

Dilated cavernous sinus:

Dilated SOV /IOV:

Proptosis:

Sinus thrombosis/ Empty delta sign:

Rule out intracranial hemorrhage:

Low density due to edema:

Rule out ear abnormalities such as aberrant vascular anatomy or glomus tumors:

Follow up CT:

Findings:

B. CT Angiography:

Date /Findings:

C. MR BRAIN:

Date /Findings:

Thrombosed or stenosed dural venous sinus:

Dilated cortical veins without a parenchymal nidus:

Prominent flow voids in the scalp:

Hyperintense signal changes in the white matter:

Other findings like thickened dural leaflet; hypertrophied pachymeningeal arteries dilated, tortuous, and variceal venous channels:

D. OPHTHALMIC DOPPLER:

Date/Findings:

E. USG ABDOMEN:

Date/Findings:

F. ANGIOGRAM:

a) Date: Day from ictus:

b) Access site:

CFA Right /Left / Bilateral Other Sites

Venous access CFV / SOV / IJV /ANGULAR VEIN/Burr hole

c) Side of fistula:

d) Site of fistula:

e) High /medium / low flow

f) Feeders:

ECA:

ICA:

VA:

g) Venous drainage:

Anterior Posterior Superior Inferior Contra lateral

Cortical rerouting Present / Absent

Venous ectasia Present / Absent

Spinal perimedullary venous drainage

Venous stenosis

Impaired / Delayed venous drainage

h) Other vascular anomalies / variations

VI) CLASSIFICATION:

COGNARD *et al.* CLASSIFICATION

	Cognard et al.
I	Venous drainage into dural venous sinus with antegrade flow
IIa	Venous drainage into dural venous sinus with retrograde flow
IIb	Venous drainage into dural venous sinus with antegrade flow and CVR
II a + B	Venous drainage into dural venous sinus with retrograde flow and CVR
III	Venous drainage directly into subarachnoid veins (CVR only)
IV	Type III with venous ectasias of the draining subarachnoid veins
V	Direct drainage into spinal perimedullary veins
	(CVR indicates cortical venous reflux)

VII) MANAGEMENT

A) ANAESTHESIA: Local / General anaesthesia

B) PROCEDURE DONE:

Time Taken:

- a) Transarterial feeder embolization:
- b) Transvenous coiling

Venous:

IPS / SOV / ANGULAR VEIN

- c) Material used:
- d) Guiding catheter:
- e) Guidewire:
- f) Microcatheter / Microwire:
- g) Others:

VIII) CHECK ANGIOGRAM:

Dural arteriovenous malformation completely closed:

Other findings:

IX) INTRA PROCEDURAL COMPLICATION & MANAGEMENT:

X) POST PROCEDURE:

Extubation

Cath lab

Elective ventilation

Reason for elective ventilation:

Post procedural neurological status:

Post procedural medications:

Details of ICU stay:

XI) FOLLOW UP:

XII) ADDITIONAL COMMENTS:

XIII) PERFORMING RADIOLOGIST:

ASSISTANT

CONSULTANT

SN	HN	NO	AGE	SEX	DM	HT	W	ISC	H	P	SZ	TNT	FND	CH	PA	CF	BR	MG	ANA	OC	MUL	FEL	SID	DIC	VRE	YP	DU	EME	NO	COM	FI	CO	SU	U	C	DSA
1	186944	V	2	0	0	0	1	0	0	1	0	1	0	0	1	0	1	0	1	3	0	4	2	2	1	0	4	2	2	1	1	2	4			
2	187538	III	1	0	0	0	1	1	0	0	0	0	0	1	0	0	1	4	1	2	0	4	3	2	1	0	4	5	7	2	0	0	1	1	1	
3	184994	VI	1	0	1	0	1	1	0	0	0	0	0	1	0	0	1	0	0	6	0	3	2	2	0	0	2	0	6	1	0	0	1	1	2	
4	188046	V	1	1	0	0	0	0	0	0	0	1	0	0	0	0	6	1	2	0		2	2	1	0	4	3	7	5	1	1	4				
5	189232	IV	1	0	0	0	1	1	0	1	0	1	0	0	1	4	0	6	0	3	2	2	0	0	2	2	2	2	1	0	6	1	1	1		
6	192670	VI	2	1	0	0	0	1	0	1	0	0	0	0	1	0	1	3	1	3	3	3	0	0	2	0	6	1	0	0	1	1	2			
7	197523	IV	1	0	0	0	1	1	0	1	0	0	0	1	1	0	1	3	0	3	1	2	1	0	3	5	7	2	0	6	1	1	1			
8	198533	IV	1	0	0	0	0	1	1	0	0	0	0	0	1	0	0	1	2	0	4	3	2	1	0	4	5	7	2	0	0	1	1	2		
9	208640	VI	2	0	0	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	1	2	1	1	6	5	7	3	0	6	1	1	1			
10	204219	VI	2	0	1	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	0	0	2	2	2	2	1	0	6	1	1	1		
11	209222	VI	2	1	1	0	1	1	0	0	0	1	1	1	1	4	0	6	0	3	1	2	0	0	2	2	2	2	1	0	0	1	1	1		
12	211232	V	2	0	0	0	1	1	0	0	0	1	1	1	0	4	0	6	0	3	2	2	0	0	2	0	6	1	0	1	1	1	1			
13	212196	III	1	0	0	0	1	1	0	1	0	0	0	1	1	5	1	3	0	3	1	2	1	0	4	5	7	2	0	0	1	1	3			
14	212820	IV	1	0	0	0	0	0	0	1	0	0	0	0	1	7	0	4	0	4	1	1	0	0	1	1	1	1	0	0	1	1	2			
15	214421	VI	1	0	1	0	0	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	0	6	1	1	2			
16	215659	IV	1	0	0	0	1	0	0	0	0	0	0	0	0	7	0	4	0	3	1	1	0	0	1	1	3	1	0	6	1	1	2			
17	239255	V	1	0	0	0	1	0	0	0	0	0	0	0	0	7	1	3	0	4	1	2	1	1	6	5	7	2	0	0	1	1	2			
18	241243	VII	2	1	0	0	0	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	0	0	2	2	2	2	1	0	6	1	1	1		
19	242441	VI	1	0	1	0	1	1	0	0	0	0	1	1	1	3	0	3	0	3	1	2	1	1	4	5	7	1	0	0	1	1	1			
20	268914	III	2	0	0	0	1	1	0	0	0	1	1	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	0	6	1	1	2			
21	270568	IV	1	0	0	0	0	1	0	0	0	1	0	0	0	4	0	6	0	3	2	2	0	0	2	2	2	1	0	6	1	1	1			
22	274355	VI	1	1	1	0	1	1	0	0	0	1	0	1	0	4	1	6	0	3	2	2	0	0	2	6		2	0	6	2	2	3			
23	279692	VI	2	1	0	0	1	1	0	0	0	1	0	0	0	4	0	6	0	3	1	2	1	0	3	5	7	2	0	6	1	1	2			
24	244113	VI	1	0	1	0	0	0	1	0	0	0	0	0	0	8	1	3	0	3	1	2	1	0	4	2	2	1	0	6	1	1	1			
25	245845	IV	2	0	0	1	1	0	0	0	0	0	1	0	0	6	1	5	0	4	3	2	1	0	4	5	7	3	0	0	1	1	1			
26	266975	VI	1	1	0	0	0	0	0	0	1	0	0	0	0	8	1	3	0	3	1	2	1	0	3	5	7	2	0	0	1	1	2			
27	285735	VII	2	0	0	0	1	0	0	0	0	1	0	0	0	5	0	6	0	3	2	2	1	0	4	1	1	1	0	6	1	1	2			
28	267485	III	2	0	0	1	1	1	0	0	0	1	1	1	0	4	0	6	0	3	1	2	0	0	2	5	7	2	0	6	1	1	1			
29	267922	IV	1	0	0	1	1	1	0	0	0	1	0	1	0	1	1	3	0	3	1	2	1	1	6	5	7	1	1	1	4					
30	268675	V	1	0	1	0	0	0	1	0	0	0	0	0	0	1	1	3	0	3	1	2	0	0	2	5	7	2	0	0	1	1	2			
31	279918	VI	2	0	0	0	0	1	0	0	0	1	0	1	0	4	0	6	0	3	1	2	1	0	4	1	1	1	0	6	2	2	3			
32	281044	VII	1	1	1	0	1	1	0	0	0	0	1	1	1	8	0	3	0		1	2	1	0	4	1	1	1	0	0	2	1	2			
33	281196	VII	2	1	0	0	0	1	0	0	0	0	0	1	0	4	0	6	0	3	2	2	0	0	2	0	6	1	0	6	2	1	3			
34	281457	VI	1	0	0	0	1	1	0	0	0	0	1	0	0	8	1	3	1	4	1	2	1	0	3	5	7	3	0	6	1	1	2			
35	284001	VI	1	1	0	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	1	0	4	1	1	1	0	6	1	1	2			
36	1E+07	IV	1	0	0	0	0	0	0	0	0	1	0	0	0	0	1	1	2	0	5	2	1	1	1	6	2	2	1	0	6	1	1	2		

37	1E+07	IV	1	0	0	0	1	0	0	0	0	0	0	0	8	0	3	0	3	2	1	0	0	1	0	6	1	0	6	1	1	2
38	27658	V	1	0	0	0	1	0	0	0	1	0	1	0	0	7	1	4	0	3	2	2	1	0	4	5	5	3	1	3	4	
39	185601	VI	1	1	0	0	1	1	0	0	0	1	1	1	0	4	0	6	0	3	2	2	0	0	2	0	6	1	0	6	2	
40	267175	VI	1	1	0	0	1	0	0	0	0	0	1	0	0	1	1	3	0	3	2	2	1	1	6	2	2	1	0	6	1	1
41	266202	III	2	0	0	1	0	0	1	0	1	0	0	0	0	8	1	3	0	3	1	2	1	0	3	1	4	1	0	6	1	1
42	265998	V	2	1	0	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	1	2	0	0	2	5	7	1	0	6	1	1
43	264484	III	1	0	0	0	1	0	0	0	0	0	0	0	0	2	1	3	0	3	2	2	1	0	4	1	4	1	0	6	1	1
44	264053	VI	2	0	0	0	0	1	0	0	0	1	0	1	0	4	0	6	0	3	1	2	0	0	2	1	1	1	0	6	1	1
45	262250	VIII	2	1	1	0	1	1	0	0	0	1	1	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	1	0	3	1
46	261613	VI	2	0	0	0	0	1	0	0	0	1	0	1	1	4	0	6	0	3	2	2	0	0	2	1		1	1	0	2	1
47	261446	VI	1	1	0	0	1	1	0	0	0	1	1	1	0	4	0	6	0	3	1	2	0	0	2	0	6	1	0	6	2	1
48	260347	VI	1	0	0	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	0	0	2	1	7	1	0	6	1	1
49	259737	VI	1	0	0	0	0	0	1	0	0	0	0	0	1	8	1	3	1	4	3	2	1	0	4	5	7	3	0	6	1	1
50	257372	III	2	0	0	1	1	1	0	0	0	0	1	0	0	7	1	3	0	4	2	2	1	0	3	5	7	2	0	6	1	1
51	254613	III	2	0	0	0	1	1	0	0	0	0	1	1	0	3	1	3	0	3	1	2	1	0	4	2	2	1	0	6	1	1
52	252298	VII	2	1	1	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	1	2	0	0	2	1	1	1	0	6	1	1
53	251549	IV	2	0	0	0	0	1	0	1	0	1	1	1	1	4	1	6	0	3	1	2	1	0	4	1	7	1	0	6	1	1
54	251400	IV	2	0	0	0	1	1	0	0	0	0	1	0	0	1	1	1	0	5	1	1	1	0	5	1	5	1	1	5	3	3
55	251130	VII	1	0	0	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	0	6	1	1
56	249278	V	1	0	0	0	1	1	0	1	0	0	1	1	1	4	0	6	0	3	1	1	1	0	5	1		1	1	0	2	1
57	249026	V	1	0	0	0	1	0	0	0	0	0	1	0	0	1	1	1	0	3	1	1	1	0	5	1	3	1	1	0	2	1
58	248972	II	1	0	0	0	0	0	0	0	1	0	0	0	0	1	1	2	0	3	3	2	1	1	6	1	5	1	1	7	4	
59	248619	IV	2	0	0	0	1	0	0	0	0	0	1	0	0	8	1	7	1	4	3	2	1	0	4	5	7	3	0	0	1	1
60	225564	VI	1	1	0	0	1	0	0	0	0	0	0	0	0	1	0	3	0	3	1	2	1	0	3	5	7	1	0	6	1	1
61	225747	V	2	0	0	1	1	1	0	1	0	1	1	1	1	4	1	6	0	4	1	2	1	0	4	5	7	1	0	6	1	
62	226208	III	1	0	0	0	0	0	1	1	0	0	1	0	0	1	1	7	1	5	3	3	1	0	5	5	7	1	0	0	1	1
63	232740	VII	1	0	0	0	0	1	0	0	0	1	1	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	1	5	3	3
64	234155	V	1	0	0	0	1	0	0	0	0	0	0	0	0	1	0	3	0	3	1	3	1	0	5	1	1	1	0	6	1	
65	234706	VII	1	1	1	0	1	1	0	0	0	1	1	1	0	4	0	6	0	3	1	2	0	0	2	2	2	1	0	6	1	1
66	234752	VII	2	0	0	0	0	1	0	0	0	1	1	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	0	6	1	1
67	234988	VII	1	0	1	0	1	0	0	0	1	0	1	0	0	2	1	3	0	4	2	1	0	0	7	1		1	1		4	
68	235650	II	1	0	0	0	1	1	0	0	0	1	0	1	0	1	0	6	0	3	1	3	1	1	6	1	1	1	0	6	1	
69	238079	IV	1	0	0	1	0	0	0	0	1	0	0	0	0	1	1	3	0	4	1	3	1	0	5	5	7	1	0	6	1	1
70	238648	VIII	1	0	0	1	0	1	0	0	0	1	0	1	0	4	2	6	0	3	1	2	0	0	2	5	7	1	0	6	1	1
71	238770	VI	2	0	0	0	1	1	0	0	0	1	0	1	0	4	0	6	0	3	2	2	0	0	2	1	1	1	1	5	3	3
72	238864	VI	1	0	1	0	1	1	0	0	0	0	1	0	0	2	1	3	0	4	2	3	1	1	6	1	3	1	1	0	3	
73	232207	V	1	0	0	0	0	1	0	0	0	0	1	1	1	1	1	4	0	4	1	2	1	1	6	2	2	1	0	6	3	3

3 NC H NO AGESEX

I 1 - 9 MALE

II 10 19 FEMALE

III 20-29 YRS

IV 30-39 YRS

V 40-49 YRS

VI 50-59 YRS

VII 60-69 YRS

VIII 70-79 YRS

IX 80-89 YRS

IMAGIISTHLO MULTISIDEDR/CVRS EYPDU LIC / EMP LIC / CONESUI / UP DSA
0 NORMAL OC 1 S0 ABSERIGII ARESIES 1 1 ER' PVA 1 RESEORMOVE OVD FIST
1 ICH AN 2 T 1 PRES LEF RCBSEBSI 2A TACOII 2 ABSE SAISANSAND FIS
2 SAH 3 TRANS BOTH BOTH 3 2E TV COI 3 2 IVIRIOIRIOI RESI
3 SINUS THROMI 4 SIGM ECA 4 2ARR IONY 4 3 ICHAIN DRSENEED
4 DILATED SUPERIOR OP 5 STR2 ICA 5 33 CIGLL 5 4 SDH
5 ENLARGED CORTIC 6 CAVEA + ICA 6 4 MB COM 6 5 INFARCT
6 FISTULA 7 MECA, ICA, VA 7 5 AILIBINATION NOT DONE
7 NARROWING OF SINUS, PIAL FEEDERS 7 OBSTRUCTIVE HYDROCEPHALL
8 FLOW VOIDS

KEY TO MASTER CHART

- 1)-Column 1- S No – Serial number
- 2)-Column 2 – H No –Hospital number
- 3)-Column 3 – Age
- 4)-Column 4 –Sex, 1- Male; 2- Female
- 5)-Column 5 – DM – Diabetes mellitus, 0 – Absent, 1 - Present
- 6)-Column 6 – HT – Hypertension, 0 – Absent, 1 - Present
- 7)-Column 7 - MISC – Miscellaneous risk factors, 0 – Absent, 1 - Present
- 8)-Column 8 - H – Headache, 0 – Absent, 1 - Present
- 9)-Column 9 – DPV – Diplopia, Proptosis, Visual field defects, 0 – Absent, 1 - Present
- 10)-Column 10 – SZ – Seizures, 0 – Absent, 1 - Present
- 11) Column 11- TNT – Tinnitus, 0 – Absent, 1 - Present
- 12)-Column 12- FND – Focal neurological deficits, 0 – Absent, 1 - Present
- 13)-Column 13- CHM – Chemosis, 0 – Absent, 1 - Present
- 14)-Column 14- PAP – Papilledema, 0 – Absent, 1 - Present
- 15)-Column 15 – CNP – Cranial nerve palsy, 0 – Absent, 1 - Present
- 16)-Column 16- BR – Bruit, 0 – Absent, 1 - Present
- 17)-Column 17 – IM – Imaging, 0 – Normal, 1 – ICH, 2 – SAH, 3 – Sinus thrombosis, 4 – Dilated superior ophthalmic vein, 5 – Enlarged cortical veins, 6 – Fistula, 7 – Narrowing of sinus, 8 – Prominent flow voids.
- 18)-Column 18- ANA – Anaesthesia, 0 – Local, 1 – General anaesthesia
- 19)-Column 19 – LO – Location, 1 – Superior sagittal sinus, 2 – Torcula, 3 – Transverse sinus, 4 – Sigmoid sinus, 5 – Straight sinus, 6 – Cavernous sinus, 7 – Multiple sinuses.
- 20)-Column 20- MU – Multiplicity, 0 – Absent, 1 - Present
- 21)-Column 21- FE – Feeders, 1 – ECA, 2 – ICA, 3 – ECA + ICA, 4 - ECA + ICA + VA, 5 - ECA + ICA + VA + Pial feeders.
- 22)-Column 22- SID – Side, 1 – Right, 2 – Left, 3 – Both sides.
- 23)-Column 23 – SD – Sinus drainage, 1 –Antegrade, 2 – Reterograde, 3 – Both
- 24)-Column 24- CVR – Cortical venous reflux, 0 – Absent, 1 - Present
- 25)-Column 25 –VE- Venous ectasia, 0 – Absent, 1 - Present
- 26)-Column 26- TY – Type, 1 – I, 2 – IIA, 3 – IIB, 4 – IIA+B, 5 – III, 6 – IV, 7 – V.
- 27)-Column 27- RO – Route, 0 – No interventions, 1 – Trans arterial, 2 – Transvenous, 3 – Burr hole, 4 – Venous cut down, 5 – Combined, 6 – Failed.
- 28)-Column 28 – EMB –Embolic agent, 1 – PVA, 2 – Coils, 3 – Alcohol, 4 – Onyx, 5 – Glue, 6 – Carotid compression, 7 – Combination.
- 29)-Column 29 – NO – Number of sitting of embolization, 1, 2, 3, 4, 5, 6, 7, 8.
- 30)-Column 30 – CO – Complication, 0 – Absent, 1 - Present
- 31)-Column 31-IM CO –Imaging of complication, 0 – Normal, 1 –SAH, 2 – IVH, 3 –ICH, 4 – SDH, 5 – Infarct, 6 – Not done, 7 – Obstructive hydrocephalus.
- 32)-Column 32- RE –Result, 1 –Improved, 2 – Same, 3 –Deteriorated, 4 – Brain dead. 33)- Column 33- FUC –Follow up clinical, 1 –Improved, 2 – Same, 3 –Deteriorated.
- 34)-Column 34 - FUDS –Follow up DSA, 1 –No fistual, 2 – Mild low flow fistula, 3 – Significant residual fistula, 4 – Worsened grade.