

**Dural Carotid Cavernous Fistulas- Clinikoradiologic  
Spectrum and Endovascular Management**



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## **DECLARATION**

I hereby declare that this thesis titled “Dural Carotid-Cavernous Fistulas-Clinicoradiologic Spectrum and Endovascular Management” has been prepared by me under the supervision and guidance of Dr Jayadevan ER, (Assistant Professor), and Dr. Kapilamoorthy T R, (Professor & Head), Department of Imaging Sciences and Interventional Radiology, Sree Chitra Institute for Medical Sciences and Technology, Thiruvananthapuram.

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# *Introduction*

Carotid cavernous fistulas (CCFs) are abnormal communications between the carotid artery and the cavernous sinus. Angiographically, these fistulas are typically divided into direct and indirect types of arteriovenous shunts. Direct CCF is a communication between the cavernous segment of the internal carotid artery and the cavernous sinus, usually due to trauma or ruptured aneurysm. Indirect or Dural CCF is an acquired communication between dural (meningeal) branches of the internal or external carotid arteries through the wall of the cavernous sinus. The clinical manifestations may overlap, although the natural history and method of endovascular treatment are often significantly different.

Dural CCFs have been classified by Barrow et al (1) into Type B, C and D based on the arterial supply by dural branches of External Carotid Artery (ECA), Internal Carotid Artery (ICA) or both. Because of their vascular supply by dural arteries, Dural CCFs have been categorized by some authors into the large group of dural arteriovenous fistulas. However Cavernous sinus dural fistulas are different in several aspects from Dural Arteriovenous fistulas (DAVF) involving other dural sinuses. Anatomically, Cavernous sinus (CS) is an extradurally located sinus, whereas other dural sinuses are located between 2 dural walls in the cranial cavity. Postmenopausal females more commonly experience Dural CCF than males experience transverse sinus DAVF, thereby suggesting the possibility of a hormonal influence. In addition, the clinical symptoms and signs are known to be benign because Cavernous Sinus has sufficient venous drainage routes. Because the symptoms are very diverse and they fluctuate, analysis of the symptomatology related to the angiographic findings does not always correspond to the disease status.

Current Classification of dural CCF by Barrow et al is based mainly on the arterial angioarchitecture and does not reflect the venous drainage pattern. Variations of venous drainage in CS have not been fully described in patients with Dural CCF. Suh et al(2) have proposed a classification based on the AV fistula and the venous drainage into three types : Proliferative type, Restrictive type and Late restrictive type. The authors also correlated the presenting symptoms with the venous

drainage patterns and divided the symptoms into the orbital, cavernous, ocular and cerebral pattern.

The primary treatment modality for dural CCF is endovascular with either transarterial embolization of ECA feeders by PVA particles/ liquid embolic agents or transvenous embolization using coils, NBCA or Onyx.

Available data in this regard is mostly western, with no available data describing the angiographic classifications involving the venous drainage pattern of dural CCF. Management strategies also differ as economic and technical considerations are different. Even though there has been increasing use of transvenous approach for the management of dural CCF in the west, embolization using transarterial approach has been successful in alleviating the patient's symptoms. This study will try to analyze these issues by assessing the clinical features, symptom pattern, angiographic features of dural CCF and to evaluate the radiologic & clinical outcome in patients treated with embolization.

# *Aims and Objectives*

The objectives of the study were as follows:-

1. To describe the angiographic types of dural CCF.
2. To assess the clinical features and symptom pattern in patients with dural CCF.
3. To evaluate radiologic & clinical outcome in patients treated with embolization.

# *Review of Literature*

## CLASSIFICATION OF DURAL CCFs

The first angiographic classification of carotid cavernous fistulas was by Wolf and Schmid (3). They described four main types of venous drainage Type A –anterior drainage via SOV, Type B-Posterior drainage via IPS and SPS, Type C-Cortical drainage via the vein of Trolard, Type D-Deep venous drainage via the perimesencephalic vein and basal vein of Rosenthal.

It was Parkinson in 1965 who first made the distinction between direct and indirect fistulas (4). He considered the first type a single fistula resulting from a tear in the wall of the carotid and the second type resulting from a tear across one of the small dural branches within the cavernous sinus.

Newton and Hoyt in 1970 subdivided DCCFs into two types. The first was characterized by a shunt adjacent to or within the wall of the CS and

the second by a shunt involving more distant dural sinuses that communicate with the CS (5)

Ferland in 1982 and Moret et al. in 1978 classified the indirect fistulas further into two types: Type I fistulas, fed by a complex network of feeders whose systematization is impossible and Type II fistulas in which all the feeders can be precisely identified(6,7).

Barrow et al. in 1985 developed a more detailed classification for spontaneous cavernous sinus fistulas(1). This schemata, until recently was the mainstay of classification, allowing differentiation of fistulas according to the type of arterial supply. According to this classification the CCFs were classified as follows

Type A - Direct fistulous communication between the cavernous segment of the internal carotid artery and the cavernous sinus, usually due to trauma or ruptured aneurysm.

Type B- Indirect fistula with feeders from dural branches of ICA (like Inferolateral trunk (ILT), Meningohypophyseal trunk (MHT))

Type C- Indirect fistula with feeders from dural branches of ECA (Middle meningeal artery (MMA), Accessory Meningeal artery (AMA), Ascending pharyngeal artery (APA), Internal maxillary artery (IMA)

Type D – Indirect fistula with feeders from dural branches of ICA and ECA

This classification provided prognostic value and had some impact on therapeutic decision making.

Barrow's classification specifically proposed for spontaneous lesions is based only on the arterial angioarchitecture and does not reflect the venous drainage pattern. It allows differentiation with regard to technical difficulties anticipated during transarterial catheterization, which was the most common endovascular approach in the 1980s. Under those aspects, Type C lesions were usually considered "easier to treat" because catheterization of ECA feeders was technically less difficult and injecting liquid embolic agents was clinically less risky. Types B and D fistulas were, on the other hand, seen as more challenging lesions (and still are by some authors), and are often considered "difficult to treat lesions" or "intractable fistulas".

Because of the increasing experience and technical advancement in using transvenous techniques in the 1990s, the technical challenges of transarterial embolizations are meanwhile of little importance. Thus, the type of arterial supply (B, C or D) has only minor impact on whether or not to perform a transvenous occlusion.

Some fistulas are located only on one side, but are supplied by bilateral feeders from the ICA and the ECA. Because of the usually associated higher flow, these fistulas have been considered by Tomsick and Barcia-Salorio et al. as more complex and more difficult to treat and were classified as Type D1 and Type D2 fistulas (8,9).

Stiebel-Kalish et al. in 2002 have studied the venous drainage pattern of dural CCFs and differentiated them into four types (10).

1. Venous outflow into anterior CS and SOV was present in 77/85 patients. In this group cortical veins were present in 25%, Inferior petrosal sinus (IPS) in 21% and Superior Petrosal Sinus (SPS) in 12%.
2. Abnormal ophthalmic venous flow with ophthalmic vein thrombosis was seen in 11/85 among which cortical veins were found in 73%, IPS in 27% and SPS in 27% of the cases.
3. IPS drainage was seen in 22 cases with 41% of cortical drainage, 18% SPS drainage and paraspinal veins (4.5%).
4. SPS drainage was seen in 11 cases with cortical drainage in all of them (100%). Suh et al. in 2005 have suggested angiographic differentiation of DCCFs into three types (2):
  1. The Proliferative type (PT):
    - -Numerous arterial feeder to the CS (network)
    - -Large AV shunt with rapid filling of CS, afferent and efferent veins
    - -Both CSs completely filled and bulging into the sinus wall
  2. The Restrictive type (RT):
    - -Less arterial feeders than PT, each identifiable
    - -Obliteration of flow in IPS, increased flow in SOV and cortical veins
    - -Less AV shunt than in PT
    - -CS margins less well defined (loss of normal contour)
  3. The Late Restrictive type (LRT):
    - -Few arterial feeders
    - -With sluggish retrograde venous flow
    - -Constrictive changes of the veins
    - -CS stasis

The authors were able to correlate presenting symptoms in 58 patients with these three drainage patterns. Patients with a PT fistula would mostly present with a

cavernous pattern such as ptosis, diplopia, anisocoria and ophthalmoplegia caused by CN deficits. Patients with a RT fistula presented with a cavernous symptom pattern or with an orbital (chemosis, exophthalmos, periorbital pain, eyelid swelling) or ocular pattern (decreased vision, increased IOP, severe ocular pain, glaucoma and retinal hemorrhage). Patients with a fistula of the LRT presented mostly with ocular pattern. A cerebral pattern with infarction in the basal ganglia or brainstem caused by reflux into cortical veins was seen in 5% of the patients with RT and LRT fistulas.

Satomi et al. (2005) in their study of 65 patients, staged the progression of the disease into three phases based on the venous drainage pattern(11).

Stage 1 fistulas drain predominantly posteriorly via the IPS and to a lesser degree anteriorly, producing mainly tinnitus.

Stage 2 fistulas are characterized by closed (thrombosed) IPS and PP redirecting the flow into the SOV and the IOV and to a lesser degree into the Sylvian vein. In this stage the patients present not with bruit but with increased ophthalmological symptoms.

Stage 3 is defined as a fistula that drains exclusively into either the Sylvian vein or the SPS due to the occlusion of initially anterior and posterior draining veins.

## **ETIOLOGY AND PATHOGENESIS OF DURAL CCF**

Despite major advances in diagnosis and management, the etiology and pathogenesis of DCCFs remains a matter of controversy and is far from being fully understood.

### **Pregnancy**

Pregnancy and childbirth have long been known predisposing factors for CCFs since their descriptions by Dandy (1937) and others (Walker and Allegre, Sattler) (12, 13, 14). Sattler, in his first review of cases reported up to 1880, had collected 32 idiopathic cases, among which 23 were women, 6 of whom were pregnant, while 1 developed the fistula during delivery (15). Sattler also described cases that occurred under certain circumstances during physical stress such as coughing, at passing stool,

strenuous work or severe vomiting. More recent statistics show that only 28% of the fistulas occur during puerperium, whereas 54% develop during menopause (8).

Physical stress during delivery may cause rupture of small dural vessels and development of DCCFs. In one of the largest series published so far by Meyers et al. in 2002, the onset of symptoms was associated in 6% of cases with pregnancy (16).

### **Hormonal Factors**

Tanaguchi et al. described predisposition in women not only during but also after pregnancy (17). The higher incidence of DCCFs in women during menopause and in men over 50 years of age also supports the theory of a tear of an arteriosclerotic altered vessel wall. On the other hand, it indicates that hormonal factors may contribute and possibly represent an important etiological factor for DCCFs occurring more frequently in elderly women. The exact underlying mechanisms remain unclear. Endogenous estrogen is responsible for inhibiting the progression of arteriosclerosis in women, while exogenous estrogen may promote thrombosis. It is possible that lack of endogenous estrogen affects the integrity of the dural micro shunt regulation and can promote the development of macro shunts (18).

### **Thrombosis**

It was Tanaguchi et al. in 1971 who recognized early on the potential of the minute vascular dural network when reacting to a thrombosing fistula between ICA and CS(17). They suggested fresh thrombus as initial stimulus for building a fistula, a concept widely accepted today. That thrombosis in sinuses involved by DCCFs occurs, was observed by Voigt and coworkers and was reported by Brismar and Brismar (1976) who documented the presence of thrombus in six patients using orbital phlebography(19). Houser et al. (1979) suggested that a DCCF might develop subsequently to a thrombus in the CS that induces an “arteriolar neoformation” and represents the trigger for the development of an arteriovenous shunt(20). Most authors share this opinion today. Although angiographically sometimes difficult to identify with certainty, a thrombus in the CS has been observed by some authors in up to 85% of the cases (Theaudin et al)(21).

Mironov (1994) has provided the largest body of work on this subject so far(22). He studied extensively the pathogenesis in 96 DAVFs in all locations and found overall concomitant phlebothrombus in more than 50%, while thrombus was seen in 62% of the 29 DCCFs. It is assumed that thrombus undergoes organization and recanalization, which triggers the opening of latent preexisting AV communications. When a fistula shunts into the sinus, it will increase the venous pressure and open more AV shunts, by some authors being considered a vicious cycle (Nishijima et al.)(23).

Tsai et al. (2004) studied 69 patients with DAVF in different locations and found in 39% associated cerebral sinus thrombosis(24). Thrombosis was found always in the sinuses around to the fistula in the sinuses downstream to the fistulas which supports the two main hypothesis, that (1) venous outflow obstruction (by thrombus) may cause opening of physiologic arteriovenous shunts or (2) venous hypertension causes ischemia and angiogenesis (3) the theory that thrombosis may be caused or augmented by the turbulent fistula flow as suggested by some authors (Chaudhary et al. , Nishijima et al. , Lawton et al. , Lasjaunias et al.) is supported as well(23,25,26,27). Areas of low flow velocity or flow stagnation found in sinuses where two parts of flow antegrade cerebral blood flow and retrograde fistula flow meet, could create such hemodynamic condition. Although this potential mechanism is not well studied, it may play a role as a trigger for sinus stenosis.

### **Venous hypertension**

The concept of venous hypertension, being another key factor in the etiology of DAVFs, is encouraged by results of animal experiments conducted by Terada et al. (1994), who demonstrated that increased venous pressure can cause newly acquired AVFs, even in absence of thrombosis (28).

It can be suspected that a similar mechanism may also play a role in humans (29). A possible causal relationship between hypertension and arteriovenous shunts at the CS has already been assumed by Potter (30) as well as by Echols and Jackson(31), who suggested that creating hypotension might be beneficial for causing spontaneous thrombosis of a CCF. There are anecdotal reports that symptoms and signs caused by

a CCF have disappeared after air travel (32, 33). Changes of atmospheric pressure seem to interfere with pressure in the cerebral venous system. They may have a bi-directional effect on the arteriovenous shunt flow, causing either an increase, or a decrease with spontaneous occlusion.

Lawton et al. (1997) were able to demonstrate a causal relationship between venous hypertension and angiogenic activity and DAVF formation (26). The authors suggested that venous hypertension is induced by a venous outflow obstruction due to a thrombus and may initiate the pathogenesis of a DAVF. Venous hypertension can cause ischemia and tissue hypoxia that may stimulate angiogenesis. This “aberrant” angiogenic activity of dural vessels could lead to arteriovenous shunting and formation of a DAVF. The subsequent arterialization will increase venous pressure and outflow obstruction and thereby create the vicious cycle mentioned above that may enlarge the AV shunt and aggravate a DAVF into a progressive lesion.

### **Trauma**

Trauma, although reported in some anecdotal cases, is probably less likely a cause of a DCCF. Tomsick gave an overview that revealed approximately 3% of DCSFs are related to trauma (8). Iatrogenic vessel injury during endovascular procedures may lead to AV shunts involving the CS and can be caused by transsphenoidal surgery of pituitary adenomas(34,35). Catheterization of cavernous dural ICA branches for embolization of meningiomas may cause rupture and lead to an “indirect” CCF as well (36).

### **Other factors**

Recently, the role of basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF) has been studied (28,37).

The higher incidence of DCCFs in women during menopause and in men over 50 years indicates a potential role of vessel wall weakening over time due to arteriosclerotic changes. Arterial hypertension and diabetes are considered predisposing factors as well, but correlative data are lacking so far.

## **PREVALENCE OF DURAL CCFs**

Epidemiologic data regarding the overall incidence of both DAVFs as well as of DCCFs are limited. Because similar diagnostic and therapeutic measures apply to all dural fistulas, DCCFs and DAVF can be considered as one epidemiologic group of disease. For both, the association with menopause (54%) and puerperium (18%) and lower incidence in men is typical (5, 8, 38).

In a metaanalysis published by Awad et al.(39) in 1990, with an additional 17 cases, the incidence of AVFs in the CS among all DAVFs was 11.9%, whereas the relationship of “aggressive” to “non-aggressive” cases was 1:6.5. The true prevalence of DCCFs is difficult to assess due to the fact that many patients present with mild symptoms, may undergo spontaneous resolution and are never diagnosed. According to series of several major endovascular centers, Type B–D DCCFs occur five times more frequently than Type A fistulas (8). Another difficulty in obtaining accurate numbers lies in the fact that many DCCFs patients are studied as different groups, either included in DAVFs, CCFs or evaluated as a separate entity. The series of Cognard et al. (1995) contained 205 cases of DAVF of which 33 were DCCF (16%) (40). Klisch et al. (2003) reported on 17 CCFs, including 11 DCCFs (41). Satomi et al. (2002) reported on 117 cases of benign DAVF (without cortical venous drainage) with DCCF representing the largest group (42.7%) (11). Tsai et al. (2004) investigated 69 patients with DAVFs among whom 30% involved the CS (24). Chung et al. (2002) found the CS as the most common (57%) location for the DAVF in 60 patients (42). Tomsick observed Type B–D fistulas in 68% of all spontaneous CCF (8).

## **NATURAL HISTORY OF DURAL CCFs**

As true for prevalence and incidence of DCCFs, exact data on natural history do not exist, which is in part due to the fact that a large number of fistulas is discovered relatively late in their course. Furthermore cases undergoing diagnostic angiography are necessarily affected by the angiographic procedure itself as contrast injection can accelerate thrombosis of the CS and “spontaneous occlusion”

(5,43,44,45). In some recent series (Satomi et al, Suh et al) the natural course is additionally influenced by particulate arterial embolization (2,11).

The number of reported “spontaneous” occlusions reported in the literature may lie between 11% and 90% (33, 46) and is on average 35% according to Tomsick (8). Suh et al. have retrospectively studied the evolution of fistulas over a mean follow-up period of 23 months(2). They found that seven (30%) of their patients, angiographically classified as proliferative type (PT), showed chronological progression to the late restrictive type (LRT).

As for DAVFs, presence of leptomeningeal drainage is a main indicator for risk assessment and decision-making in patients with DCCFs by most authors. The number for cortical or leptomeningeal reflux varies from 10% to 31%. Nevertheless, the associated risk of intracranial hemorrhage (around 2%) seems, however, relatively low compared to patients with DAVFs in other locations, especially at the tentorial sinus or in the anterior cranial fossa (47). Consequently, cortical or leptomeningeal drainage in DCCFs must not necessarily be seen an indicator of a progressive or “malignant” course or nature, as in DAVFs.

## **CLINICAL FEATURES IN DURAL CCFs**

### **Extraorbital ocular symptoms**

#### **Orbital pain**

Persistent frontal or periorbital cephalgia of varying intensity, often starting acutely, is a frequent initial symptom in patients with DCCF. These headaches are usually caused by local thrombosis within the CS or the SOV. Furthermore, hemodynamic turbulences in the fistula leading to painful irritation of the meninges can increase during physical exercise or elevated blood pressure.

Extraocular or extraorbital symptoms beside headaches are rare. Anecdotal reports of life-threatening epistaxis or intracerebral hemorrhages in case of leptomeningeal venous drainage (48) or ischemic brain stem infarcts after sinus venous thrombosis (49) and atypical trigeminal neuralgia (50) exist. Fistulous connections in the posterior CS may cause mechanical compression of the Gasserian

ganglion or vascular steal phenomena affecting in particular the first or second division, resulting into neuralgia and dysesthesia (51, 52). They can initially occur isolated and thus may easily be mistaken for idiopathic trigeminal neuralgia (50, 53). Patients with periorbital cephalgia and accompanying ophthalmoplegia, initially misdiagnosed as suffering from migraine, cluster headaches, Tolosa-Hunt syndrome or intracranial aneurysms, have been described as well (54, 55,56).

### **Cranial nerve deficits and ophthalmoplegia**

Unilateral ophthalmoplegia can be seen in about 50% of the patients with DCCFs and often represents the first objective symptom (57). It usually becomes manifest after weeks or months following development of a fistula. The elevated intraorbital venous pressure leads to a progressive swelling of the ocular muscles and to a reduced contractility and limited motility of the eye bulb. On the other hand, dilated vessels and vascular steal phenomena result in mechanical and ischemic oculomotor nerve damage.

Due to their course through the CS, the sixth CN is most frequently (46%–85%), the third CN less frequently (36%) and usually in cases with posterior drainage, and the fourth CN (11%) is rarely involved (33). In cases of anterior drainage, the ophthalmoplegia is often accompanied by other orbital symptoms such as exophthalmos and chemosis. In contrast, fistulas with posterior drainage via the IPS may be the cause for isolated ophthalmoplegia and should be included in the differential diagnoses of intracranial neoplasms, cavernous aneurysms and meningitis (58).

### **Orbital Symptoms**

#### **Exophthalmos**

As a result of the chronically elevated intraorbital venous pressure a prolapse of the orbital soft tissue may develop, which is commonly less prominent than in patients with direct CCFs. In most cases it is less than 5 mm and can initially be overlooked.

A massive exophthalmos, rarely observed in cases of DCCF, can lead to chemosis, lid swelling, lagophthalmos and corneal damage. If an acute increasing exophthalmos, associated with pain and lid swelling occurs and is followed by a spontaneous improvement, a thrombosis of the SOV or the CS should be considered.

### **Conjunctival engorgement and chemosis**

Typically, arterialization of conjunctival veins is associated with other ophthalmologic manifestations, particularly with exophthalmos, and can be found in 82%–100% of patients (33,45,52) with intraorbital symptoms. It may lead to dilatations and tortuosities of conjunctival veins, which are often the cause for misdiagnosis such as inflammatory conjunctivitis. However, the conjunctival injection in patients with CCF is different from allergic, viral or bacterial conjunctivitis, characterized by bright-red, corkscrew veins. Except for cases of secondary infections, it usually occurs without inflammatory secretion. Conjunctival chemosis is defined as an edema of the sclera and occurs in 25%–90% of the cases, accompanying conjunctival injection in patients with Dural CCFs (33, 46, 52). Chemosis may occur before proptosis and is invariably limited to the inferior palpebral conjunctiva.

### **Retinal hemorrhage**

In severe cases of venous dilation and elevated intravenous pressure, optic disc swelling and retinal hemorrhages, caused by venous stasis and impaired retinal blood flow, with secondary ischemia or hypoxia, can occur. These intraretinal hemorrhages can be both, flame-shaped (located in the nerve fiber layer) and punctuate (located in the outer retinal layers) and can be associated with central retinal vein occlusion (59).

Dehydration of the cornea, usually painful, caused by lagophthalmos, is the main cause for corneal irritations in patients with DCCFs. Progressive damages of the corneal epithelium can be initially painless but may lead to local infections, corneal ablations and ulcerations.

## **Orbital bruit**

The bruit over the temporal bone or the orbit can be subjective and/or objective (auscultatory) and is usually synchronous with the heartbeat. It may appear as a buzzing, swishing or roaring and represents one classical symptom of high-flow CCFs. It is found in only 25% of patients with DCCFs, mainly in cases with posterior drainage (60). The bruit develops due to arteriovenous turbulences within the CS, which may reach the inner ear organ via sound transmission through the skull.

## **Secondary glaucoma and visual loss**

After manifestation of orbital symptoms, the chronic elevated intraorbital venous pressure may lead in 20% of patients with DCCF to a blockage of Schlemm's canal. This may subsequently lead to gonioscopically detectable elevated intraocular pressure (secondary glaucoma), retinal ischemia and usually reversible loss of visual acuity. Many authors postulate that the elevated episcleral venous pressure is responsible for secondary glaucoma, rarely for papilla edema, retinal ablations, central vein thrombosis and hemorrhagic retinopathy (2%)(45,61,62). In 11% of the patients bilateral symptoms are found. In general, the clinical severity of visual loss correlates more with the venous drainage pattern than with the volume and flow velocity of AV shunt. Meyers et al. (16) found diminished visual acuity is found in up to 31%. In patients with rapid visual loss, neuropathy of the optic nerve and occlusion of the superior ophthalmic vein are found, respectively, while distal stenosis merely contributes to clinical improvement (60).

## **Other neurological symptoms**

Although cortical or leptomeningeal drainage can be found in 31% of the patients, intracerebral hemorrhage seem to occur less frequently than in DAVF, in only 1.5% (16). In general, central nervous system symptoms or dysfunction are less frequent and have been observed in larger patient groups in only 7/85 cases with vertigo (5%), intracerebral hemorrhage (2%) and cortical venous infarct (1%) (10).

Cerebral or cerebellar symptoms can be caused by a venous outflow restriction or venous hypertension. They are rare events and occur less frequently than in DAVFs

and are only observed, if a cortical venous drainage is present. Iwasaki et al. have recently reported a DCCF complicated by pontine venous congestion(63). The authors observed isolated sixth nerve palsy in a 71-year-old woman that was caused by brain stem edema due to an AV shunt with exclusive venous posterior drainage into SPS, cerebellar cortical veins and inferior vermian vein. Only a few more of such cases have been reported so far (49,64), showing that in fact a “cortical” venous drainage is not always the cause, but often a leptomeningeal retrograde venous drainage of the AV shunt instead (65).

Suh et al. divided 58 patients into four main symptom pattern (2)

- Orbital pattern (chemosis, exophthalmos, pain, eyelid swelling): 53%
- Cavernous pattern (ptosis, diplopia, anisocoria, ophthalmoplegia): 71%
- Ocular pattern (decreased vision, IOC > 20 mm Hg ocular pain, glaucoma, retinal hemorrhage): 64%
- Cerebral pattern (seizures, hemorrhage): 5%

The following table shows the incidence of frequent signs and symptoms in DCCFs in recent series (Meyers et al. (16); Stiebel-Kalish et al. (10); de Keizer (66); Theaudin et al. (21))

**Table 1**

Clinical Features	Meyers et al. (2002) % of 135	Stiebel-Kalish et al. (2002) % of 85	De Keizer (2003) % of 68	Kim (2006) % of 65	Theaudin et al (2006) % of 27
Conjunctival injection	93	76		-	41
Chemosis	87	21		32	37
Proptosis	82	76		21	37
Diplopia	68	-		34	45
Bruit	49	28		-	-
Decreased visual acuity	31	-		13	30
Headache/retroorbital pain	34	-		34	-
Retinal hemorrhage	-	18	-	-	-
Raised IOP	34	-	-	72	-

## **RADIOLOGICAL DAIGNOSIS OF DURAL CCF**

When a dural CCF is suspected, computed tomographic (CT) scanning, CT angiography, magnetic resonance (MR) imaging, MR angiography, orbital ultrasonography, transorbital and transcranial color Doppler imaging, or a combination of these tests may be of benefit in confirming the diagnosis. CT and MR imaging often are used in the initial work-up of a possible CCF.

### **CT and MRI**

CT findings in CCFs include proptosis, enlargement of the extraocular muscles, enlargement and tortuosity of the superior ophthalmic vein, and enlargement of the ipsilateral cavernous sinus.

MR imaging findings in CCFs are similar to those seen on CT with the addition of orbital edema and abnormal flow voids in the affected cavernous sinus. In the setting of a high-flow fistula and retrograde cortical venous reflux, MR or CT studies may reveal dilatation of leptomeningeal and cortical veins(67). In patients who have cerebral venous congestion and elevated intracranial pressures, cerebral edema and/or hemorrhage may be encountered.

### **Doppler Sonography**

Doppler sonography has shown value for diagnosis and follow-up in patients with CCF (68-71). It allows for the assessment of direction and velocity of blood flow, as well as the differentiation of a typical venous flow pattern from an arterialized vein with characteristic biphasic flow (70).

### **DSA**

The gold standard diagnostic test, however, remains a catheter angiogram.

Selective angiography of both internal and external carotid arteries on both sides should always be performed including superselective angiograms of IMA and APA. Picard et al. (1987) emphasized the need for an exhaustive angiographic work-up for visualization of all potential feeders: the cavernous branches of ILT and MHT, the jugular, hypoglossal and carotid rami arising from the APA, IMA branches (cavernous branch of MMA and AMA as well as artery of the foramen rotundum) and recurrent deep ophthalmic artery. A dural arteriovenous fistula of the CS can be uni-

or bilateral and often involves multiple feeders of both ICA and ECA territories, also in cases where the shunt is unilateral (72). Precise analysis of the venous anatomy is crucial for successful transvenous embolization.

The arteriogram should provide the following angiomorphological information:

- Localization of the fistula site
- Differentiation between indirect and direct fistula
- Demonstration of the entire arterial supply, in particular in cases with DCCF with visualization of so-called “dangerous anastomoses”
- Visualization of the CS and its tributaries with possible stenosis, thrombosis, ectasias, draining veins such as IPS, SOV, ICS etc.
- Identification of fresh thrombus in the CS or IPS
- Identification of risk factors such as CS varices, intercavernous pseudoaneurysms or cortical drainage
- Identification of trauma signs such as dissection or transection
- Identification of diseased, stenosed or tortuous supraaortic arteries
- Preexisting disease such as FMD or Ehlers-Danlos syndrome
- Visualization of collateral circulation with adjacent territories such as carotid bifurcation

The aim of angiography in patients with Dural CCF is to determine the exact location of the arteriovenous communication, and to decide which compartment of the CS is involved: anterior or posterior (or both), left or right. Dural arteries can cross the midline to supply an AV shunt at the contralateral CS, occasionally even when there is no significant ipsilateral supply.

Lasjaunias et al. have recommended performing selective catheterizations using even a microcatheter if necessary, to visualize the smallest pedicles and to identify the so-called “dangerous anastomoses”

(73, 74). Being aware of their existence is indispensable for avoiding ischemic complications during transarterial embolizations in ECA branches

## **TREATMENT MODALITIES**

- **Endovascular treatment**
- **Alternative treatment modalities**

## **ENDOVASCULAR TREATMENT**

The goal of treatment in this condition is to interrupt the fistulous communications and decrease the pressure in the cavernous sinus. This goal can be accomplished by occluding the arterial branches supplying the fistula (transarterial embolization) or, more commonly, by occluding the cavernous sinus that harbors the fistulous communications (transvenous embolization).

Transarterial embolization of dural CCFs generally is cumbersome because of the small size, tortuous anatomy, and multiplicity of arterial feeders. The transvenous approach often is simpler and carries a high rate of success. The venous approach may fail in a small percentage of patients, however; in these cases, transarterial embolization still can be a viable alternative.

### **Transarterial methods**

Transarterial techniques involve distal catheterization of the small meningeal branches supplying the fistula. Ideally, superselective microcatheter placement is performed with microcatheter tip as close as possible to the point of fistulous communication. Once a satisfactory microcatheter position is achieved, particulate agents or liquid embolic agents (NBCA/Onyx) are injected under fluoroscopic control with the goal of occluding the fistulous connections and penetrating the cavernous sinus. Although coils and particulate agents have been used, these agents used alone cannot cause permanent occlusion of the fistula. In patients with a fistula fed only by meningeal branches of the external carotid artery, the embolization material is introduced via a microcatheter placed in the external carotid artery and passed into the specific branch or branches that feed the fistula. In this setting, successful closure of the fistula is almost always possible, resulting in rapid resolution of all symptoms and signs. When the fistula is fed by meningeal branches from both the external and internal carotid arteries, only the branches from the external carotid artery are usually

embolized in the hope that the flow to the fistula will be sufficiently decreased to result in its subsequent closure. The internal carotid artery is usually not embolized in this setting unless the interventionalist can successfully catheterize the meningohypophyseal trunk or other meningeal feeders from the artery. In case of dural CCF involving the anterior meningeal branch of the middle meningeal artery it should be spared while using liquid embolic agent due to anastomoses between it and the ophthalmic artery to prevent filling of liquid embolic agent. The internal carotid artery can be protected by placing a balloon at the cavernous carotid artery. Temporary dilation of balloon can also be adopted in the pedicle of the middle meningeal artery so that Onyx reflux can be prevented and Onyx can be pushed into some small feeding vessels. But this may cause transient V and VI cranial nerve deficit after the procedure.

### **Transvenous methods**

Techniques of Transvenous Catheterization-There are a number of venous pathways connected to the cavernous sinus.

### **Approaches**

#### **Inferior petrosal sinus**

Transvenous access to the cavernous sinus can be achieved via the inferior petrosal sinus in the majority of cases (75). The inferior petrosal sinus route is the easiest, shortest, and safest approach to the cavernous sinus(76,77). Anatomically, its course is relatively straight, with a horizontal and vertical segment forming a wide angle that does not cause luminal narrowing or catheter kinking with friction that hinders advancements of coils. Another advantage is the mechanical “stability” of the sinus. Its course along the petro-occipital suture (petroclival fissure), covered by the dura mater provides stability resembling a “pipe-like” structure. This stability is particularly important when a larger amount of coils need to be pushed through tortuous anatomy (e.g. like the SOV), eventually causing friction within the microcatheter, which may easily become a procedure-limiting factor. The IPS allows for advancing a guidewire or a microcatheter even when its lumen is narrowed, irregular or completely occluded due to thrombosis.

The topographical anatomy of the IPS opening into the jugular vein allows a relatively stable positioning of a large guide catheter within the vein, or sometimes even into the IPS itself. This makes navigating a microcatheter into the ipsilateral or contralateral CS considerably easier compared to the retrograde navigation through the SOV, where positioning of a guiding catheter is less stable.

If the IPS is not identifiable on either side, even after repeated phlebograms, careful image analysis is crucial in order to identify the small “notch” or a stump that is often the only residual filling of a thrombosed sinus. This should include diagnostic arterial injections, particularly in cases in which there is no posterior drainage, or the IPS is thrombosed. Braided, reinforced microcatheters advanced over a 0.012”, 0.014” or 0.016” guidewire, usually allow easier navigation through the IPS and the CS. Reinforced micro-catheters are more stable throughout the procedure, an important factor in achieving a dense coil packing in the fistulous compartment. In cases where the IPS cannot be identified with certainty, the residual “notch”, if visible, should be catheterized very gently with a small, hydrophilic guidewire. The choice of microguidewires and microcatheters depends, to a large degree, on personal preferences, as well as on the type and level of acquired manual skills. Benndorf et al. have described “loop technique” for advancing a catheter through an irregular, stenosed, or even occluded sinus (77). The guidewire is advanced not with a straight end, but instead with a loop that is formed within the narrowed lumen by bending the tip. Such a distal shape can be navigated much more easily than one would expect as it adapts better to an irregular lumen than a straight guidewire. It gets less entangled in small septi or stenotic segments of the trabeculated cavernous sinus structure.

In order to avoid unintentional catheterization of bridging or pontine veins and to prevent perforation with subsequent intracranial hemorrhage, the simultaneous use of bi-plane “blank road mapping” and fluoroscopy of the native background are helpful.

When the posterior CS is approached through the IPS, it is usually possible to navigate the microcatheter into the anterior compartment and its connection with the SOV without difficulties. It is advisable to bring the microcatheter as close as possible to the SOV, preferably in its first segment. Coil deployment should begin within the

SOV approximately 2–3 mm before its entry into the CS. This initial coil positioning is crucial to avoid increased drainage of the AV shunting into the SOV and ophthalmological deterioration. On the other hand, coils deployed in the anterior SOV (2<sup>nd</sup> or 3<sup>rd</sup> segment) may cause thrombosis and occlusion of the entire vein with blockage of the central retinal venous drainage leading to serious clinical consequences. To achieve dense packing from the beginning, it is wise to start with a coil size that can be easily stabilized in an enlarged SOV. Three-dimensional or complex coils may also be used to create a basket that, similar to aneurysms, is subsequently filled with smaller and softer coils. The more stable this initial packing within the proximal SOV or at the CS-SOV junction is, the better it may be used as a scaffold for subsequent coils. It will allow a dense packing of the CS, without the risk of dislodging coils into the draining vein. Commonly, the coil placement is performed from anterior to posterior to keep the access through the IPS free until the end of the procedure. This is a useful strategy because there is always the chance that coils get inadvertently placed early during the procedure in the posterior CS, or even in the IPS. If this happens, before the actual AV shunt is occluded, complete packing of anteriorly located fistulas may become difficult, or the entire procedure may be jeopardized. Losing access to the CS or to a fistulous compartment in cases of an incomplete occlusion creates the risk of rerouting the drainage towards cortical veins, causing cortical venous hypertension and possibly hemorrhages.

In cases with drainage towards the contralateral side of the fistula, the microcatheter should always be advanced there first. Such a “cross-over” approach via the intercavernous sinus allows blocking the drainage before withdrawing the catheter and packing coils at the fistula’s side.

If cortical drainage is present, the most crucial part of the procedure is to block the connection between the CS and the cortical vein at the beginning of the occlusion, even before placing coils into the SOV. Only such blocking will prevent persistence, or even worsening by rerouting of the venous drainage that is associated with increased risk of intracranial hemorrhage. At the end of the procedure, occluding the IPS itself should be avoided as it may be needed as an access route in a subsequent

session. Secondly, an occlusion of the IPS may, if it serves as drainage for the CS, compromise the normal cerebral circulation of the posterior fossa.

The angiographic endpoint of the endovascular procedure is either subtotal or complete occlusion of the fistula. If there is a minimal residual AV shunt visible, the procedure can usually be stopped, because the postoperative normalization of the ACT will further promote ongoing thrombosis within the CS. Ongoing thrombosis in the CS may be impeded by the heparinization of the patient during the procedure. Subtotal occlusion is often sufficient and will lead to complete occlusion within a few days or even 24 h when the coagulation system is normalized.

The IPS approach was initially described as an access route to the CS for treatment of direct CCF (78). After its introduction as a venous route for embolization of indirect (dural) CCFs by Halbach et al (75) in 1989, the IPS has been increasingly used by numerous other groups (16,21,76,79, 80,81, 82).

Halbach et al. in 1988 described first the successful catheterization of an angiographically occluded, but eventually passable IPS in two patients (83).

Further, Yamashita et al. were able to successfully catheterize the sinus in two cases, despite it being “angiographically occult” (76). Quinones et al. in 1997 reported on a 30% success rate for IPS catheterization, including angiographically occluded sinuses(84).

According to Shiu et al. (1968), four main variants of the IPS/IJV junction exist and may play a role in the overall success rate of retrograde IPS catheterizations. Because of its complex architecture and the lack of connection to the IJV, the IPS could not be catheterized in 31% of the cases.

### **Superior ophthalmic vein approach**

When the ipsi- or contralateral IPS approach clearly fails, a transfemoral SOV approach may be performed during the same or a subsequent session. This is done in the following way: A 4-F guiding catheter (0.038” inner lumen) is introduced into the facial vein and navigated as distally as possible. The closer the tip of this catheter is placed to the angular vein, the easier the catheterization of the SOV will be with a microcatheter. A too proximally placed guide often requires more microcatheter

manipulations in the facial and angular veins. These veins tend to become more mobile within the subcutaneous soft tissue, making advancing the microcatheter through a tortuous SOV difficult.

The transfemoral approach through the facial vein or the SOV was introduced by Komiyama et al. (86) in 1990 and has been used by various groups with different success rates since then(21,79,). It may be an effective alternative route and is indicated when the SOV is significantly enlarged due to anterior drainage, and the IPS cannot be passed. This approach is usually feasible when the SOV is dilated enough and allows even cross-over and packing of the contralateral CS. It may be technically challenging though, if the SOV is tortuous, stenosed or thrombosed.

Two aspects seem important. First, the SOV approach is in general less feasible than the IPS approach, due to the less favorable anatomy. There is a longer distance to be catheterized combined with tortuosities of the angular and superior ophthalmic veins. Furthermore, the guiding catheter usually has a mechanically less stable position within the IJV or the facial vein than for the IPS approach.

### **Approaches via the Middle Temporal Vein or the Frontal Vein**

Catheterization of an enlarged draining middle temporal vein, that communicates with the angular vein for example, is not difficult (86, 87, 88). Kazekawa et al. (2003) recently reported on two cases successfully embolized with such an approach after direct puncture with an 18-G needle (87).

### **Transfemoral Superior Petrosal Sinus Approach**

As described first by Mounayer et al. (2002), transfemoral approach of the SPS may be successfully used for catheter navigation into the CS and is an alternative to IPS or SOV approach(89). Theaudin et al. reported the use of the SPS in two patients of which one procedure was successful(21). The authors emphasize the fact that the SPS should be patent for catheterization because recanalization may become hazardous due to anatomic proximity to the vein of Labbé.

### **Transfemoral Pterygoid Plexus Approach**

This approach has been described by Debrun for management of a direct CCF(90), and by Jahan et al. for treatment of a Dural CCF (91). Catheterization of the

pterygoid plexus may technically be more difficult than of the IPS, but when successful, it may provide a stable positioning for microcatheters, even when a contralateral approach is necessary.

### **Transcutaneous SOV Approach(SOV cannulation)**

In 1969, Peterson et al. reported the direct cannulation of the SOV after upper eyelid or sub-brow cut reported for treatment of a traumatic CCF (92). This was one of the first venous approaches to the CS ever described. Tresset al. (93) published most likely the first DCCF treated using this approach, which was the repeated by Uflacker et al. (94) and Labbé et al. (95). Miller reported on two CCFs and 10 DCCFs, who were successfully treated via SOV approach (96).

Other approaches which have been described in literature are transorbital SOV Approach (Direct puncture) & direct puncture of the sylvian vein.

### **Embolic Agents**

#### **Polyvinyl Alcohol (PVA) and Embospheres**

PVA particles (Contour PVA, Boston Scientific, Fremont; Tru Fill TM PVA, Cordis Endovascular, Miami Lakes, FL)

PVA particles are manufactured by different vendors in a size between 45–150  $\mu\text{m}$  and up to 700–2000  $\mu\text{m}$ , and are selected based on the caliber of the vessel in the targeted territory. One long-standing disadvantage of PVA has been the fact that these particles not only varied in size (ranges), but also had an irregular surface causing aggregation, clumping and occlusion of catheters and proximal vessel segments. In addition, the particles showed a tendency to swell after being in a contrast suspension for some time and usually had to be replaced by a new mixture several times throughout the treatment session. Newer PVA particle types come as hydrophilic microspheres in a calibrated size (Contour-SE, Boston Scientific). They are naturally opaque with a more uniform size distribution, a wider range of sizes and come pre-hydrated in saline in a prefilled syringe. Alternatively, Trisacryl gelatin microspheres (Embospheres, Guerbet Biomedical, Louvres, France) can be used and may offer some advantages because they are precisely calibrated at 100–300  $\mu\text{m}$  and have fewer tendencies to aggregate. The concentration of PVA in the contrast suspension is

chosen depending on the size of the vessels supplying the Dural CCF, but should be very dilute at first to avoid obstruction of the microcatheter. According to changes in the local hemodynamics that progressively occur during injection due to increasing blockage of the vascular bed, the concentration of particles and their size may be adjusted throughout the procedure. To start with smaller and continue with gradually increased particle sizes is usually most effective. For optimal visual monitoring of the embolic flux and early detection of reflux a magnified “blank road mapping” is strongly recommended. The more distal a catheter is placed, the smaller the particles that should be chosen. On the other hand, the injection of particles smaller than 150–300  $\mu\text{m}$  into the MMA, IMA or AMA may lead to cranial nerve palsy. A more global injection into the IMA using larger particles (possible through a 4-F diagnostic catheter) may show an immediate angiographic change, but is usually ineffective to achieve a long-term occlusion of an AV shunt. As an adjunct for transvenous coil occlusion, or when the aim is mainly to induce flow reduction and to promote thrombosis, such a strategy may be appropriate. The angiographic endpoint should be slow antegrade or stagnant flow.

### **Platinum (Non-detachable) Pushable Microcoils**

These coils have also been called “free” or pushable coils and are available in different lengths, diameters and shapes (straight, helical, flower or spiral). Some manufacturers have added Dacron fibers to increase the thrombogenicity while friction is minimized for the use in small microcatheters. Constant flushing of the microcatheter with heparinized saline is required to avoid friction within the microcatheter that may cause blockage and damage of the catheter lumen, necessitating exchange. Pushable fibered coils have been used in addition to detachable coils, to increase the thrombogenicity of the bare platinum coil mesh. They are also available in complex configurations, such as VortX-coils (Boston Scientific). VortX-coils and other fibered coil configurations have become available with a detachment system as GDCs. Other manufacturers have developed similar devices such as the nylon fibered coils (NXT, EV3), in sizes ranging from 2×20 mm to 3×100 mm, combining the advantages of being highly thrombogenic and controllable by a similar detachment system.

Some operators have used this type of coil for transarterial embolization of ECA feeders (97). However, while their placement leads to a proximal occlusion, resulting in a shunt reduction, it seldom produces a permanent obliteration. The major downside of transarterial coil embolization in AV shunting lesions is that future arterial approaches through the same pedicle are compromised and new treatment sessions may become jeopardized, unless TVO is used. Thus, proximal arterial occlusion with coils should be avoided whenever possible. It has never been considered useful, except when targeting a selective vessel blockage to avoid untoward migration of embolic agents via ECA-ICA anastomoses (e.g. MMA-OA anastomoses in transarterial embolization)

### **Detachable Platinum coils**

The first detachable coil, the GDC system (Guglielmi detachable coil system), is a non-fibered, soft bare platinum coil mounted on a stainless steel wire that can be detached by electrolysis after placement in the desired location. One of the first successful treatments using GDCs was a direct CCF, occluded by F. Vinuela in 1990 using only two coils (98). GDC Soft coils are made of a thinner wire than standard coils and thus more pliable (e.g. GDC-10 Soft coils are 38% softer than GDC-10 standard coils). In order to minimize the mechanical stress applied to cranial nerves coursing through the CS, the use of softer coils for the CS packing is beneficial. At the same time a denser coil mesh can be achieved that is similar to aneurysm treatments – a major factor for achieving complete occlusion of the fistula. The use of coils with a complex or spherical configuration at the beginning of the coil packing, when a basket in a certain compartment of the CS needs to be accurately built, is also advantageous. This is the case at the connection between the CS and the SOV or a cortical vein.

Beside electrolysis, other detachment techniques have been developed using a mechanical mechanism such MDS (Balt Extrusion) or DCS (William Cook Europe), heat (Micrus Endovascular Corporation, San Jose, CA) or hydraulic pressure (MicroVention, Cordis Neurovascular) that allow deployment of a platinum coil in a quick, safe and reliable manner.

## **Liquid embolic agents**

### **Cyanoacrylates**

In the 1980s, NBCA (N-butyl-2-cyanoacrylate), initially known as Avacryl in the US and as Histoacryl in Europe (Braun-Melsungen, Germany) replaced IBCA (Isobutyl-2-cyanoacrylate), being the first acrylic glue for medical application (99). Histoacryl is a tissue adhesive that polymerizes when in contact with ion solutions such as contrast, saline or blood. The polymerization of NBCA starts after a few seconds of contact with blood and can be controlled only to some degree, depending on various factors such as blood flow velocity, speed of the injection, pH of the blood and temperature of the glue. In order to increase radiopacity for fluoroscopic control, Histoacryl is mixed by most investigators with Lipiodol (ethiodized oil), a cottonseed-oil-based contrast agent made by Laboratoire Guerbet (France). In addition, this mixture decelerates the polymerization depending on the concentration of lipiodol from 1–30 s. Prior to the embolization with glue, the microcatheter must be flushed with 5% glucose (dextrose in water) to avoid premature polymerization within the catheter. Each injection of glue must be carefully observed under fluoroscopic control in order to recognize early even the smallest reflux into the proximal feeder and to avoid gluing the catheter to the vessel wall. Additional use of tantalum powder in the mixture further enhances radiographic visibility. A concentration as low as 10%–15% has been used by adding tungsten and lipiodol for TAE of five complex Dural CCFs by Liu et al. in 2000(100). Gounis et al. in 2002 showed that predictability of the embolization process with NBCA can be improved by adding glacial acetic acid to the embolic mixture (101). The intravenous injection of glue for occlusion of Dural CCFs has been reported only to a limited degree by Wakhloo et al (102) and was used in a few cases by Benndorf et al(103).

### **Onyx (Ethylene-Vinyl Alcohol Copolymer)**

Ethylene-vinyl alcohol copolymer (EVOH, Onyx, EV3, Irvine CA) is a newer liquid embolic agent whose main characteristic is its non-adhesiveness. In contrast to adhesive acrylates, Onyx™ is a precipitating embolic agent that mainly causes a mechanical vessel occlusion. It prevents microcatheters from gluing to the vessel wall, and thus allows significantly prolonged injection times (up to 40 min or more).

It is mixed with a solvent, dimethyl sulphoxide (DMSO) and tantalum powder and comes in ready-to-use vials for AVMs and AVFs in three concentrations: 6% (Onyx 18), 6.5% (Onyx 20) and 8% (Onyx 34), dissolved in DMSO. Onyx is a pre-mixed, radiopaque, injectable embolic fluid that solidifies upon contact with aqueous solutions or physiologic fluids. In contrast to NBCA, this property allows for temporarily pausing the injection to prevent untoward leakage into a non-targeted territory (104). It forms a spongy polymeric cast and a “skin” – solidifying from the outside while continuing to flow, “much like lava”, in the liquid center. Thus, Onyx can be delivered in a relatively cohesive manner.

Onyx-induced cavernous sinus obliteration has several distinct advantages compared with coils or NBCA. Unlike coils, which can compartmentalize the cavernous sinus and result in incomplete obliteration, Onyx gradually permeates the sinus interstices. In contrast to NBCA, which rapidly polymerizes, Onyx gradually precipitates in a radial fashion from outside inwards, forming a cast, and therefore can be injected more slowly and accurately while minimizing the need for repeated catheterizations. These inherent properties allow deep penetration within the vasculature and are particularly useful in filling the fine fistulous communications commonly seen in indirect CCFs. Furthermore, the injection may be interrupted several times during the procedure to allow assessment of the embolization pattern and early recognition of dangerous anastomoses. In contrast to NBCA, Onyx does not adhere to vessel walls, and thus a greater degree of reflux can be tolerated during embolization without fear of catheter retention.

Despite the efficacy and inherent advantages of Onyx in the treatment of CCFs, it is not without hazards. Cranial neuropathies following embolization may occur as a result of several mechanisms. Nerve ischemia/infarct from inadvertent embolization of CN nutrient arteries, CN compression secondary to postembolization cavernous sinus thrombosis and swelling, or possibly angiotoxic effects of DMSO may explain such events. The slow injection of DMSO to disperse in the bloodstream is a safe method to avoid possible angiotoxicity, and the slow injection of the Onyx is the main success factor to obtain further penetration without reflux.

## **Results of transarterial embolization**

One of the first series was reported by Vinuela et al. (46), describing 10 patients with Type D fistulas, of which seven were occluded by embolizing the ECA feeders using PVA particles or IBCA. Cure was documented in five patients (50%) after 5 months. One patient developed hemiplegia and aphasia due to reflux of IBCA through the FRA into the ICA and MCA. Another experienced acute deterioration of his vision. Barrow et al. (1) achieved good results in three of five patients (60%) treated by embolizing the ECA supply. Grossman et al. reported complete resolution of symptoms after particulate embolization in five of seven patients (105).

Picard et al. communicated results in a group of 32 patients; 25 (78%) underwent superselective embolization, achieving complete clinical and anatomical cure in 18 (72%) and demonstrated clinical cure without anatomic cure in six (24%). One patient (4%) suffered from stroke due to IBCA migration into the cerebral circulation and died after 3 months(106).

Halbach et al. achieved clinical cure in 77% and an improvement in 18% (n=22) patients treated between 1978 and 1986 by TAE of ECA branches using IBCA(60). One permanent deficit (4.5%) was seen in a patient who developed a stroke due to clot formation in the guiding catheter, as well as transient deficits in three cases (13%). The group later reported a complete cure rate of 78%, improvement in 20%, and a complication rate of 4%(107).

In the earlier series published by Debrun et al, who treated 25 patients with either PVA or Histoacryl, complete occlusion was reported in 48%(32).In two cases (8%), enlargement of ECA feeders occurred; additional TVO had to be performed in two others (8%), and one patient (4%) required surgical exposure of the SOV. Sonier et al. in 1995 reported a 61% success rate by particulate embolization of IMA branches (108). In two cases (25%), TAE had to be repeated to achieve a complete occlusion, and transient facial edema was observed in another.

Kupersmith et al. reported the successful embolization of ECA branches in 88% of their patients using PVA and IBCA with two recanalizations (12%) and four complications (25%), including hemiparesis and hemianopia, permanent 12<sup>th</sup> nerve palsy and persisting visual field defect(33).

Vinuela et al. reiterated their experience based on 74 patients, reporting complete cure in 31% and positive clinical response in 85%. The morbidity was 3.2%; in two patients untoward glue migration into the intracranial circulation occurred, leading to hemiparesis and aphasia in one (109).

Liu et al. in 2001 communicated the only larger series (n=55) in patients who were followed prospectively (110). In a subgroup of 41 patients (75%), TAE was performed using PVA (150–250  $\mu\text{m}$ ) and Histoacryl, injected into the distal ECA branches. The involved ICA branches were not approached. A 70.9% complete clinical cure rate was achieved, with improved symptoms in 14.5%. The authors do not specify whether these results were any different in the embolized (41 patients), or the non-embolized group (14 patients). Further, in 24 of these patients (58.5%), a transient worsening was observed that might be considered a transient adverse event, or even a complication (16,103). In four patients (9.7%) there was no improvement, while four demonstrated aggravation of symptoms (9.7%). The authors suggest the use of TAE to convert Type D into Type B fistulas and to shorten the time to complete cure with “conservative management”.

Theaudin et al. recently reported a success rate of only 25% (1/5 patients) in patients undergoing TAE with 300–500  $\mu\text{m}$ , followed by 500–700  $\mu\text{m}$  until flow in the internal maxillary artery stopped(21). It is emphasized that none of these patients became completely asymptomatic, although clinical improvement was seen. A relatively large particle size (>300  $\mu\text{m}$ ) is recommended by some operators for ECA embolizations to avoid cranial nerve damage (107), while others suggest 100–300  $\mu\text{m}$  (109). Larger particles will more likely produce an occlusion proximal to the fistula site, triggering recruitment of collaterals and recanalization.

It must be borne in mind that the dural branches feeding a Dural CCF may also be involved in the normal blood supply of CN (4<sup>th</sup>, 5<sup>th</sup>, 6<sup>th</sup>, 7<sup>th</sup>, 9<sup>th</sup>, 10<sup>th</sup>, 11<sup>th</sup>, 12<sup>th</sup>) (107) Paradoxical worsening caused by SOV thrombosis may occur following TAE (111), and has been documented in a significant number (61.5 %) of patients by Liu et al (110).

Aside from passage of PVA particles or liquid embolic agents into the brain circulation via ECA-ICA anastomoses, a major risk associated with transarterial

injections of embolic agents is reflux of the embolic agent from cavernous ICA branches.

Although liquid adhesives have been largely replaced by PVA particles and microspheres for TAE, their use can be a feasible option under certain conditions. Liu et al. injected 10%–15% mixtures of NBCA with lipiodol and tungsten into ECA branches, achieving complete resolution of symptoms after 1 month without definite neurological complication(100). If anatomy of the arterial supply is favorable, complete cure may be achieved by injecting even small amounts via an enlarged ILT or MHT. This technique, however, is not without risk and requires an experienced operator.

In a study by Zhang et al, amongst 22 patients, 10 patients underwent transarterial embolization with onyx. Two patients were treated transvenously after transarterial embolization(112). All patients had complete anatomical and clinical cure. Complications related to transarterial embolization procedures (n=11) occurred in one patient (9%) with transient V cranial nerve damage.

### **Results of Transvenous Embolizations**

In 1989, the first relevant series reporting transfemoral venous approaches was published by Halbach et al. (n=13)(75). The authors primarily used a transfemoral approach through the IPS, the SOV and the basilar plexus, achieving 90% angiographic cure and 77% clinical cure. They observed two complications; one patient developed a stroke after placement of a balloon, while another suffered from transient vision loss.

Yamashita et al. achieved complete angiographic cure in 14 of 16 (88%) cases, interpreting the failure in two cases with coils that were not optimally placed within the CS(76). This may have been caused by intracavernous trabeculae, thrombosis or unfavorable anatomy of the SOV. There were 12% transient and 6% permanent deficits.

Goldberg et al. in 1996 presented the first larger series of SOV cannulation and achieved immediate improvement or clinical cure in 100% of their patients

(n=10)(113). They were unsuccessful in two additional patients and observed one case of severe intraorbital bleeding.

Quinones et al. achieved a 92% occlusion rate in 12 successfully catheterized patients and a clinical recovery in all but two patients (83%)(84). The authors observed two delayed complications. One patient developed a palpebral silk granuloma, another transient contralateral cavernous sinus syndrome (6<sup>th</sup> nerve palsy).

Roy and Raymond in 1997 reported on 24 patients with DAVFs; 12 with Dural CCFs(114). Nine were treated using transvenous occlusions only; 89% demonstrated complete anatomic occlusion. In this series, transient CN deficits occurred relatively frequently (50%), and in one patient permanent sixth nerve palsy was seen (8%). The authors explained these complications with local thrombosis inside the CS that led to CN irritations, and considered mechanical pressure less likely the reason, since the symptoms were irreversible.

Oishi et al. reported results and complications that may occur when employing different venous approaches(115). They achieved complete angiographic cure in 89% of cases with a relatively high complication rate of 32%. These complications consisted of transient 6<sup>th</sup> nerve palsies (n=3), dissections of the IPS (n=1), blepharoptosis (n=2), as well as permanent dysesthesia of the forehead (n=1) due to upper lid incision for the SOV approach.

Gobin et al. (2000) reported angiographic cure in 24 of 26 patients (92%) and a complete clinical cure in 25 patients (96%)(116). The authors observed two complications (7%): one transient 6<sup>th</sup> nerve palsy (4%) and one case of visual loss due to thrombosed central retinal vein (4%).

Cheng et al. (2003) treated 27 patients with TVO achieving complete angiographic obliteration in 89% (30% immediate) and clinical cure in 96% (79). Two patients presented with recurrent symptoms and underwent a second procedure. The authors observed transient 6<sup>th</sup> nerve palsy in three patients (11%), which occurred with delay in two, suggesting progressive thrombosis and inflammation inside the CS.

Meyers et al. (2002) in their series had 135 patients followed over a period of 15 years (16). The majority (76%) of patients undergoing EVT were treated by

transvenous approach, achieving angiographic and clinical cure rates of 90%. Eight patients (6%) experienced symptomatic complications, including infarction (n=1), visual deterioration (n=2), diabetes insipidus (n=1) and orbital ecchymosis (n=1). The overall procedure-related permanent morbidity was 2.3%; however, the authors do not specify whether it was related to TVO or TAE. Angiographic follow-up was obtained in 54%; one third of the patients required more than one intervention.

More recently, Theaudin et al. (2007) reported on 27 consecutive patients undergoing transvenous occlusions (n=16) or transarterial embolizations (n=4)(21). Complete occlusion was achieved in 14/16 patients (88%) with early-improved symptoms in 12 (75%). One patient (6%) developed a temporal lobe hemorrhage immediately following transvenous occlusion in a fistula with cortical venous drainage, possibly due to blockage of the fistula drainage by placement of the guiding catheter into the IPS. The patient fully recovered without permanent deficit when seen at 1 year follow-up.

Yu et al. (2007) reported a series of 61 patients undergoing 64 successful TVO procedures and achieved anatomical cure in 95%(82). In 38 patients the fistula was occluded immediately after the procedure, in 20 a mild residual fistula was documented and completely occluded in FU exams (3–16 months). Three patients showed persistent symptoms and underwent repeat TVO, while 16 patients showed cure within 2 weeks, 22 after 3 months. There were two patients with transient 6<sup>th</sup> nerve palsy. Using catheterization of the SOV either via the facial or the middle temporal veins the authors were successful in 11/11 cases. The authors' technical success rate increased from 71.6% to 86.5% after adapting transfacial SOV approach. In 7/8 patients with residual symptoms the IPS was used as initial route.

Kim et al. (2006) achieved an immediate 75% occlusion rate (complete or nearly complete) with cure or improvement of symptoms in 91% of patients(81). A total of 11 complications (20%) were observed, including six cranial nerve palsies, three venous perforations and two patients who developed brainstem congestion. Transient CN palsies seen in six cases (10.7%) were likely due to overpacking of the cavernous sinus or extensive thrombosis within the CS. Venous perforations during IPS catheterizations were seen in 5.4% (three cases), none of which resulted in

clinical sequelae due to immediate recognition and coil embolization. Rupture of an IPS may not cause serious clinical complications as most perforations occur when a catheter is advanced through a thrombosed or occluded IPS with minimal or no AV shunt flow. Venous congestion in association with Dural CCFs has been reported by a number of authors (117,118). Kim et al. (2006) demonstrated a rate in 3.6%, attributed to rerouted venous drainage after coil packing within the CS(81).

The results achieved by the various groups show anatomic cure rates ranging from 52%–100%, depending on whether or not immediate complete or near complete (subtotal) obliteration of the AV shunt is considered the end-point. Similarly, the rate of clinical cure ranges from 63%–100%, with the majority of groups achieving more than 80%–90% clinical cure.

## **ALTERNATIVE TREATMENT OPTIONS**

### **Spontaneous thrombosis**

Early reports on spontaneous occlusion of CCFs include the monographs from Sattler (1930), Dandy (1937) and Hamby (1966), which reported 5.6%–10% occlusion rates (12,119,120). Some data suggest that about 30% of the patients in all series show a spontaneous occlusion of the AV shunt, although the published material is quite heterogeneous (8).

Data on the “natural history” of DCCFs are in general incomplete, because some “spontaneous” occlusions occurred following cerebral angiography (5,43,44), manual compression therapy (Kai et al. 2007)(121), or in groups of patients undergoing transarterial embolizations (11,122).

“Spontaneous” occlusions of CCFs that follow intravascular contrast administrations have been described by several investigators (30, 43, 44, 123). Voigt et al. (1971) reported the spontaneous occlusion of a bilateral DCCF associated with cerebral angiography (44). The authors questioned the role of vasoconstrictor effect of the contrast medium triggering local thrombosis. They favored a theory of stasis following changes in pressure gradients during angiography. The role of general anesthesia was explained usually accompanied by a lowered systemic blood pressure.

The latter theory has been considered by others as well (30, 124) and reveals some evidence in fistula occlusions achieved by induced hypotension.

In 1980, Seeger et al presented six patients with spontaneous occlusions (43). They discussed the role of contrast medium that likely induces thrombosis by direct interaction with the endothelium that causes aggregation of platelets and white blood cells, accelerating the clumping of erythrocytes and thrombosis.

Spontaneous occlusions may be accompanied by exacerbation or regression of the symptoms, because a fresh thrombus in the CS may redirect AV shunting flow towards the SOV, increasing IOP. That is why a paradoxical increase of symptoms due to on-going thrombosis can be seen in some of the patients (43, 54,105).

### **Manual compression therapy**

The most widely used non-invasive conservative management of CSF patients is intermittent manual compression therapy (MCT). This involves a simple maneuver to reduce the arterial inflow and the venous outflow of CSF and was used by Scott in 1834 for diagnostic purposes.

Gioppi (106) suggested four different compression techniques: (1) from anterior to posterior between the two heads of the sternocleidomastoid muscles, (2) using the 2<sup>nd</sup>, 3<sup>rd</sup>, and 4<sup>th</sup> finger of the left hand along the lateral margin and the thumb along the medial margin of the sternocleidomastoid muscle, while the right hand pushes the head to the involved side, (3) using the second finger at the anterior margin of the sternocleidomastoid muscle posteriorly and slightly lateral, and (4) slight compression against the larynx or trachea.

Manual compression therapy as a treatment option for CCFs has been advocated by numerous investigators. Halbach et al. in 1987 asked patients with DCCFs to compress their carotid arteries and jugular veins with their opposite hands while sitting for 10 s several times per hour(60). When tolerated, the compression was increased 30 s over a total of 4–6 weeks. Patients with angiographic evidence of cortical venous drainage were excluded. The authors achieved complete cure in 7/23

(30%) patients undergoing carotid jugular compression. In 1992, complete cure was reported in 34% of 53 patients (108).

Kai et al. (2007) studied a group of 23 patients, achieving complete resolution of symptoms in eight cases (35%)(121). The authors identified lower ocular pressure, a shorter interval between symptom onset and compression treatment and venous drainage solely via the superior ophthalmic vein without involvement of the inferior petrosal sinus as factors that would favor a complete occlusion achievable by this technique.

### **Radiotherapy**

Radiotherapy of DCCF represents an effective alternative (up to 90%) (Barcia-Salorio et al. 2000) and should be considered a valuable complement of the therapeutic spectrum for DCCFs(9). In selected cases in which endovascular means remain unsuccessful, ineffective, contraindicated or considered too risky, irradiation using a linear accelerator or gamma knife should be considered. In elderly patients with comorbidities, lengthy endovascular procedures under general anesthesia could be avoided, especially when they have already failed in previous sessions. Radio-surgery should focus primarily on small, low-flow shunts and, if possible, be combined with transarterial embolization or manual compression.

### **Direct surgery**

Direct surgical treatment of CSFs was the primary treatment modality in the pre-endovascular era; however, it was associated with significant morbidity and mortality. Current endovascular tools and high-resolution imaging capabilities have minimized the need for direct surgery in the therapeutic management of DCCFs. It should be applied in selected cases only to facilitate endovascular approaches.

# *Materials and Methods*

This was a retrospective study carried out in the department of IS & IR, at SCTIMST, Trivandrum. For the purpose of this study, patients who had undergone endovascular management for Dural CCFs in the department of IS & IR, SCTIMST, dating from 1<sup>st</sup> Jan 2006 to 31<sup>th</sup> Dec 2012 were included. There were a total of 42 cases in the initial list.

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, imaging, angiographic characteristics, details of the embolization procedure, post procedural status and the follow up was collected.

All patients underwent a baseline ophthalmological evaluation prior to the procedure which included visual acuity, visual field charting, fundoscopy and IOP measurement. All the patients had a baseline imaging done – either a CT or MRI of the brain. Subsequently, the patients underwent a complete four vessel diagnostic cerebral angiogram. All the studies were performed on Advantx digital subtraction angiography unit (GE Milwaukee, USA) or Innova biplane flat panel digital subtraction angiography unit (GE Milwaukee, USA). A wide variety of catheters and embolic materials was used for the procedures as was best suited for the individual patient depending on the angioarchitecture.

Diagnostic angiographies were done under local anesthesia when they were performed separate from interventional procedure or performed as a check angiography for follow up. Transarterial embolizations were also done under local anaesthesia. The transvenous embolization procedure was done under general anesthesia. Premedication (Inj. Pethidine/ Tramadol 25-50mg IM & Inj. Phenergan 12.5- 25mg IM) was given before the local procedures. Post procedure these patients were managed in the wards when angiography/embolization was performed under local anesthesia. For procedures carried out under general anesthesia, the patient was monitored in the neurological/radiology intensive care unit.

After discharge, the patients were followed up with clinical evaluation for improvement in their symptoms. Follow up imaging or angiograms were evaluated when available.

Patients were advised carotid compression (to be performed with contralateral hand in sitting/lying position for 10-30s multiple times in a day) while definitive endovascular management was being planned and in cases of incomplete anatomic cure/unsuccessful endovascular management.

A complete evaluation of the patient including demographic profiles (age, sex), clinical presentation, imaging features, treatment method and outcome including complications and follow up was carried out as per the proforma attached.

#### **Technique of embolisation:**

All procedures were performed via the transfemoral route using the Seldinger technique. The guiding catheters used during the procedure were continuously flushed with heparinized saline (2000 units /L) with systemic heparinisation of 3000- 5000 IU as bolus and 1000 IU every hour monitoring the ACT.

The transarterial embolization procedures were performed under local anesthesia. A guiding catheter was placed in the main feeding arteries(ECA). The feeders were selectively cannulated with either a 4F Vertebral glide catheter or a microcatheter. They were then embolized using PVA particles with or without gelfoam. One patient underwent transarterial embolization using onyx. While performing non selective embolization of MMA, anterior branch was blocked with coils(fibre/liquid) to prevent reflux into MMA-OA collaterals.

For transvenous coiling under general anesthesia, after arterial and venous femoral punctures and placement of 6/7-F introducer sheaths, an intravenous bolus of 50-100 IU/kg of body weight heparin was administered. Monitoring was performed keep the activated clotting time (ACT) at a level of approximately 250–300 s or 2× above the normal level. A 5-F vertebral glide catheter was advanced into the ECA or ICA on the side of the dominant fistula supply to allow for road mapping and control injections throughout the procedure. A 6-F angled guiding catheter or 6 or 7F long

sheath was placed in the IJV on the side that showed dominant drainage, or that looked most promising on the diagnostic angiograms. The catheters were flushed with heparinized saline (2000 IU per liter). Arterial and venous femoral punctures were performed uni- or bilaterally.

In patients who underwent embolization using IPS approach navigation through the IPS was attempted initially with a 4F braided catheter over 0.035” guidewire. If that was unsuccessful then braided, reinforced microcatheters advanced over a 0.012”, 0.014” or 0.016” guidewire were used for navigation through the IPS and the CS. If the IPS was not identifiable on either side, even after repeated phlebograms, careful image analysis was done in order to identify small “notch” or a stump that was often the only residual filling of a thrombosed sinus. The residual “notch”, if visible, was catheterized very gently with a small, hydrophilic guidewire. After navigating through the IPS microcatheter was navigated to the anterior cavernous sinus and tip kept in SOV, preferably in its first segment. Coil deployment was commenced within the SOV approximately 2–3 mm before its entry into the CS. It continued from anterior to posterior till complete packing of the fistula was achieved. In case of any residual fistulous communication onyx (EVOH) was injected slowly under road map guidance.

For patients who underwent transvenous coiling using SOV approach 6F guidecatheter/long sheath was navigated into the ipsilateral IJV. Then with coaxial technique using 4F/5F catheter (vertebral glide/Multipurpose) and braided microcatheter/microwire combination facial vein/angular vein was cannulated. Microcatheter was taken into the SOV and cavernous sinus which was then packed with coils.

The catheters and wires used for the purpose are shown in table 2.

**Table 2 : MATERIAL USED FOR ANGIOGRAM AND EMBOLIZATION**

1.	Sheath – Radiofocus, Cordis	5, 6, 7 Fr
2.	Long sheath	6,7 Fr
3.	Diagnostic catheters – Vertebral glide (Terumo), Right coronary (Cordis), Mani cerebral (Cordis), Simmons (Cordis), Multipurpose (Cordis)	5, 4 Fr
4.	Guide wires – Terumo exchange length, Terumo standard,	150 cm – Standard 260 cms - Exchange
5.	Guiding catheters – Vistabrite, Launcher (Cordis), Neuron	6, 7 Fr
6.	Microcatheters – Echelon-10, Excelsior 1018,Rebar-18,	1.5 to 1.8 Fr
7.	Microguidewires– Transcend, Transcend EX	.010-.014”of varying length (190-205cm)
8.	Embolic materials –PVA, Gelfoam, GDC/Axium/Fibre coils, Onyx- 18	PVA-150-350,300-500,500-700 microns

Follow up data from all patients were obtained from hospital case record, during their OPD visit and also telephonic conversation with patients or their immediate relatives. All patients underwent ophthalmological evaluation on follow up visits. Follow up angiography was performed in case of worsening in the symptoms on clinical follow up.

## *Results*

### Demographic profile

There were a total of 43 cases initially. However, only patients with sufficient clinico radiologic data were included in the study. The final list thus included 42 patients. The age of the patients ranged between 14-77 years (mean 51.9 years) and male to female ratio was 14: 28. The distribution in various decades of life is as follows:-

**Table 3**

<b>Age group(yrs)</b>	<b>Number of patients</b>
0-9	Nil
10- 19	1
20- 29	4
30- 39	3
40-49	9
50-59	12
60-69	9
70-79	4

## Clinical Features

The clinical features are summarized in the following table

**Table 4**

<b>Clinical Features</b>	<b>Number of patients</b>
Conjunctival injection	32(76%)
Chemosis	20(47%)
Diplopia	21(50%)
Proptosis	25(59%)
Ophthalmoplegia	20(47%)
Dimunition of vision	13(30%)
Raised IOP	9(21%)
Headache	18(42%)

7 patients had tinnitus and 3 had bruit. 1 patient had hemorrhagic choroidal detachment and 1 had epistaxis.

## Symptom pattern

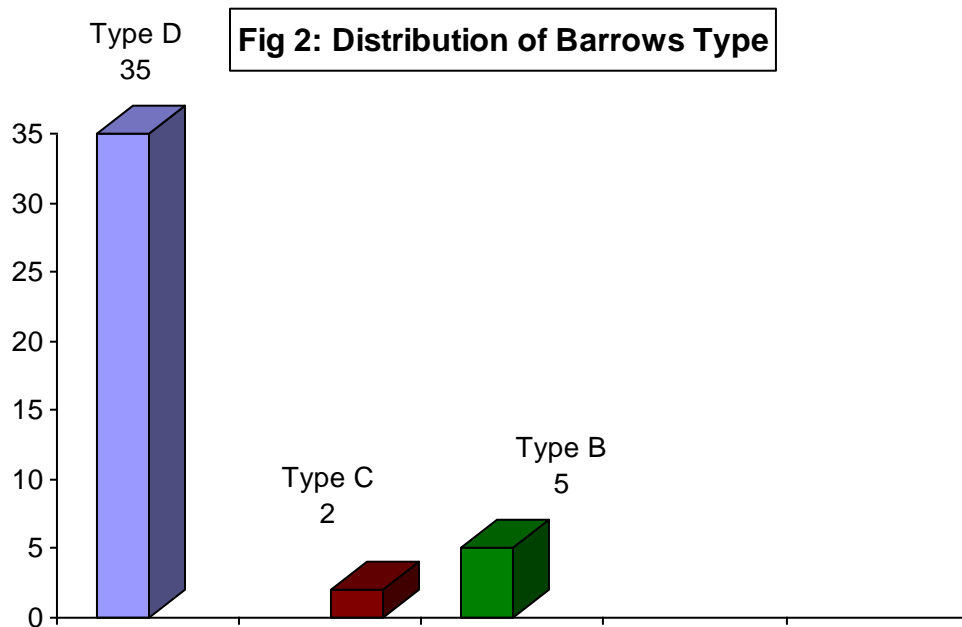
29 patients had a mixed symptom pattern. 10 patients had orbital pattern and 3 patients had cavernous pattern.

Out of the 29 patients with mixed symptom pattern 10 had orbital + ocular + cavernous pattern and 10 had orbital with cavernous pattern and 9 had orbital with ocular pattern. None of the patients had cerebral pattern.

### **Angioarchitecture**

#### **Barrows stage type**

35 of the 42 patients were of Barrows Type D, 5 were of Type B and 2 of Type C



Out of the 42 patients all except one had unilateral fistulas. 31 patients had bilateral supply and 11 patients had unilateral supply. 24 patients had filling of both anterior and posterior compartments of the cavernous sinus while 15 and 3 patients had filling of posterior and anterior compartments respectively.

#### **Proliferative/Restrictive/Late restrictive**

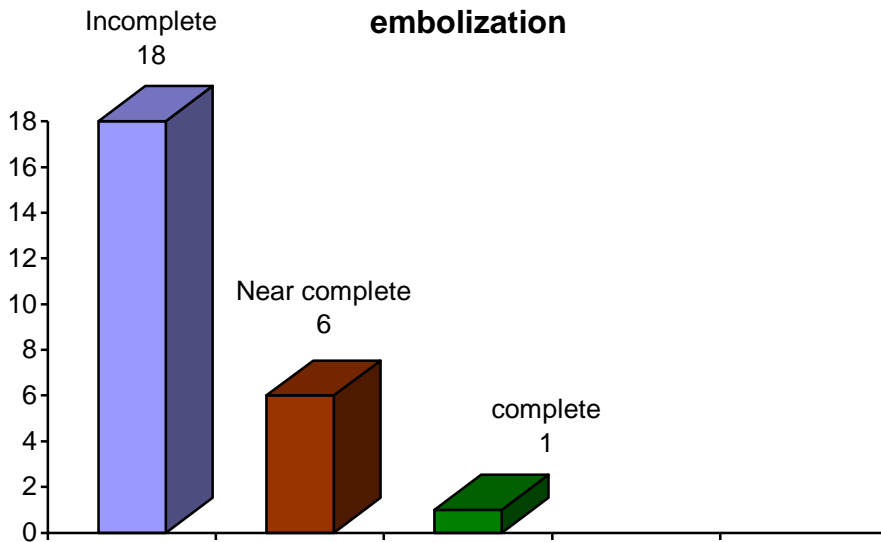
11 patients had proliferative pattern, 16 had restrictive pattern and 15 had late restrictive pattern. 1 patient who had a proliferative pattern on initial angiogram

showed restrictive pattern on follow up angiogram after transarterial embolization. Cortical/leptomeningeal venous reflux was seen in 7 patients. 1 of these patients had also reflux in the spinal perimedullary veins. 3 other patients revealed cortical reflux after follow up angiograms after transarterial embolization.

## **Management**

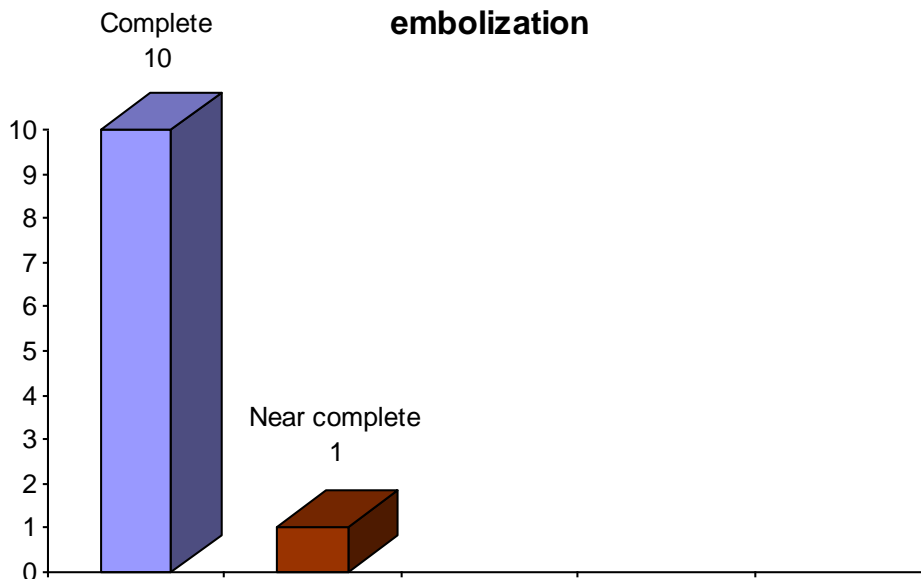
For the purpose of description, the extent of embolization has been divided into complete (no residual shunt), Near complete (minor residual shunt) and incomplete (significant residual shunt). Clinical symptoms after embolization have been divided into normalized, improved and unchanged/worsening. 2 patients had spontaneous obliteration of their CCF when follow up angiogram was done prior to embolization. Both these patients had complete resolution of clinical symptoms on follow up. 1 patient (Barrows Type B) had complete normalization of symptoms only with carotid compression. 25 patients underwent transarterial embolization with PVA particles/gelfoam (2 out of these underwent failed transvenous embolization earlier). Out of these 25 patients, 1 patient (Barrows Type C) had complete angiographic obliteration of the CCF and showed normalization of symptoms. Six patients had near complete obliteration of the fistula. Three of these patients showed normalization of symptoms on follow up; two showed improvement in symptoms and one was lost to follow up. 18 patients had incomplete angiographic obliteration following transarterial embolization. Out of these six patients had no change/worsening of the clinical symptoms on follow up. Three of the six patients underwent transvenous coiling (two through facial vein/angular vein/SOV approach) and one through IPS approach (onyx was used in addition to coils). 1 patient underwent 2<sup>nd</sup> sitting of transarterial embolization using onyx with incomplete obliteration of fistula. 2 patients were lost to follow up. 2 patients who had incomplete obliteration after transarterial embolization showed normalization of symptoms on follow up. 6 showed clinical improvement while rest were lost to follow up.

**Fig 3a: Extent of fistula obliteration after transarterial embolization**



8 patients underwent transvenous coiling in the first sitting itself. All of these underwent embolization through the IPS approach. Out of these, 4 patients had IPS which was not visualized in diagnostic angiograms. In 7 of the 8 patients complete angiographic obliteration was achieved while in one patient near complete obliteration was achieved. Onyx was used in addition to coils in 6 of these patients. All the patients had shown clinical improvement immediately after procedure and complete resolution of symptoms on follow up. 2 patients underwent radiotherapy after unsuccessful attempts at transvenous embolization.

**Fig 3b: Extent of fistula obliteration after transvenous embolization**



The following table shows the clinical outcome of endovascular interventions

**Table 5**

	Normalized	Improved	Unchanged/ worsened	Lost to Follow Up
Transvenous embolization(n=11)	11	-	-	-
Transarterial embolization(n=25)	6	8	6	5

### **Complications**

One patient had increase in proptosis, extraocular movement restriction and blurring of vision after transarterial embolization with PVA particles due to SOV

thrombosis. This was managed with IV heparin and IV methyl prednisolone followed by oral warfarin following which the patient showed improvement in symptoms. One patient had dissection of internal maxillary artery while cannulation during transarterial embolization. However the patient did not have any symptoms. One patient had dissection of vertebral artery while performing diagnostic angiogram but with no neurological sequelae. Two patients had trigemino-cardiac reflex with transient bradycardia while performing transvenous embolization with onyx which was managed with IV atropine.

## *Discussion*

The study was carried out with an aim to assess epidemiologic, clinical and management aspects in all the dural CCF cases who were treated at a tertiary care referral institute, with a focus to generate data in Indian context as most of the extensively published data has been from the western literature.

The study included 42 patients treated in this institute between Jan 2006 and Dec 2012. Patients showed spread out distribution among second to seventh decades with patient age range from 14-77yrs (Mean 51.9yrs) with preponderance in fifth to seventh decade. This is similar to one of the largest published retrospective evaluation of 135 consecutive patients by Meyers et al where patient age ranged from 18 to 87 years, with a mean age of  $60 \pm 1.6$  (mean  $\pm$  standard error) years.

The male to female ratio (14:28 – 1:2) in our study showed a female preponderance. This is in concordance with previously published literature which has shown female preponderance of dural CCF.

The most common clinical presentation was conjunctival injection followed by proptosis, chemosis and diplopia. This is similar to two large series by Meyers and Stiebel Kalish et al. The clinical presentation of dural CCF in various series is shown in Table below

**Table 6**

Clinical Features	Meyers et al. (2002) % of 135	Stiebel-Kalish et al. (2002) % of 85	Kim (2006) % of 65	Present series % of 42
Conjunctival injection	93	76	-	76
Chemosis	87	21	32	47
Proptosis	82	76	21	59
Diplopia	68	-	34	50
Decreased visual acuity	31	-	13	30
Headache/retroorbital pain	34	-	34	41
Retinal hemorrhage	-	18	-	-
Raised IOP	34		72	21

In our series there were 29 patients who had a mixed symptom pattern, 10 patients had orbital pattern and 3 patients had cavernous pattern. Out of the 29 patients with mixed symptom pattern 10 had orbital + ocular + cavernous pattern and 10 had orbital + cavernous pattern and 9 had orbital + ocular pattern. So there were 39 (92%) patients having orbital pattern, 23 (54%) with cavernous pattern, 19 (45%) with ocular pattern and no patient with cerebral pattern. In the series by Suh et al of 58 patients 53% the patients had Orbital pattern, 71% had Cavernous pattern, 64% had Ocular pattern and 5% had cerebral pattern.

As far as angioarchitecture features are concerned, most of the dural CCF in our series were of Barrows Type D (83%) followed by Type B (12%) and Type C(5%). In a study by Debrun et al out of 32 patients of dural CCF there were 28 patients (87%) with Type D and 4 patients(13%) with Type C while no patient had Type B fistula. In the series of Barrow et al there were six patients with Type D fistula, 5 had Type B and 2 Type C fistulas. Our series has shown that Type D fistula which has feeders from both dural branches of ICA and ECA are the most common type of dural CCF as shown in previous studies.

11(26%) patients had proliferative pattern, 16(38%) had restrictive pattern and 15(35%) had late restrictive pattern. In the series by Suh et al 40%, 40% and 20% patients were found to have proliferative, restrictive and late restrictive patterns respectively (2). 1 patient who had a proliferative pattern on initial angiogram showed restrictive pattern on follow up angiogram after transarterial particle embolization. Suh et al had also reported change of the type during follow-up in 7 patients. Changes from PT to RT were noted in 3 patients. One of these patients received no treatment, one received particle embolization, and one received coil embolization. Changes from PT into LRT were noted in another 3 patients, out of which one received transarterial particle embolization.

Cortical/leptomeningeal reflux was seen in 7 patients (16%). 1 of these patients had also reflux in the spinal perimedullary veins. Paraspinal vein drainage was seen in 4.5% of patients in the series by Stiebel Kalish et al(10). 3 other patients revealed cortical reflux after follow up angiograms after transarterial embolization. None of the patients with cortical/leptomeningeal reflux in our series developed intracranial hemorrhage. As per the literature also associated risk of intracranial hemorrhage (around 2%) is relatively low compared to patients with DAVFs in other locations, especially at the tentorial sinus or in the anterior cranial fossa. The following table shows the frequency of cortical/leptomeningeal venous drainage(C/LVD) and associated hemorrhage in the literature

**Table 7**

Authors	Dural CCF	C/LVD	Hemorrhage
Halbach et al	30	3(10%)	-
Awad et al	45	n/a	n/a
Cognard et al	33	4(12%)	0
Tomsick	50	8(16%)	0
Satomi et al	65	17(26.1%)	1(1.5%)
Theaudin et al	27	5(18.5%)	0
Stiebel Kalish et al	88	22(26%)	2(2.2%)
Meyers et al	135	41(31%)	2(1.5%)
Present series	42	7(16%)	0

2 patients (4.7%) in our series revealed spontaneous obliteration of the fistula on follow up angiogram prior to definite endovascular management. The number of reported “spontaneous” occlusions reported in the literature lie between 11% and 90% (33, 46) and is on average 35% according to Tomsick et al(8). Both of the patients had undergone diagnostic angiogram previously (3mths and 1 week before) and were taken up for definitive endovascular management under GA when repeat DSA revealed complete obliteration of the CCF.

This could be the result of vasoconstrictor effect of the contrast medium triggering local thrombosis and stasis following changes in pressure gradients during angiography. It may also be due to thrombosis induced by contrast medium by direct interaction with the endothelium that causes aggregation of platelets and white blood cells, accelerating the clumping of erythrocytes and thrombosis. The role of general anesthesia may also play a role because it is usually accompanied by a lowered systemic blood pressure leading to spontaneous thrombosis.

Out of 25 patients who underwent transarterial embolization only 1 patient (Barrows type C) had complete angiographic obliteration of fistula. This patient showed normalization of symptoms on clinical follow up. 6 patients had near complete obliteration out of which 3 had normalization of symptoms, 2 had clinical improvement while 1 was lost to follow up. 18 patients had incomplete obliteration of fistula with residual fistulous communication between ICA feeders/residual ECA feeders. Out of these six patients had no change/worsening of the clinical symptoms on follow up. Three of the six patients underwent transvenous coiling (two through facial vein/angular vein/SOV approach) and one through IPS approach (onyx was used in addition to coils). 1 patient underwent 2<sup>nd</sup> sitting of trans-arterial embolization using onyx with incomplete obliteration of fistula. 2 patients were lost to follow up. 2 patients who had incomplete obliteration after trans-arterial embolization showed normalization of symptoms on follow up. 6 showed clinical improvement while rest were lost to follow up.

In all anatomic cure was achieved in 1/25 patient, near total obliteration was observed in 6/25 patients. Clinical cure was achieved in 6/25 patients while 8/25 patients showed clinical improvement.

In the earlier series published by Debrun et al. (1988), who treated 25 patients with either PVA or Histoacryl, complete occlusion was reported in 48% (32). Sonier et al. (1995) reported a 61% success rate by particulate embolization of IMA branches (109). Theaudin et al. (2007) reported a success rate of only 25% in patients undergoing TAE with PVA particles (21). This is similar to our series where complete/near complete obliteration was seen in 28% of cases (7/25).

One patient with a Type B CCF on carotid compression therapy achieved normalization of symptoms on follow up. Manual compression may also have played a role in patients who underwent transarterial embolization and displayed normalization of symptoms/complete obliteration on follow up angiograms. In literature, complete obliteration has been reported after manual compression in 30-35% of patients. (60,108,122)

8 patients underwent transvenous embolization as 1<sup>st</sup> endovascular modality while 3 patients had transvenous embolization following transarterial embolization. Out of 11 patients 10 patients had complete obliteration of the fistula while one had near complete obliteration. All the patients had normalization of clinical symptoms on clinical follow up. All patients(100%) had complete/near complete obliteration of the fistula after transvenous embolizations. These results are similar to/better than the results obtained in most of the series so far which have shown anatomic cure rates ranging from 52%–100%, depending on whether or not immediate complete or near complete (subtotal) obliteration of the AV shunt is considered the endpoint. Similarly, the rate of clinical cure ranges from 63%–100%, with the majority of groups achieving more than 80%–90%.

4 patients underwent successful catheterization of unopacified IPS. The success rate of catheterization of IPS in cases with no visualization of IPS on angiogram is reportedly 30%–50% (41, 46, 75, 84).

Our better results may be explained because of improvement in the neurointerventional hardware/techniques over the years. In addition, use of onyx in addition to coils in 7 out of 11 patients who underwent transvenous coiling have also contributed in complete obliteration of the fistula. The results are similar to those by Li et al who had treated 6 patients with Onyx with or without coils using transvenous approach and had achieved 100% anatomic and clinical cure (126). This study shows that Onyx is suitable for transvenous casting of the cavernous sinus due to its nonadhesive and cohesive properties (127,128). The slow injection of the agent enabled gradual filling of the interstices of the cavernous sinus and blocking of minute fistulous communications. This enabled complete occlusion of the fistula in one procedure.

Two patients had transient bradycardia/hypotension during transvenous coiling with onyx which was managed with atropine. These findings are comparable with what have been reported by Li et al(126) and Lv et al(129).In both the studies two out of six patients treated with onyx had transient bradyarrhythmias with no morbidity.

Trigemino-cardiac reflux may be related to irritation of the trigeminal ganglion or afferent fibres within the cavernous sinus, respectively by the DMSO injection, Onyx formation or sudden change in venous hemodynamics during the embolization procedure. It is most likely due to chemical irritation/neurotoxicity of DMSO on the trigeminal cranial nerve within the cavernous sinus.

One patient had worsening of proptosis with restriction of extraocular movement and blurring of vision after transarterial embolization with PVA particles which was managed with IV heparin, steroids followed by oral warfarin. Paradoxical worsening caused by SOV thrombosis may occur following TAE (112), and has been documented in a significant number (61.5 %) of patients (111).

None of the patients had any periprocedural hemorrhage/post procedural cranial nerve deficit as reported in various studies.

## *Conclusion*

This series helped us in reaching following inferences regarding clinical features, angioarchitectural features and practical management of dural CCFs based on observations and outcomes on analysis of data :-

1. Neurological deficits or intracranial hemorrhage associated with DCCFs are seldom observed (none in this series), despite a relatively frequent occurrence of cortical venous drainage (28% in this series).
2. Barrows type D is the most common type of dural CCF in this series. Transarterial embolization did not achieve complete anatomic obliteration in most of the patients but achieved clinical improvement/cure in 56% patients. It can be used in cases of Type C fistulas as well as in cases of unsuccessful transvenous embolizations. In Indian context this may also be used as a cheaper alternative to transvenous embolization.
3. The primary approach to endovascular treatment of dural CCF is the transfemoral transvenous coiling. If symptoms merit intervention, the transvenous approach offers a high success rate, close to 100% with a high rate of clinical cure (100% in this series) with a relatively lower likelihood of serious complication.
4. Onyx can be a useful adjunct to coils for transvenous embolization procedures and can improve the anatomic and clinical outcomes.
5. Visualization of a patent IPS is useful as an access route for transvenous therapy but is not required, and a thrombosed IPS can be traversed in many circumstances.
6. Atraumatic superselective micro catheterization of the cavernous sinus is a key point in the endovascular treatment of dural CCFs through the transvenous approach and requires neurointerventional skills as well as thorough knowledge of anatomy and angioarchitecture.

7. In addition to endovascular occlusion techniques, therapeutic options of DCCFs should encompass conservative management or alternative treatment methods such as manual compression and radiosurgery.
  
8. This series shows relatively lower complication rates in both transarterial and transvenous embolization procedures as compared to quoted rates in Literature which may be due to use of better neurointerventional techniques and hardware.

## *Representative Cases*

## Case No 1

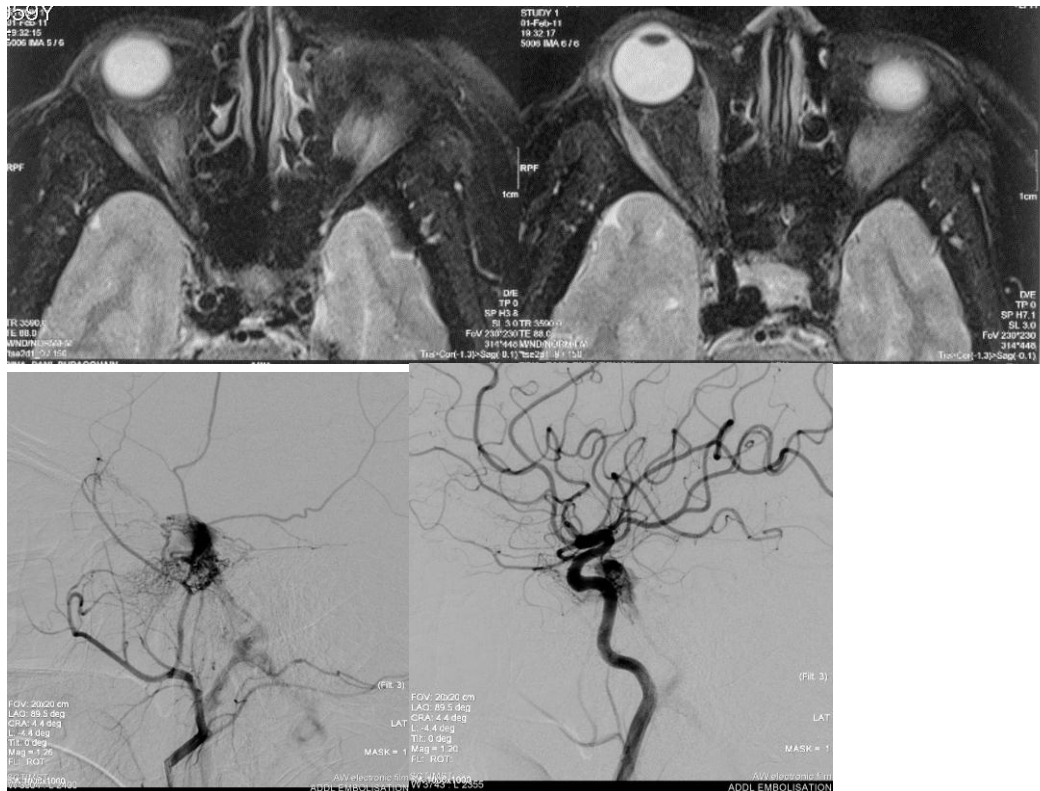
### **HISTORY:**

58 years female patient presented with complaints of tinnitus right ear for past 3 months. She also complained of swelling of the right periorbital region and diplopia since one month.

### **EXAMINATION:**

Mild swelling of periorbital region on the right. Mild chemosis of right eye was seen. Restriction of abduction, elevation and mild restriction of depression of right eye movements. There was no bruit. Proptosis of 3mm on right. Pupils bilaterally equal and reactive. VA-6/6 in right eye, 6/6 in left eye. Visual fields normal by confrontation. Fundus- Mild papilledema in right eye. No other cranial nerve deficits.

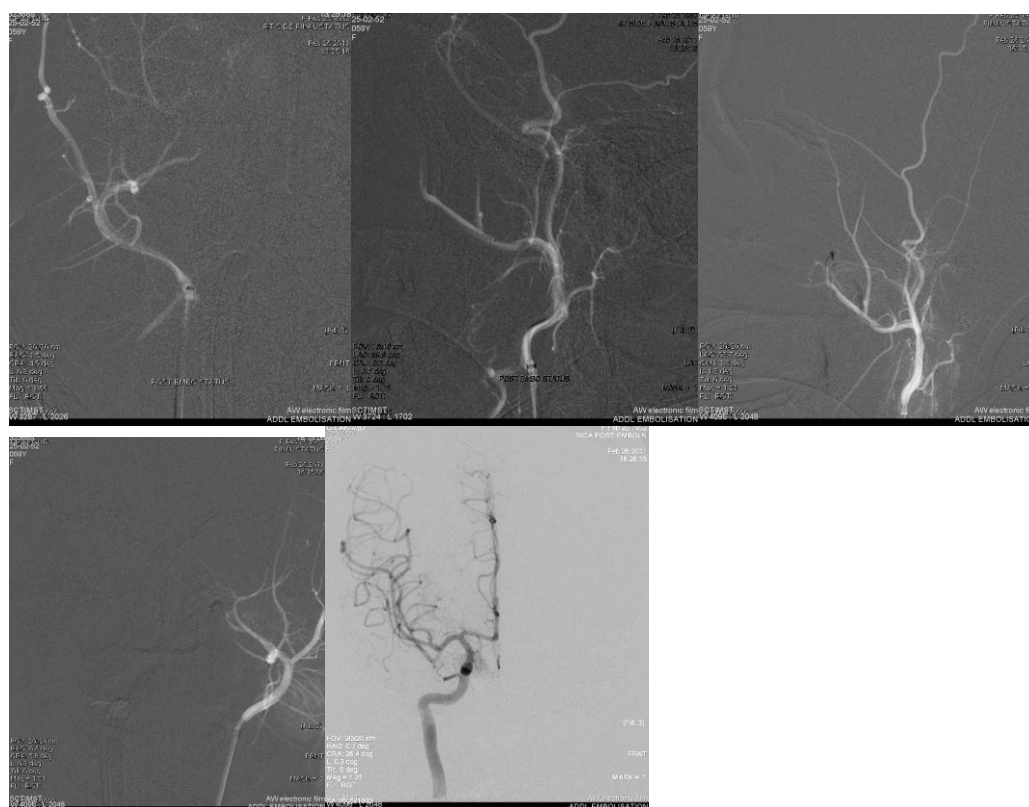
### **IMAGING**



**MRI Brain** revealed evidence of multiple flow voids in right cavernous sinus with prominent right SOV

**DSA** showed evidence of indirect carotico-cavernous fistula with feeders from dural branches of B/L cavernous ICA, bilateral MMA, bilateral Accessory meningeal artery, bilateral middle temporal arteries and right ascending pharyngeal artery. Venous drainage was predominantly through right IPS and retrograde flow through left SOV. No venous rerouting noted.

## INTERVENTION



6F vistabrite was placed in right ECA. Under road map guidance right MMA posterior branch was selectively cannulated with Rebar microcatheter and was embolized with 250-355 micron PVA particles. Using same microcatheter right AMA and middle temporal artery were cannulated separately. The AMA was embolized with 250-355 micron PVA particles followed by 355- 500 micron PVA particles. The right middle deep temporal artery was embolized with 355-500 micron PVA particles. After that the microcatheter was placed in right ascending pharyngeal artery and

embolized with 355-500 micron PVA particles. Check angiogram showed near total obliteration of the right extracranial fistulas. 6F vistabrite guiding catheter was then placed in left ECA. The left MMA posterior branch was catheterized with Rebar microcatheter and embolized with 355-500 micron PVA particles. The microcatheter was then passed distally in to MMA anterior branch. A 2mm x 10 mm fiber coil was placed in anterior branch of MMA just distal to the feeding branch origin and the feeding branch was embolized with 250 - 355 micron PVA particles. The left accessory meningeal artery and middle temporal artery were cannulated separately and they were embolized with 355-500 micron PVA particles. Check angiogram showed near total obliteration of the fistulae.

## Case No 2

### **HISTORY**

26yr old female presented with proptosis right eye since 1 month which is insidious onset, gradually progressive. There is no history of preceding trauma. There is no history of watering from eye, diplopia, ptosis or visual deficit. She also complains of redness of right eye. There is no history of headache or neurological deficit.

### **EXAMINATION**

Proptosis of right eye with dilated episcleral vessels.

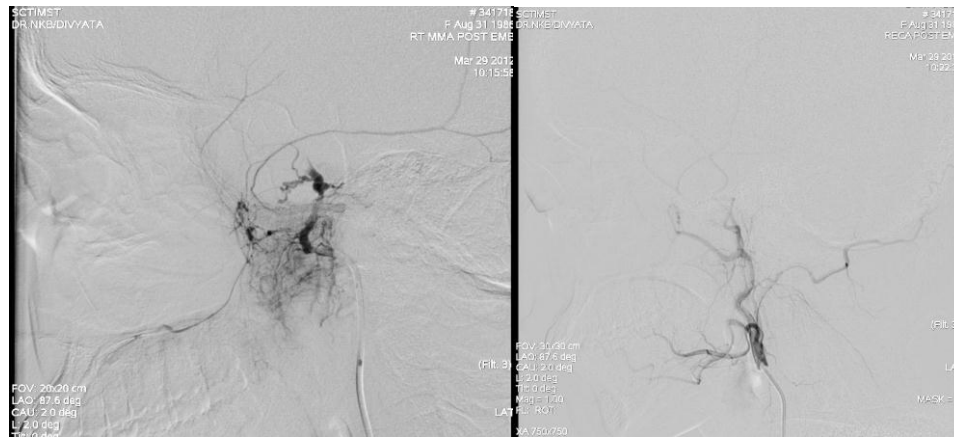
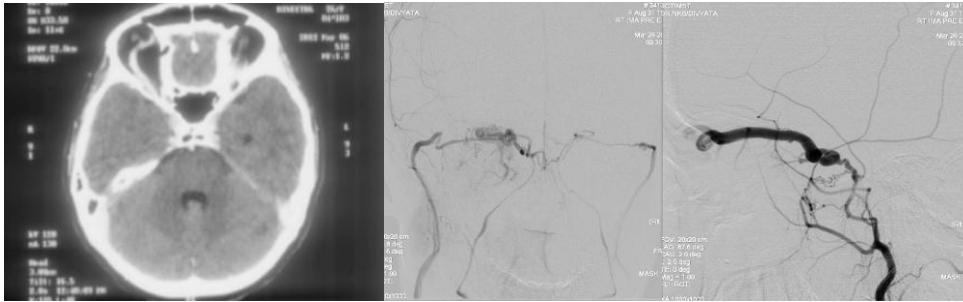
VA 6/6 in both eyes.

Extraocular movements are full.

IOP = Rt-20, Lt-14.

Bruit over right eye ball present.

## IMAGING & INTERVENTION



**CT BRAIN** - Mild proptosis of right eye ball with dilated SOV

### DSA

Angiograms showed a fistula between parietal branch of right MMA and superior ophthalmic vein. No ICA feeders are seen (Type C).

### INTERVENTION

Selective cannulation of right MMA branch was performed using 4F vertebral glide catheter. Transarterial embolisation was done using 500-710 micron PVA particles, followed by gelfoam pieces Post procedure angiogram showed complete obliteration of fistula. ICA angiogram showed no abnormality

### Case No 3

#### **HISTORY:**

48year old male patient noticed gradual, painless, progressive protrusion of the right eyeball. No h/o increase in protrusion on coughing / bending. There was associated diminished vision in the right eye with redness of same eye and diplopia.

#### **ON EXAMINATION:**

Right eye- ptosis with proptosis and chemosis present. Pupils dilated, sluggish reaction to light. VA- RE 6/24 (with correction=6/18); LE 6/5(with correction=6/5). No bruit present over right eyeball. Extraocular movements full in left eye, lateral gaze restriction in right eye

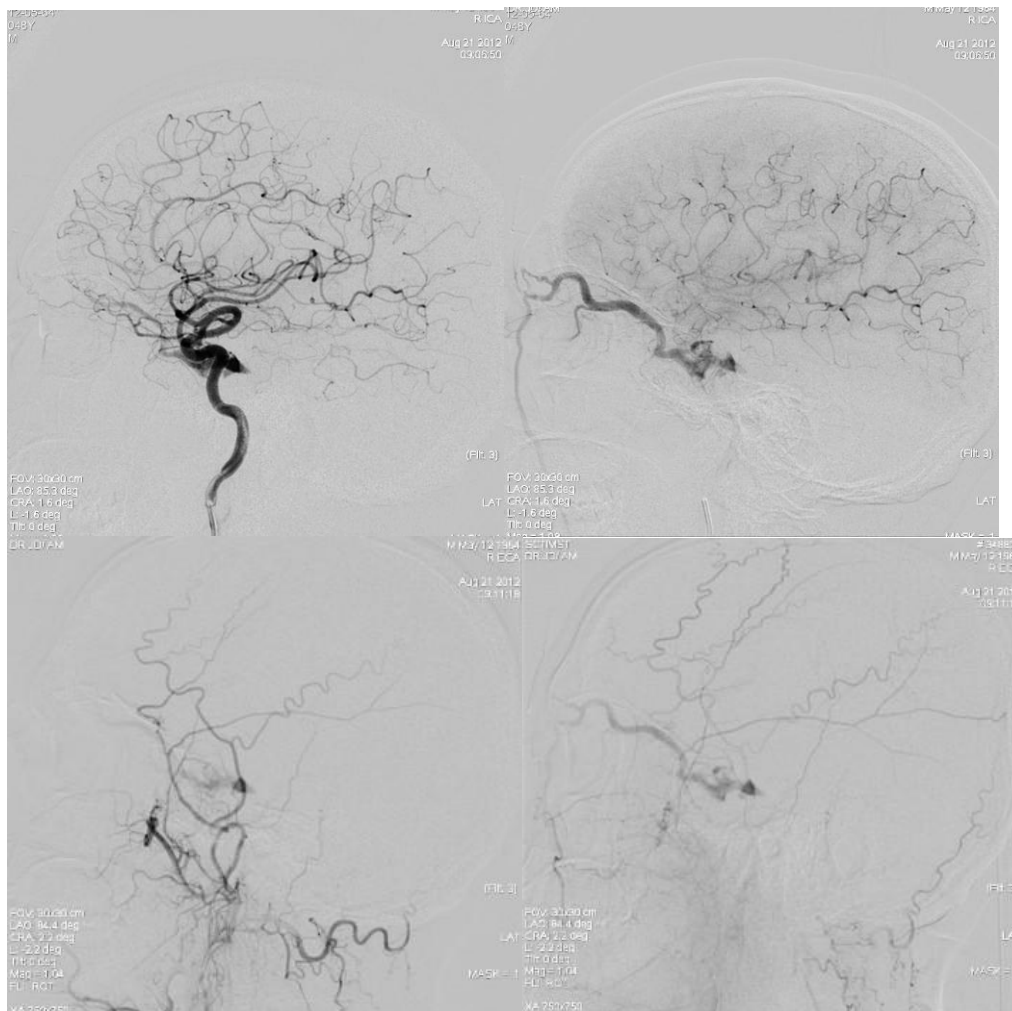
#### **CT -**

Right mild proptosis

#### **MRI-**

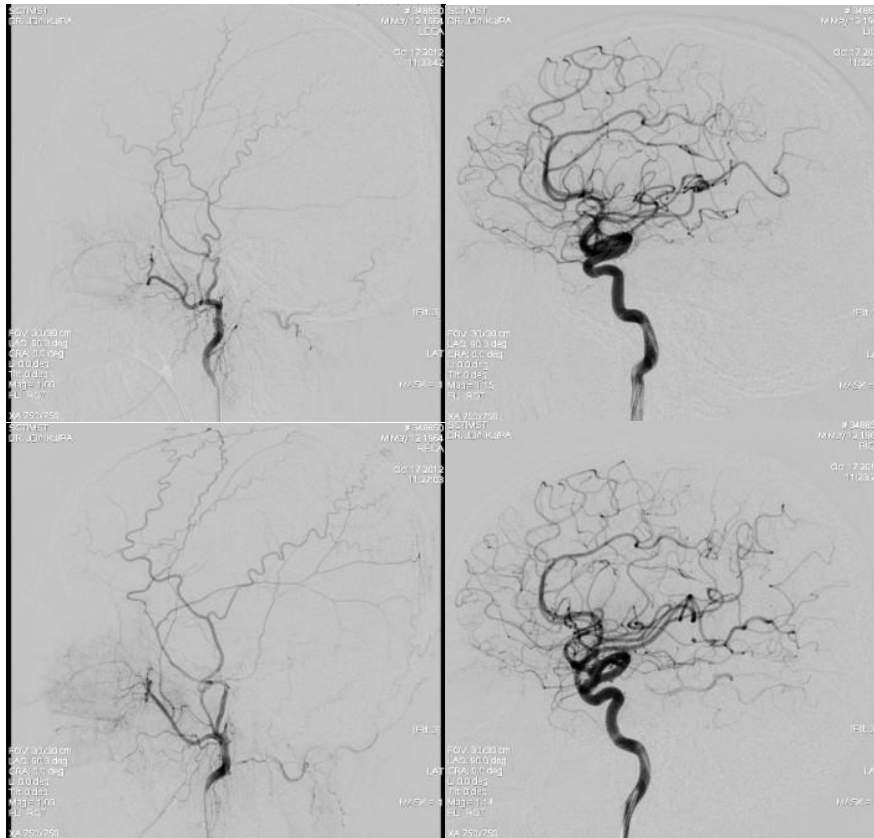
Mild proptosis of right eye with dilated arterialised right superior ophthalmic vein. Early filling and enlarged right cavernous sinus

## DSA



Angiograms revealed indirect Type D CCF on the right with feeders from the dural branches of right ICA and also from small feeders from the right middle meningeal artery. Venous drainage is through superior ophthalmic vein--> Angular vein---> Facial vein. The right superior and inferior petrosal sinuses are not opacified. There is no evidence of any cortical venous reflux.

## FOLLOW UP DSA (AFTER 3 MONTHS)



Under GA, with all aseptic precautions right CFA and CFV was punctured and 7F sheaths were secured. Using 5F VG selective angiograms of bilateral ICA, ECA and left vertebral arteries were obtained. Angiograms revealed no evidence of any carotico-cavernous fistula suggestive of spontaneous obliteration. Selective ICA and ECA angiograms done on either side didn't reveal any evidence of fistula

## Case No 4

### **HISTORY**

60yr old female, housewife redness of both eyes, dimness of vision of left eye and diplopia on looking to left side associated with pain in the left eye.

### **ON EXAMINATION**

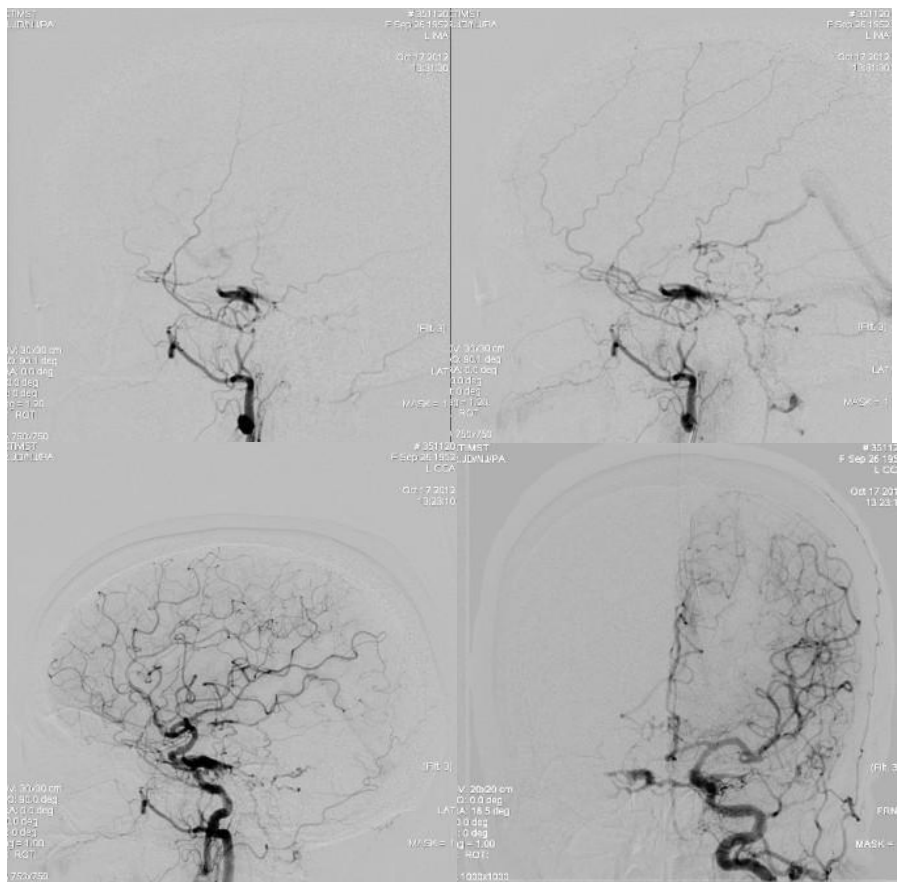
Chemosis in both eyes

Restriction of lateral abduction in left eye

Raised IOP left eye

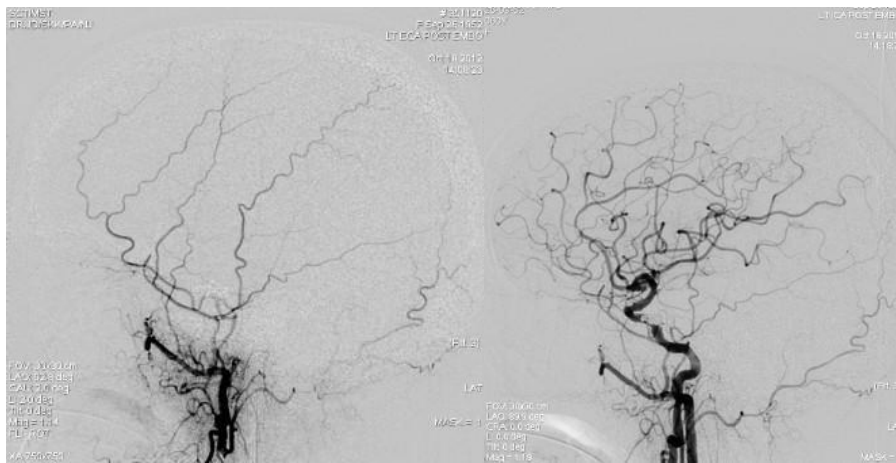
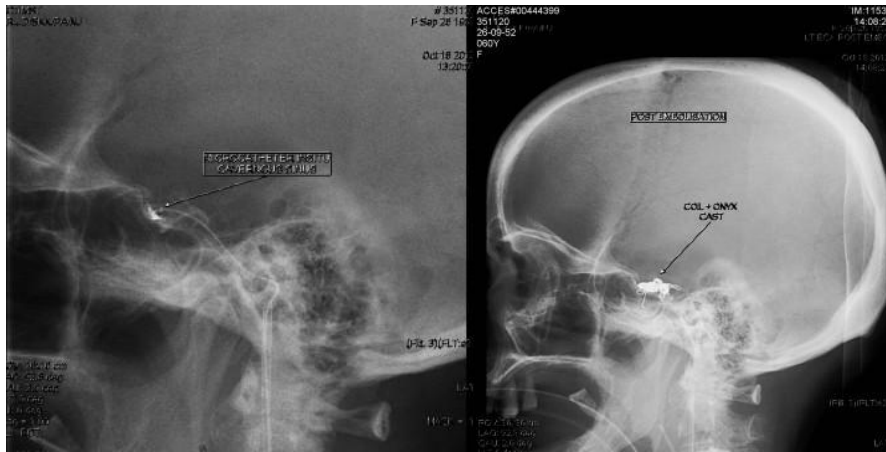
### **DSA**

Angiograms showed dural CCF on the left with feeders from small branches from parietal division of left middle meningeal artery and meningohypophyseal trunk of left ICA. Sluggish retrograde flow is seen in left SOV and across intercavernous sinus into right SOV (Late restrictive type). Leptomeningeal venous drainage is seen in the posterior fossa veins as well as into spinal perimedullary veins.



## INTERVENTION

Under GA, with all aseptic precautions, right CFA and CFV were punctured and 7F sheaths secured. Left IJV was then cannulated using 5F RC over 0.035 terumo wire. Later RC catheter was exchanged and 7F Launcher guide catheter was positioned at left jugular bulb. Left IPS was cannulated using 4F MP over 0.035" terumo wire and Rebar-18 over .014" transcend microwire. Left cavernous sinus was cannulated and tip of Rebar-18 microcatheter positioned at left half of intercavernous sinus. Starting from this point, left cavernous sinus was coiled using 9 coils. Check angiogram done after this showed mild reflux into cortical veins posteriorly. Hence, further embolisation was carried out using 0.4ml of Onyx-18. Check angiogram showed complete obliteration of fistula.



Follow up Lt ECA and ICA angiogram

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## *Annexures*

## ANNEXURE 1: PROFORMA FOR STUDY

### (Dural CCFs : Clinicoradiologic spectrum and endovascular management)

1. Hospital Number
2. Age
3. Sex
4. Clinical Features-
  - a. Orbital pain
  - b. Conjunctival injection
  - c. Chemosis
  - d. Proptosis
  - e. Eyelid swelling
  - f. Diplopia
  - g. Ptosis
  - h. Cranial nerve deficits and ophthalmoplegia
  - i. Bruit
  - j. Glaucoma
  - k. Retinal hemorrhage
  - l. Elevated IOP
  - m. Decreased visual acuity
  - n. Seizures
  - o. Intracranial hemorrhage
4. Clinical patterns
  - Orbital pattern
  - Ocular pattern
  - Cavernous pattern
  - Cerebral pattern
  - Mixed
5. Non invasive imaging - CT/CTA, MRI/MRA, Orbital Doppler
6. Angiographic features
  - p. Arterial supply – Barrows Type B, C or D
  - q. Unilateral/Bilateral Fistula
  - r. Unilateral/Bilateral supply
  - s. Proliferative/Restrictive/Late restrictive type
  - t. Venous drainage pattern – Anterior, posterior or mixed
  - f. Any cortical venous rerouting
7. Endovascular intervention
  - a. Transarterial coiling
    - i. Approach routes
    - ii. Hardware used
    - iii. Type of embolic materials used

- b. Transvenous coiling
    - i. Approach routes
    - ii. Hardware used
    - iii. Type of embolic material used
  - c. Mixed
  - d. Conservative management
  - e. Experience of the neurointerventionist
8. Immediate Post embolization status
- a. Angiographic
    - i. Complete occlusion (No residual shunt)
    - ii. Near complete occlusion (Minor residual shunt)
    - iii. Incomplete occlusion (Significant residual shunt)
  - b. Clinical
    - i. Normalized
    - ii. Improvement
    - iii. Unchanged or worsening
  - c. Any complications
9. Clinical Follow up
- a. 6 weeks
  - b. 3 months
  - c. 6 months
  - d. One year
- Angiographic follow up (if any)

## ANNEXURE 2: MASTER CHART OF THE PATIENTS INCLUDED IN THE STUDY

S No	Age	Sex	Symptom pattern	Angiographic features	Embolization	Angiographic obliteration	Clinical Follow up
1	65	F	Mixed(Orbital+cavernous)	Type D,LR	TAE	Near complete	Improved
2	67	F	Mixed(Orbital+Ocular)	Type D,R,CVR			Lost to follow up
3	37	M	Orbital	Type D, P	TVC(coils)	Complete	Normalized
4	58	F	Mixed(Orbital+Cavernous+Ocular)	Type D,R	TAE	Near complete	Improved
5	25	F	Mixed(Orbital+Ocular)	Type C,R	TAE	Complete	Normalized
6	39	M	Mixed(Orbital+Cavernous+Ocular)	Type B,LR,CVR	TVC(coils/onyx)	complete	Normalized
7	14	M	Orbital	Type D, LR	TAE(2 <sup>nd</sup> sitting with onyx)	Incomplete	Improved
8	56	F	Mixed(orbital+Cavernous)	Type D,P,CVR	TVC(Coils/Onyx)	Complete	Normalized
9	48	F	Mixed(Orbital+Ocular)	Type B,LR	TVC failed twice(Refd to RT)		NA
10	37	F	Mixed(Orbital+Cavernous+Ocular)	Type D,P	TAE	Incomplete	Normalized
11	44	F	Mixed(Orbital+Ocular)	Type D,R	TAE	Near complete	Normalized
12	48	M	Mixed(Orbital+Cavernous+Ocular)	Type D,R	Spontaneous obliteration	Complete	Normalized
13	66	F	Mixed(Orbital+cavernous)	Type C,LR	TAE	Incomplete	NA
14	51	F	Mixed(Orbital+cavernous)	Type D,LR	TAE(after TVC failed)	Incomplete	Worsening-Lost to FU
15	65	M	Mixed(Orbital+Ocular)	Type D,R	TAE	Incomplete	Improved
16	60	F	Mixed(Orbital+Cavernous+Ocular)	Type D,LR,CVR	TVC(Coils/Onyx)	Complete	Normalized
17	76	F	Mixed(Orbital+Ocular)	Type B,LR			Lost to FU
18	57	M	Orbital	Type D,CVR	TVC(Coils, Onyx)	Complete	Normalized
19	40	M	Mixed(Orbital+Ocular)	Type D,LR	TVC(Coils,Onyx)	Complete	Normalized
20	49	F	Mixed(Orbital+Cavernous)	Type D,P	TAE	Near complete	Normalized
21	46	M	Cavernous	Type D,LR	TAE	Near complete	Normalized
22	23	F	Mixed(Orbital+Ocular)	Type D, R	TAE(after failed TVC)	Incomplete	Improved
23	48	F	Mixed(Orbital+Cavernous+Ocular)	Type D, LR	Spontaneous obliteration	Complete	Normalized
24	46	F	Orbital	Type D,R	TAE(worsening)-TVC(coils/onyx)	complete	Normalized
25	28	F	Orbital	Type D, P	TAE(unchanged)-TVC(coils)	complete	Normalized
26	66	F	Mixed(Orbital+Cavernous)	Type B, LR	Carotid compression		Normalized
27	53	M	Orbital	Type D, P, CVR	TAE followed by TVC	Near complete	Normalized
28	50	F	Cavernous	Type D, P			FU NA

29	50	F	Orbital	Type D, R	TAE	Incomplete	Improved
30	65	M	Mixed(Orbital+Cavernous+Ocular)	Type B,LR	Carotid compression		NA
31	59	F	Cavernous	Type D, R	TAE	Incomplete	NA
32	53	F	Orbital	Type D, R	TAE	Near complete	Improved(FU angio-N)
33	59	F	Mixed(Orbital +Cavernous)	Type D, R			Lost to FU
34	65	F	Orbital	Type D, R			Lost to FU
35	59	M	Mixed(Orbital+Ocular)	Type D, R	TAE-TVC failed twice-refd to RT	Incomplete	NA
36	42	F	Mixed(Orbital+Cavernous+Ocular)	Type D, P	TAE-TVC(Coils)	Complete	Normalized
37	29	F	Mixed(Orbital+Cavernous+Ocular)	Type D, P, CVR	TAE	Incomplete	Improved(FU angio-N)
38	67	M	Orbital	Type D, R	TAE	Incomplete	Normalized
39	72	M	Mixed(Orbital+Cavernous)	Type D, LR	TAE	Incomplete	Improved
40	73	F	Mixed(Orbital+Cavernous+Ocular)	Type D, R	TAE	Incomplete	Lost to FU
41	77	F	Mixed(orbital+cavernous)	Type D, LR	TVC(Coils)	Complete	Normalized
42	51	M	Mixed(orbital+cavernous)	TypeD, P(LR after TAE)	TAE-2 sittings	Incomplete	Improved

### Key to Master Chart

M=Male

F=Female

P=Proliferative

R=Restrictive

LR=Late restrictive

TAE= Trans arterial embolization

TVC=Transvenous coiling

RT=Radiotherapy

FU=Follow up

NA=Not available