ENDOVASCULAR MANAGEMENT OF CAROTICO-CAVERNOUS FISTULAS

RETROSPECTIVE AND PROSPECTIVE ANALYSIS

DEPARTMENT OF IMAGING SCIENCES AND INTERVENTIONAL RADIOLOGY

SREE CHITRA TIRUNAL INSTITUTE OF MEDICAL SCIENCES AND TECHNOLOGY

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DECLARATION

I, Dr. Hima S. Pendharkar hereby declare that I have actually carried out the project "**ENDOVASCULAR MANAGEMENT OF CAROTICO-CAVERNOUS FISTULAS -RETROSPECTIVE & PROSPECTIVE ANALYSIS**" *independently under supervision and guidance in this institute.*

Date - October 2007

Thiruvananthapuram .

Dr.Hima S.Pendharkar

CERTIFICATE

This is to certify that the work contained in this thesis "**ENDOVASCULAR MANAGEMENT OF CAROTICO-CAVERNOUS FISTULAS -RETROSPECTIVE & PROSPECTIVE ANALYSIS**" *has been carried out by Dr. Hima S.Pendharkar in the Department of Imaging Sciences and Interventional Radiology, Sree Chitra Tirunal Institute of Medical Sciences and Technology ,Trivandrum ,under my guidance and is to my satisfaction.*

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INTRODUCTION

INTRODUCTION

The field of medicine has seen many a changes in the treatment of almost all human ailments over the past few years. The newer developments in technology have also benefited the medical field in every aspect.

The discovery of x-rays by sir W.C.Roentgen in 1895 revolutionized the field of medicine. With developments in imaging and technology, diagnostic radiology became the corner stone of medicine. By the early 1950s, crude fluoroscopy was available for clinical use. Following this we saw the development of angiographic studies. In 1953, Seldinger described the use of a hollow-bore needle and guide-wire system for accessing the femoral artery (1). Interventional radiology was born on January 16, 1964, when Dotter percutaneously dilated a tight, localized stenosis of the superficial femoral artery (2). Over the years, the imaging technology has seen a sea change. With the development of high-end fluoroscopic machines and the various materials available, interventional radiology has today become an inseparable therapeutic modality.

We deal with a wide variety of vascular lesions at our institute. CCFs are one of them. CCFs are acquired or spontaneous connections between the branches of cavernous internal carotid artery (ICA) and the cavernous sinus (CS). Since the introduction of the detachable balloon by Serbinenko in 1974 (3), the management of CCF has evolved from a surgical approach, usually consisting of trapping of the fistula by ligation of the ICA above and below the CS, to an endovascular approach in which one or more detachable balloons are placed inside the CS, and finally to closure of fistula by using detachable coils that are introduced into the CS either from a venous or an arterial approach.

Our centre is a major tertiary referral centre for management of all neurovascular disorders in India. The department of IS& IR has been actively involved in the endovascular management of various vascular lesions. We hereby review our experience in the endovascular management of CCFs over the last 21 years.

CAROTICO-CAVERNOUS FISTULA

CAROTICO CAVERNOUS FISTULA

CCFs are spontaneous or acquired connections between the branches of cavernous segment of ICA and the CS. According to Barrow et al, CCFs are classified into direct [type A] and indirect [types B–D] (4).

BARROWS CLASSIFICATION OF CCFs

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

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

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

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

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

A direct CCF is a high flow shunt created by direct connections between the ICA and CS. An indirect CCF is usually supplied by dural branches of the ECA or ICA but can be supplied by dural branches of both.

A direct CCF may occur as a result of trauma, a ruptured intracavernous carotid artery aneurysm, collagen deficiency syndromes, fibromuscular dysplasia, or direct surgical trauma (5). The exact cause of indirect CCF is unknown; however, factors associated with their development include pregnancy, sinusitis, trauma, surgical procedures, and cavernous sinus thrombosis (4, 6).

Typically CCFs are seen in adults and are rare in children .They occur in children with connective tissue disorders such as Ehlers-Danlos syndrome or after trauma (7).

CLINICAL SYMPTOMATOLOGY

The classical signs of CCF feature a triad of pulsatile exophthalmos, orbital bruit, and conjunctival injection. Ocular motor palsy, ocular pain, and visual impairment also occur frequently. The clinical presentation in direct CCFs is related to their size, duration, location, adequacy and route of venous drainage and presence of arterial and venous collateral vessels .It reflects the pathophysiology related to the venous drainage of the orbit.

The superior ophthalmic vein (SOV) and inferior ophthalmic vein (IOV) provide normal venous drainage into the cavernous sinus. The CS normally drains through the SPS & IPS and through emissary veins to the pterygoid plexus. Reversal of flow through the ophthalmic veins or sphenoparietal sinus is possible if arteriovenous connection develops in the CS, especially if normal routes of venous drainage of the CS are occluded.

Elevated venous pressure in veins draining the orbit may produce orbital venous congestion, transudation of interstitial fluid into the orbit with resultant proptosis, and raised intraocular pressure because of impaired drainage of aqueous humor and secondary glaucoma. Elevated venous pressure and increased intraocular pressure can compromise retinal perfusion and result in severely diminished visual acuity. Diminished arterial pressure to the orbit may also result from a "steal' away from the ophthalmic artery into the fistula through reversal of flow in the supraclinoid carotid artery into the fistula. Edema of orbital contents because of impaired orbital venous drainage can result in mechanical limitation of EOM, and can contribute to the development of diplopia. Diplopia, however may be a direct result of opthalmoplegia from a cavernous sinus syndrome, caused by mass effect on the CS from the fistula, or because of cranial nerve injury by direct trauma (8, 9). Although the sixth cranial nerve is the most often involved because of its location in the middle of the cavernous sinus near the ICA, any of the cranial nerves can be affected alone or in combination.

If there is reversal of venous drainage into the sphenoparietal sinus with resultant cerebral cortical venous hypertension or presence of epistaxis these patients are at increased risk of ICH and should receive emergent treatment.

Traumatic CCFs are usually unilateral. A bilateral traumatic CCF is rare and is generally associated with more severe head trauma and is more commonly fatal(10). Rarely, a unilateral CCF may present with bilateral orbital symptoms, by supplying the contralateral CS with arterialized blood by the way of circular sinus, thus creating an intercavernous communication (11).

Iatrogenic causes of direct CCFs are less common. These include injury to the ICA during transsphenoidal hypophysectomy (12), forgaty catheter manipulation for carotid angioplasty (13), thrombendartrectomy (14), trigeminal rhizotomy and nasopharyngeal biopsy (15).

Indirect CCFs or dural cavernous sinus fistula is clinically difficult to diagnose. The symptoms of these low-flow fistulas are not as dramatic as in traumatic high flow fistulas. Most patients do not have bruit or pulsatile exophthalmos, features that indicate a vascular lesion. Insidious onset red eye may be the only subtle finding (16).

THE ANGIOGRAPHIC FEATURES OF THE CCFs:

THE ARTERIAL SUPPLY:

In case of traumatic direct CCF, a rent in the wall of the intracavernous carotid artery or rupture of one of its branches that traverse and are surrounded on all sides by the CS, produces an arteriovenous fistula. Other causes of a direct CCF such as ruptured cavernous aneurysms also lead to the formation of an arteriovenous fistula, contained within the sinus.

The anatomy of spontaneous low flow CCFs concerns itself primarily with the small vessels emanating from the intracavernous carotid artery supplying the regional dura and cranial nerves and their collateral connections to various branches of the ECA. Angiographically, the most proximal trunk of the intracavernous carotid artery which can be implicated in low-flow CCFs is the meningohypophyseal trunk. It arises from the apex of the initial proximal curve of the intracavernous carotid artery within the CS.

the apex of the initial proximal curve of the intracavernous carotid artery within the CS. It usually gives off three branches: the tentorial artery (artery of Bernasconi-Cassonari), the dorsal meningeal artery to the clivus, and the inferior hypophyseal artery. Of these branches, the dorsal meningeal artery to the clivus is perhaps most often implicated in low-flow CCFs. This vessel may anastomose within the dura of the clivus with collateral branches from the neuromeningeal trunk of the ascending pharyngeal artery, a branch of the ECA system.

The trunk of the intracavernous carotid artery most commonly contributing to low-flow dural CCFs is the artery of the inferior cavernous sinus, also called the "inferolateral trunk" or the "lateral main-stem artery". The artery of the inferior CS corresponds to the proximal or carotid remnant of the embryonic dorsal ophthalmic artery. The main trunk of the artery of the inferior CS arises from the lateral aspect of the intracavernous carotid artery and curves over the sixth cranial nerve, after which it usually gives rise to three branches supplying the dura and cranial nerves in the cavernous sinus region .The first is a superior or tentorial ramus which supplies the roof of the cavernous sinus and the third and fourth cranial nerves as they enter the sinus. The second is an anterior ramus which usually divides into two branches: a medial and a lateral branch. The medial branch courses to the superior orbital fissure region supplying dura and the third, fourth, and sixth cranial nerves as they enter the orbit. The lateral branch is more important, and courses to the foramen rotundum which supplies dura in the region of the temporal fossa and terminates at the artery of the foramen rotundum. This branch may anastomose with the distal internal maxillary artery (IMA) via the artery of the foramen rotundum and with the middle meningeal artery via small temporal rami.

The third branch of the inferior cavernous sinus artery is a posterior branch which also divides into two rami: a medial branch to the sixth nerve, Gasserian ganglion, and motor root of the fifth cranial nerve, and a lateral branch which also supplies the Gasserian ganglion and adjacent dura. The medial branch courses to the region of foramen ovale and can anastomose with the accessory meningeal artery of the proximal IMA. The lateral branch can anastomose with the cavernous branch of middle meningeal artery (MMA) as it emerges from the foramen spinosum. The blood supply to the cavernous region thus functions as a balanced circuit interconnecting the branches of the cavernous ICA and the ECA (4).

VENOUS DRAINAGE OF THE FISTULA

The venous drainage can be anterior, posterior, inferior, superior, or contralateral. Combined anterior, posterior, and contralateral venous drainage is also often associated.

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

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

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

It is essential to analyze the angiographic anatomy of a given CCF in order to plan the appropriate endovascular therapy. The angiographic features also help to identify the patients who need an emergent treatment as opposed to those who can be managed conservatively.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

1. To describe the spectrum of angiographic features of all types of Carotico cavernous fistulas (CCFs) that were treated at the Department of Imaging Sciences & Interventional Radiology (IS&IR), Sree Chitra Tirunal Institute Of Medical Sciences and Technology (SCTIMST), over a period of 21 years.

2. To analyze the role of clinical presentation of CCFs, the angioarchitecture of the lesion, details of the procedure and management protocol of the various CCFs.

3. To review the endovascular approaches for the management of various types of CCFs.

4. To analyze the outcome of the endovascular management of CCFs.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The treatment of CCF has evolved over a period of time. Early treatment for CCF consisted of various surgical approaches. Trapping of the fistula by ligation of the cervical and intracranial ICA was introduced in the early 1930s.This was followed by efforts at embolisation using a variety of materials delivered by direct carotid exposure. By the mid 1960s, surgical techniques had progressed to a point where direct exposure of the cavernous sinus with surgical packing could be considered.

In 1971, Donald J Prolo, described the intraluminal occlusion of CCF with a balloon catheter (18).

In 1974, Parkinson reported successful direct surgical repair of direct CCFs with preservation of the parent artery (19). Subsequently, other direct interventional treatments have been attempted such as electrothrombosis with use of a copper wire inserted directly into the cavernous sinus by a stereotactic method or through the superior ophthalmic vein (20).

The first report of successful embolisation of CCF using endovascular delivery of detachable balloons is attributed to Serbinenko in 1974 (3). The procedure was refined by Debrun et al who in 1978 reported the successful treatment of direct CCF with detachable balloons (21). By the early 1980s, balloon embolisation had achieved wide acceptance as the treatment of choice for direct CCF and has now become the established treatment for direct CCF.

CCFs can be classified A) pathogenetically into spontaneous or traumatic fistulas; B) hemodynamically into high-flow or low-flow fistulas; and C) angiographically into direct or dural fistulas. (4).

Traumatic CCFs are almost always Type A fistulas formed by a tear in the cavernous portion of the ICA resulting in a high-pressure high-flow anomalous interconnection between the main arterial trunk and the cavernous sinus (6). Traumatic fistulas usually occur in young men, whereas ruptured aneurysms occur most frequently in middle-aged and elderly women (22).

In traumatic CCF, the acceleration-deceleration injury usually results in a direct fistula by causing a tear in the muscular wall of the ICA, or by completely transecting the ICA by shearing forces (23).

The majority of spontaneous CCFs are idiopathic. These idiopathic CCFs are usually low-flow and can be angiographically divided into Types B, C, or D. Various implicated conditions for their development include pregnancy, sinusitis, trauma, surgical procedures, and cavernous sinus thrombosis (4,6) .They tend to appear in middle-aged women, and generally present with insidiously progressive glaucoma, proptosis, or a "red eye" – signs and symptoms that are usually less severe than those seen in direct fistulas(4) .

Hemodynamically, high-flow fistulas fill the cavernous sinus and efferent veins within a fraction of a second and the intracranial branches of the ICA fill partially or cannot be visualized. An angiogram of a low-flow fistula will reveal slower drainage into the venous system and filling of the intracranial branches of the ICA (4).

Type A fistula rarely resolves spontaneously and requires immediate treatment if there is progressive visual loss, an intolerable bruit, headache, corneal exposure, severe retro-orbital pain, or cortical venous drainage (24). On the contrary, the spontaneous dural CCFs have a tendency to resolve without treatment .Still a sizeable number of patients with spontaneous CCF will suffer progressive loss of vision, diplopia, or intractable glaucoma and hence warrant interventional therapy (4).

Transarterial embolisation with detachable balloons is considered the best initial treatment for direct CCFs. The goals of treatment are to eliminate the fistula and maintain the patency of the ICA (24).

However, preservation of the ICA may be nearly impossible with detachable balloons when there is a large tear or a nearly complete transaction of the ICA. In these cases, the balloon may protrude into the lumen of the ICA and create a significant stenosis. Stenosis reduces flow through the ICA and turbulence around the balloon, creating ischemic episode or stroke. Also, the balloon may shift, resulting in ICA occlusion, or may migrate distally into the distal cerebral vasculature.

Balloons that are inflated only with iodine contrast material (iso-osmolar to blood or not) and detached, shrink progressively and are totally deflated after 1 month. If they deflate too quickly, a venous pouch or false aneurysm will occur. When this pouch is small, it remains asymptomatic for years and does not alter in size. Big pouches, between 1 and 2 cm in diameter, generally occur a few days after occlusion of the fistula because the balloon has deflated too quickly. The fistula continues to be occluded, but the false aneurysm develops in place of the inflated balloon, and the pouch is the same size as the initial size of the inflated balloon. The pouch can induce intractable retro-orbital pain or oculomotor nerve palsy. If this complication occurs, it is necessary to permanently occlude the carotid artery and the neck of the false aneurysm (17).

Once occluded, the long-term risks are low for fistula recurrence, symptomatic pseudoaneurysm formation, or cerebral ischemia after transarterial embolization using either latex or silicone detachable balloons (24).

Balloons, however, have inherent disadvantages: premature detachment may occur, or when the balloon does not occlude the fistula but blocks part of the venous drainage, rerouting of venous drainage to cortical veins may lead to hemorrhage. Distal embolisation of a detachable balloon may occur or a pseudoaneurysm may form if the balloon deflates prematurely (25).

In certain cases, ICA occlusion by surgical means may be needed. However, proximal ligation of the ICA by a surgical approach has been shown to be ineffective; in addition it prevents endovascular access to treat the fistula and may induce a severe steal phenomenon (26).

The spontaneous closure of direct CCFs is considered rare. Predisposing factors leading to spontaneous closure of direct CCFs are traumatic and iatrogenic ICA dissections, fistulas with a small ostium, partial balloon occlusion (>95%) closure, and attempts to place the balloon (24).

Goto and colleagues described that direct fistulas with high flow have approximately a 3% rate of resolution without surgical intervention (27).

Aneurysms of the cavernous segment of the ICA usually become symptomatic by mass effect on the adjacent cranial nerves, causing ophthalmoplegia and, sometimes, decreased vision or trigeminal neuralgia. Rupture of an intracavernous aneurysm leads to carotid cavernous fistula (CCF), epistaxis, or subarachnoid hemorrhage (SAH) if the aneurysm extends through the dura into the subarachnoid space. The preferred treatment is endovascular, either by selective occlusion of the aneurysm or endovascular occlusion of the parent internal carotid artery, with or without previous bypass surgery. The primary goal of treatment of CCFs caused by ruptured cavernous sinus aneurysms is closure of the fistula by balloon or coil occlusion of the aneurysm or occlusion of the carotid artery by trapping the fistula. In the authors series the incidence of CCF by a ruptured cavernous sinus aneurysm was 1.5% while CCF was the presenting symptom in 24.4% of treated symptomatic cavernous sinus aneurysms (27a).

Indirect CCF are rarely life threatening. They being low flow arteriovenous shunts, may thrombose spontaneously in time (28).

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

Seeger et al believed that a spontaneous cure of dural cavernous fistula in most cases is due to partial or complete thrombosis of the cavernous sinus and/or its tributaries (30).

The persistence of the physiological venous flow from the cavernous sinus to the petrosal sinuses or a thrombosis upstream to the dural fistula may be factors favoring spontaneous occlusion (31).

A high frequency (85%) of thrombosis of both the ipsilateral inferior and superior petrosal sinuses associated with DCCF has been noted. However, whether petrosal sinus thrombosis is a cause or a consequence of the DCCF is as yet unknown (31). With manual jugular and carotid compression, spontaneous fistulas may have upto a 30% rate of resolution (23).

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

Serbinenko described the use of detachable balloon to occlude the cavity of arterial aneurysm or the afferent vessels of arteriovenous aneurysms. They also described the balloon as a useful means to shut off the blood flow to arterial aneurysms and CCF when access is difficult (3).

Ohta et al described a traumatic CCF treated with polyurethane foam attached to a monofilament suture, obviating the need for ligating the ICA in the neck (32).

Mullan described the treatment of carotid cavernous aneurysms by attempting to occlude the aneurysm while preserving carotid flow. They described wire thrombosis, needle thrombosis, and balloon thrombosis (33).

Debrun et al described the treatment of 54 traumatic CCFs with detachable balloons .they described 3 approaches for balloon placement - endarterial route, venous route thro- IJV, IPS and CS , or surgical exposure of CS with occlusion of fistula by detachable balloon directly positioned in the CS (17).

Vinuela et al described the management of 20 spontaneous CCFs. 17 were unilateral & 3 bilateral .These DAVF were treated with PVA and IBCA. Two patients underwent post embolisation surgical procedures. They used detachable balloons to occlude the fistula associated with 2 giant intracavernous aneurysms and a small dural intracavernous AVM. 8 patients received no therapy. In 2, spontaneous obliteration of the fistula occurred. Of the 9 cavernous AVM embolised with particles or IBCA, complete embolisation was achieved in 7 cases and partial embolisation followed by surgery in 2 cases. Successful balloon obliteration of the giant intracavernous aneurysm was obtained in two cases .In one patient right hemiplegia with aphasia resulted from reflux of IBCA emboli through the artery of foramen rotundum to the MCA (28).

Debrun et al described the treatment of 132 CCFs. Ninety two of 95 traumatic CCFs were treated by detachable balloons. Direct surgical exposure of the cervical or cavernous internal carotid artery was necessary in the remaining 3 pts who had undergone unsuccessful surgical trapping. Three ruptured cavernous aneurysms and 2 spontaneous CCF had type A connections. They observed no type B fistulas, 4 type C CCFs and 28 cases of type D CCFs. These patients with type D CCF underwent several attempts at embolisation with total eradication of the fistula in 12 of 25 cases. There were 3 spontaneous cures in type D CCFs (34).

Adam Lewis et al described the treatment of 100 direct CCFs. Of these 100 CCFs, 76 were traumatic , 22 resulted from a ruptured aneurysm and two were iatrogenic .The most common presentation was orbital bruit 80%, proptosis 72% chemosis 55% , abducens palsy in 49% and conjunctival injection 44% . They described the use of balloons in 88 fistulas, ICA flow was preserved in 66pts (75%). Spontaneous closure was reported in 4 patients, surgical closure in 5 pts and use of non detachable balloons in two patients.

They stated that the direct CCFs can be treated by the transvenous route if the ICA is occluded or narrowed, is tortuous, with a failed transarterial attempt or fistula recurrence after trapping. This needs the IPS or SOV to be patent to approach the cavernous sinus.

However, the pitfalls of the transvenous approach include converting the cavernous sinus into an aneurysm by blocking all venous drainage without occluding the fistula .Occluding the posterior venous drainage results in diversion of flow into the ophthalmic veins and worsening of vision loss or ocular symptoms .Similarly, occluding the anterior drainage may increase cortical or petrosal venous drainage and cause cerebral or brainstem ischemia.

Surgical approach was described by the authors for cases in which transarterial and transvenous route both failed. A standard pterional craniotomy with a Dolenc approach is sufficient to unlock the cavernous sinus and provide access to the fistula (24).

D.Lo reported a case of traumatic CCF due to gunshot with associated carotid dissection. Their patient had an ipsilateral ICA occlusion secondary to dissection thus preventing ipsilateral approach. They treated the patient by contralateral arterial approach accessing the fistula via ACom and coiling it with complete closure of the fistula. The ocular symptoms resolved completely (35).

However, closure of CCF with coils from an arterial route poses many problems. The CS with a long standing fistula may be quite dilated and variable in dimensions. In addition, the fistula may immediately communicate with a wide variety of venous drainage pathways .Closure of the fistula close to the orifice can be difficult. If the microcatheter recoils during placement of the coils, then a part or entire coil can get deposited in the ICA. This can be minimized by using shorter coils, placement of a non detachable balloon in the ICA across the origin of the fistula during coil placement and the use of a Teflon pusher wire.

In some of these cases, occlusion of venous draining pathways may be attempted. However, the problem in occluding the draining venous pathway is the high compliance of the vein or sinus which can allow its dilatation and subsequent migration of emboli. The SOV dilates anteriorly thereby increasing the risk of migration of emboli placed posteriorly in this structure (36).

The placement of wire coils into the CS (intraoperatively) to produce the closure of a CCF was first reported by Hosobuchi (37).

Rarely transarterial embolisation may be unsuccessful. The fistula orifice may be too small to allow entry into the CS, the venous compartment may be too small to allow balloon inflation or sharp object may puncture the balloon during inflation. In some patients who have had prior balloon embolisation , with subtotal occlusion, navigation of additional balloons into the fistula is often unsuccessful owing to the presence of embolic material (balloons partially blocking the fistula orifice) .This then needs alternative routes for treatment of the fistula.

Guido Guglielmi et al described a case of CCF caused by a ruptured intracavernous aneurysm treated with electro thrombosis by the detachment of two platinum coils into the aneurysm via an endovascular transvenous approach. The small size of the fistulous communication prevented the introduction of a detachable balloon into the fistula through a transarterial approach and hence a transvenous approach was used with complete closure of the fistula. The advantages of using electrodetachable platinum coils include thrombogenicity, controllable deposit, radiopacity, and biocompatibility. Limitations of using GDC include the loss of the coil during its deposition. This complication is rare in present days in view of the expertise and technical developments (38).

G.Bavinszki described their initial experience in treatment of post traumatic CCFs using GDC coils in six patients. The transarterial route was chosen in five and a transvenous in one case. In one of their patients the intracavernous carotid artery was occluded by balloons after coil embolisation because of improper coil position and the fear of possible thromboembolic events. They followed all patients with angiograms at 1 & 6 months after therapy and demonstrated complete cure in all 6 patients Regarding the extent of occlusion, the authors state that partial CCF occlusion with GDC is sufficient to induce and enhance progressive thrombosis of the fistula (39).

R.L. Young et al described the case of a woman with no h/o collagen vascular disease who developed dissections of both carotids and both VA as well as a direct CCF after a motor vehicle collision. The patient was managed conservatively with heparinisation and a follow up angiography at 15 weeks demonstrated significant resolution of the dissections as also complete resolution of the CCF (23).

V. Halbach states that if proximal carotid occlusion has occurred, a safe transarterial approach may be impossible. The transvenous route has few concerns. Although arterialization of venous structures can occur in long-standing shunts, the draining veins and dural sinuses are still thin-walled and can be perforated by catheter and guidewire manipulations. With increased pressure and flow of the arterialized blood in these structures, small perforations can result in rapidly fatal hemorrhage. Hence, caution needs to be exercised when approaching via the venous route. He stated that if the posterior drainage is closed, a contralateral inferior petrosal sinus or superior ophthalmic vein approach is used. If the superior ophthalmic approach fails and the vein is occluded, fistula flow may be diverted into cortical veins, increasing the risk of hemorrhagic complications (41).

The traditional approach through the transvenous route to the CS is via the IPS. The IPS route has certain advantages - a) It is less time consuming especially when it is draining the fistula. b)-The anatomical route is straight and does not cause significant friction inside the catheter. c) - It allows the stable positioning of the guiding catheter inside the IJV which facilitates catheter navigation to the I/L or even C/L CS. Even if thrombosed, it can be used as a route for embolisation (42).

In their cases the angiogram did not show opacification of the IPS, indicating that it neither drained the arteriovenous fistula nor the cerebral venous outflow. The authors state that it should be clearly distinguished if non-opacification of the IPS occurs during the early arterial phase (draining the fistula) or in the late venous (draining the brain) or in both phases. Cases belonging to the second group (not draining the fistula but draining the brain) have a better chance of successful catheterization to the CS.

If the IPS does not serve as a venous outflow at all , then it is highly likely that this sinus has undergone subtotal or complete thrombosis .However this does not preclude the existence of anatomic connections and therefore the possibility of reaching the fistula site with a suitable microcatheter . A large volume jugular bulb phlebogram may be useful in visualizing a tiny vascular structure as a remnant of the thrombosed IPS which can then be successfully catheterized The authors also state that in 31% of cases, the IPS cannot be catheterized due to its plexiform architecture and lack of connections with the IJV. Nevertheless it is not possible to predict if a catheter can successfully be navigated through the IPS in a given case (42).

Halbach first described 2 cases in which the IPS was not demonstrated by arteriography but successful catheter navigation through the non opacified IPS was possible (41). Yamashita and collegues were able to catheterize the IPS in 2 patients whose sinuses were not demonstrated in angiography (43).

Apart from the IPS route, the SOV route is also well established. Derang et al (44) described the treatment of CCF via the retrograde SOV route. According to them

indications for embolising CCF though the SOV are -

1. The SOV is the main draining vein, obviously dilated & arterialized. Symptom

duration is over 3 months (45,46).

2 Type A CCF, which is difficult, dangerous, and frustrating to embolise via the arterial route, and is prone to recur after embolization via arterial route (47).

3 Type B, C and D CCFs that are not spontaneously cured after six months.

The venous drainage of CCFs generally falls into six categories. SOV is the main draining vein in most cases. Superficially, the SOV is a confluence of the medial angulus oculi vein and superior orbital vein. It perforates the orbital septum superior to the ligament of medial angulus oculi, goes into the orbit, then reaches the lateral aspect of the superior rectus muscle, and connects the CS through the superior orbital fissure medial to the III, IV, VI cranial nerves. Because of the increase of local arterial blood pressures, SOV becomes dilated, thickened, strengthened and arterialized. The SOV has no valve, is relatively straight and can be arterialized. If the venous drainage of the CCF is a mixed type, or if the history is not long enough, the wall of the SOV may not be thick enough, increasing the risk of rupture during the operation. In these conditions, it is better to delay treatment for 3 months until the wall grows thicker and the lumen becomes bigger. If the SOV ruptures, it is very difficult to stop the bleeding and formation of an orbital hematoma will damage visual acuity.

The advantage of the SOV route is that is preserves the arterial supply, while taking less time. The embolization is done only in the venous system, reducing the opportunity for occlusion and vasospasm of ipsilateral arteries (44).

Miller et al described the technique to access and cannulate surgically the SOV, to reach the CS (47). They described the treatment of 12 patients. These patients were treated successfully by advancement of a detachable balloon catheter through the I/L SOV into the CS with subsequent inflation and detachment of the balloon or by introduction of multiple of coils into the fistula via the I/L SOV. All patients had complete resolution of signs and symptoms after successful occlusion of the CCF .There were no intraoperative complications. They stated that in some cases the SOV approach is not only less time consuming, but easier for the patient than attempts to perform embolisation of multiple ECA feeding vessels (47).

SOV cannulation can be facilitated by various means a)- ensure adequate hydration of the patient with I.V.fluids to open the venous channels, b)- use GA for the patient during the procedure for relaxation of the facial muscles and c)- map the patients facial veins by MRI before starting the procedure (48).

At times, there might be no transarterial or transvenous access. In such cases, a percutaneous puncture of the SOV can be performed deep in the orbit, permitting venous occlusion without complications. The authors described that if surgical exposure or percutaneous puncture of the SOV does not allow passage of a microcatheter to the cavernous sinus because of mechanical obstacles, direct puncture of the posterior SOV behind the globe may afford access and obviate a craniotomy .They stated that the gentle manipulation of 21 G needle through the orbit for puncture of an arterialized vein is safe (49).

Apart from SOV route, other venous routes have also been described in the literature. Venture et al described endovascular treatment of a CS dural arteriovenous fistula by transvenous embolisation through the SOV via cannulation of a frontal vein (50). Percutaneous transfemoral embolization of an indirect CCF with cortical venous access to the CS has also been described (51).

In cases of spontaneous CCFs, the indications for urgent treatment include visual decline, which can progress rapidly, and if it is severe (to the point of light perception only) it is often irreversible, even with prompt closure of the fistula. Any patient with angiographic findings of a cavernous sinus varix should have emergent treatment to eliminate the risk of subarachnoid hemorrhage. Patients with angiographic evidence of a pseudoaneurysm have a grave prognosis and should also receive immediate treatment. Differentiating the two, the onset of the pseudoaneurysm is usually coincidental with the trauma, as opposed to the delayed onset of a varix, which develops after the occlusion of venous outflow pathways (52).

Spontaneous CCFs have been reported to close with carotid – jugular compression .In the author's series, combined carotid and jugular compression resulted in complete cure in 7/23 patients and improvement in one additional patient . There were no complications in these patients. Patients in whom carotid –jugular compression failed or who demonstrated CVR, or visual decline, were treated with endovascular embolisation (52).

Once clinical cure of the symptoms has occurred, suggesting closure of the fistula, a check angiogram need not be done. Follow-up angiography was performed by the authors only if patients had persistent symptoms or new symptoms, or if the control angiogram obtained after embolization demonstrated subtotal occlusion (52).

In a retrospective analysis of 27 patients with dural CCFs by M.Theaudin 20 patients received endovascular treatment via a transvenous route (n=16) or via a transarterial route (n= 4). Complete occlusion of the fistula was obtained in 14/16 patients treated by transvenous approach and in 1 out of four patients treated via transarterial approach. Seven patients did not receive any endovascular treatment. Three had spontaneous obliteration and 4 had minor clinical symptoms and no leptomeningeal venous drainage. 16 pts had early clinical improvement after endovascular treatment. One had a cerebral hemorrhage after transvenous embolisation of DCCF with leptomeningeal drainage.On follow up, all patients treated by the transarterial route remained symptomatic while 10/14 patients treated via transvenous route were asymptomatic (31).

As regards the extent occlusion of a dural CCF, subtotal occlusion is often sufficient for clinical improvement and subsequent angiographic and clinical cure is common (52).

CCFs can be bilateral. They can be post traumatic or can be seen if there is bilateral CS venous drainage of a unilateral CCF.

Describing bilateral CCFs, the authors stated that the presence of bilateral fistulae imposes further stress on arterial and venous hemodynamics. In this situation, neither carotid artery can be relied on to support its fellow in maintaining cerebral perfusion and a greater burden is placed on the vertebral system. Further, U/L ICA occlusion may increase the intravascular pressure on the contralateral side sufficient to expand the shunt (54).

A.M. vd Vliet discussed two cases with bilaterally located CCF. One was traumatic and the other spontaneous. Most traumatic CCFs are direct, U/L and of a high flow type. The first case described was a traumatic B/L direct fistula with a low flow. The second case, had bilaterally located fistulas occurring spontaneously. Peculiarly they were direct, instead of indirect, as is to be expected in spontaneously developing fistulas and of a low flow type. Both cases are rare. The authors managed both the patients conservatively and state the point that conservative therapy must be considered in low-flow fistulas, because they have a high tendency towards spontaneous resolution (55).

Cases of a double traumatic CCF on the same side and the balloon occlusion of the fistula have been described by S.Kumar et al (56) and Wilms G (10).

Significant advances in stent and catheter design now make it possible in many instances to deploy either balloon-expandable or self-expanding stents designed for use in the coronary arteries in the cavernous and supraclinoid segments of the internal carotid artery. With these devices, it is possible to reconstruct a severely injured artery Gary Redekop et al have described the use of stents to treat traumatic aneurysms and AVF of the skull base in six cases (57).

A step forward, the traumatic CCF can be treated by stents.

The authors treated two patients with unilateral traumatic CCF who had a failed occlusion with detachable balloons and coils. The authors used a self expanding covered stent to obliterate the fistula. Covered stents can be used as a primary therapeutic modality in cases in which detachable balloons or coils with or without bare stent have failed to obliterate the fistula (58).

The development of the permanent liquid agent Onyx for embolisation of AVMs drastically changed the management strategies of AVMs. Onyx can also be used to treat CCFs. Using a stent placed across the orifice of the fistula may prove safe to occlude the CS with this novel agent. Though the technique has not yet been attempted with traumatic CCF, but it seems likely that it would succeed if the origin of the fistula remains below the carotid siphon (59).

CCFs are rare in children and occur in those with connective tissue disorders such as Ehlers-Danlos syndrome or after trauma. Spontaneous childhood fistulas are extremely unusual, the youngest reported patient being in a 5-year-old boy (60). The authors report a case of an 11-month-old female patient with a symptomatic direct CCF without a preceding history of trauma or coexisting collagen vascular disorder (61).

There have also been several case reports of patients with direct CCF as a result of iatrogenic ICA laceration associated with intracranial angioplasty or after transsphenoidal surgery(TSS) for pituitary adenoma treated by various methods .

Raymond et al (62) reported that arterial injuries during or after TSS occurred in about 1% of cases and that they were associated with notable morbidity (24%) and mortality (14%). Factors predisposing to ICA injury have been well described (63).

Carotid artery injury occurs most frequently a few millimeters below the origin of the ophthalmic artery (62). Angiographic findings of iatrogenic ICA injury after TSS may include the following: contrast material extravasation within the sphenoid sinus, carotid artery occlusion and/or stenosis, false aneurysm in the carotid artery, and CCF (62). CCF is a rare complication associated with TSS, and it is reported infrequently in the literature (64,65). Iatrogenic ICA injury may create a communicating channel between the sphenoid and/or cavernous sinus and the sidewall of ICA. Such an event may present as an acute hemorrhage, false aneurysm, or CCF. If the fissure communicates with only the cavernous sinus, the result is a CCF .CCF is the only type of arterial injury for which treatment with detachable balloons or coils is appropriate. If severe hemorrhage occurs during TSS, intraoperative packing is the first and often the only immediate step that can be taken. When packing completely fails to control the hemorrhage, the situation is acutely life threatening and needs occlusion of the ICA immediately (63).

The transarterial route has been used successfully in many case series for balloon embolisation; however, there may still be a need for parent artery occlusion in a substantial percentage of patients with type A CCF who require occlusion of the ICA for successful treatment.

Stuart Coley et al describe trapping of the CCF in four cases .They elected to occlude the carotid artery proximal to the fistula with detachable balloons and then place coils distal to the fistula via the Acom {three cases}or PCom {one case} (59).

Ehler's Danlos syndrome is a widely recognized connective tissue disorder with various subtypes of which type IV has florid vascular manifestations. van Meckeren in the late 1860s first described individuals with extraordinary skin elasticity who may represent the of Ehlers-Danlos syndrome (66) . EDS 4 subcategory is the most malignant form of EDS because of the high likelihood of developing spontaneous blood vessel rupture from aneurysms, dissections and transmural tears. Other fatal complications of EDS 4 include hollow viscera rupture, mitral valve prolapse, and spontaneous pneumothorax. Its biochemical cause rests upon an abnormality in the gene that codes for the pro L1 (III) chain in type 3 collagen, which is a major component of distensible tissues such as skin, blood vessels, and hollow viscera. The responsible allele, COL3A1, is located on chromosome 2. From a structural standpoint, vessels in EDS 4 have reduced total collagen content, thin walls with irregular elastic fibrils, and reduced cross-sectional area. Each of these abnormalities predisposes patients to aneurysms, dissections, spontaneous ruptures, and fistula formation (67).

North et al reviewed 202 patients with EDS 4, 19 of whom (9.4%) had cerebrovascular complications that included fistulas, aneurysms and dissections(67). Debrun et al (68) and Graf (69) reported three and two cases of spontaneous DCCF, respectively. Halbach et al reported on four patients with EDS 4 in their series of 212 patients with DCCF (70).

Almost all reports of patients with symptomatic EDS 4 refer to the danger inherent in performing angiographic procedures through fragile vessels. In Freeman et al's series (71), 18 patients underwent angiography with a 22% major complication rate and 5.6% death rate. Schievink and colleagues (72) reported a 35% morbidity rate and 12% mortality rate with diagnostic angiography, and a 17% death rate as a result of interventional therapy. Halbach et al's series of four EDS 4 patients with DCCF had a 50% major morbidity rate and 25% mortality rate (70). In 1967, Schoolman and Kepes (73) reported the first case of bilateral DCCF and EDS 4. Immediately after angiography, the patient developed chest pain and died secondary to an intimal tear of the ascending aorta. One of two patients reported by Graf in 1965 died secondary to cardiac rupture (69). Michael H. et al describe two patients of EDS who succumbed to remote catastrophes, one had a cardiac papillary muscle rupture and the other iliac artery perforation (74).

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

MATERIALS AND METHODS

MATERIALS AND METHODS

This was a retrospective study carried out at the department of IS & IR, at SCTIMST, Trivandrum. For the purpose of this study, patients who had undergone endovascular management for CCF at the department of IS & IR, SCTIMST, dating from January 1986 to 31st August 2007 were reviewed.

The clinical data of the patients was obtained by reviewing their case sheets obtained from the Medical Records Department (MRD) & the imaging data was obtained from the DSA lab records. From these, the data regarding clinical presentation, angiographic characteristics, and details of the embolisation procedure, post procedural details and also the follow up data was collected. The patients were advised follow up at 1 month, 6 months and one year and yearly thereafter. For the purpose of this study, the patients were followed up with questionnaires.

A total of 145 cases of CCF were treated at our institute. Of these, the records of 129 patients were available for review.

All patients underwent a complete neurological evaluation prior to the procedure. The patients had a baseline imaging done – either a CT or MRI of the brain. Subsequently, the patients underwent a complete six vessel diagnostic cerebral angiogram. Initially for a period of three years (1986-1989) the embolisation was carried out under fluoroscopic guidance. From 1989 to 1995, the procedures were carried out on Digitron Siemens and later from 1996 till date we have been using Advantx digital substraction angiography unit by GE (GE Milwaukee, USA). A wide variety of catheters and embolic material was used for the procedures as was best suited for the individual patient depending on the angioarchitecture.

In most of the patients, the procedure was done under local anesthesia after premedication (Inj. Pethidine 50mg i.m & Inj. Phenergan 25mg i.m for adults , and as per body weight for children). Post procedure these patients were managed in the wards. For procedures carried out under general anesthesia, the patient was monitored in the neurological intensive care unit.

After discharge, the patients were followed up with clinical evaluation for improvement in their symptoms. The patients were followed with a color Doppler for imaging evaluation. Follow up angiograms were not performed in all patients but only in those cases where recurrence was suspected.

PATIENT EVALUATION

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

DIGITAL SUBTRACTION ANGIOGRAPHY

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

Each type of lesion, however presents its own sets of special considerations that should affect how the angiographic evaluation is conducted. Ideally, the initial angiographic work up should be sufficient to both establish a diagnosis and plan therapy.

Demonstration of site of fistula

In direct CCFs the demonstrationof the site of CCF is very essential. In high flow DCCF, at times, it may not be possible to identify the morphology of the fistula on selective ICA angiogram without specific maneuvers to slow the flow through fistula.

The Mehringer – Hieshima maneuver:

It consists of a gentle ipsilateral ICA injection and manual compression of the ipsilateral carotid artery, while filming over the fistula. By eliminating the high flow arterial input from the ipsilateral carotid artery, the fistula fills at a slower rate, which allows for better delineation of the fistula site.

Alcock's maneuver:

Injecting the dominant vertebral artery while compressing the ipsilateral carotid permits contrast to fill the ICA from above by way of the Posterior communicating (PCom) artery. The retrograde opacification of the carotid stops at the exact point of the fistula where cavernous sinus opacification begins.

These maneuvers demonstrate the site of fistula in direct CCFs with great precision. Also with these maneuvers, the unusual case of a CCF with more than one tear or complete transaction of the ICA may be revealed. Having demonstrated the fistula/ feeders to the CCF, further embolisation can be planned.

Technique of embolisation:

The procedures were done under local / general anesthesia. A guiding catheter was placed in the cervical ICA. For direct CCFs, detachable balloons were mounted on the microcatheters. [Previously we used coaxial double microcatheters for balloon attachment with latex threads (Nycomed, France), but now we prefer to use single microcatheter (BALT extrusion, France) for balloon deployment]. The balloon-mounted microcatheter is then negotiated slowly into the fistula by flow guidance. Some times microwires were also used to support the assembly for proper navigation. Confirmation of the location of the balloon and character of the targeted fistula was achieved with the aid of injection of contrast agent through the guiding catheter. Once satisfactory position of the balloon was achieved, it was inflated to see status of the fistula. Sometimes balloon was repositioned inside the fistulous sac for proper closure of the fistulous site. Subsequently, the balloon was detached. At times, more than one balloon was used to close the fistula adequately. In few cases, where balloon could not be negotiated in the sac or the fistula size was very small, transarterial coil occlusion of the fistula was also performed. Electrolytically or mechanically detachable coils were also used to close the fistulae. Coil position was properly checked prior to detachment for any prolapse in to the ICA.

In cases of indirect fistula, the sac was accessed via the venous route. A femoral puncture was followed by placement of the guiding catheter into the main venous trunk – IJV for IPS approach or the facial vein for SOV approach. The microcatheter was then negotiated into the cavernous sinus and coils were placed to occlude the fistula. The coils were released after confirming their position in the sac.

A complete check angiogram was performed after all embolization procedures.

Post procedural management:-

The patient was shifted to the ward or the neurological ICU for 24 hours after the procedure as required.

External compression

In patients having CS DAVF without cortical venous drainage, ipsilateral carotid artery compression can result in cure in some patients. The patient is instructed to compress his or her carotid artery with the contralateral hand for a period of 10- 30 sec, several times per hour, in the sitting position. Patients are warned to discontinue treatment immediately if any symptoms of cerebral ischemia, such as weakness, sensory changes, blindness, or altered mental status, developed. The contralateral hand is used so that if weakness develops unknown to the patient, the compressing hand will fall and treatment will automatically be stopped. Maximal effect is achieved within 4-6 weeks. Patients with cortical venous drainage are excluded from compression therapy because combined carotid artery and jugular compression may increase cerebral venous pressure and could theoretically lead to venous infarction or rupture.

ANALYSIS OF CASES

A total of 129 patients with CCF were analysed . Of these, Type A fistula was noted in seventy patients (54.26%), type B in nine patients (6.97%), type C in eight patients(6.2%) and type D in forty two patients (32.55%) .

TYPE A

Type A CCF was noted in 70 patients out of which 50 (71.4%) were males and 20 (28.5 %) were females .The age range was from 6 years to 65 years (mean age 32.7 yrs). Duration of symptoms was from immediate to four years (mean 3.3months).

TYPE B

Type B CCF was noted in 9 patients out of which 5 (55.5%) were males and 4 (44.4%) were females .The age range was from 30 years to 67 years (mean age 49 years). Duration of symptoms was from 10 days to 17 months (mean - 4months 7 days).

TYPE C

Type C CCF was noted in 8 patients out of which 2 (25%) were males and 6 (75 %) were females .The age range was from 24 years to 72 years (mean age 48.7 yrs). Duration of symptoms was from 15 days to 6 years (mean 19 months).

TYPE D

Type D CCF was noted in 42 patients out of which 20 (47.6%) were males and 22 (52.3 %) were females .The age range was from 13 years to 72 years (mean age 48.8 years). Duration of symptoms was from 10 days to three years (mean 3 months).

PROFORMA

DEPARTMENT OF IMAGING SCIENCES AND INTERVENTIONAL RADIOLOGY

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

PROFORMA FOR EVALUATION & MANAGEMENT OF CAROTICO- CAVERNOUS FISTULA

I) PATIENT DETAILS

Name:

Age/Sex: Hospital No:

DOA: DOD: D/O PROCEDURE:

Occupation:

Address:

Phone:

E-Mail:

II) PRESENTING COMPLAINTS:

a) Diplopia:

b) Eye redness:

c) Proptosis:

d) Headache:

e) Bruit:

f) Diminished vision:

g) Facial numbness:

h) Ocular pain:

i) LOC / Altered sensorium:

j) Seizures:

B. H/o Trauma:

Date:

Mode of trauma:

Penetrating injury: Blunt injury:

Period of onset of symptoms after trauma:

C. Personal history:

HT / DM / MI / CAD / Stroke:

Br Asthma / TB:

History of Allergies:

Smoking / Alcohol:

H/o prior hospitalization:

D. Family History:

Similar medical illness in family members:

E. Prior treatment history:

III) <u>EXAMINATION:</u>

A. GENERAL EXAMINATION:

Cutaneous stigmata:

Features suggestive of connective tissue disorder:

B. NEUROLOGICAL EXAMINATION:

Higher mental functions:

Cranial nerve deficits:

Motor deficits:

Sensory deficits:

Meningeal signs:

Cerebellar signs:

C. LOCAL EXAMINATION :

Side of involvement:

Unilateral: Bilateral:

Signs:

Dilated episcleral vessels:

Diminished vision:

Elevated intra ocular pressure:

Chemosis:

Papilledema:

Third nerve palsy:

Sixth nerve palsy:

Other cranial nerve palsies:

Bruit:

D. OPTHALMOLOGICAL EXAMINATION:

Proptosis: U/L: B/L:

Visual acuity:

Visual field charting:

Pupils:

Intra ocular tension:

Fundus:

E. OTHER SYSTEM EXAMINATION :

Respiratory system:

Cardiovascular system:

Per abdomen:

IV) INVESTIGATIONS:

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

V) IMAGING:

A. CT:

Date:

No. of days after trauma:

Site of fracture:

Dilated cavernous sinus:

Dilated SOV /IOV:

Proptosis:

Other findings:

Follow up CT:

Findings:

B. CT Angiography:

Date /Findings:

C. MR BRAIN:

Date /Findings:

D. OPTHALMIC DOPPLER:

Date/Findings:

E. USG ABDOMEN:

Date/Findings:

F. ANGIOGRAM:

a) Date: Day from ictus:

b) Access site:

CFA Right /Left / Bilateral Other Sites

Venous access CFV / SOV / IJV /ANGULAR VEIN

c) Side of fistula:

d) Site of fistula:

e) Size of defect :

f) Demonstration of fistula via ACom / PCom

g) High /medium / low flow

h) Cross circulation: Satisfactory / Unsatisfactory

i) Patency of ACom / PCom :

j) Associated false aneurysms :

k) Feeders:

ECA:

ICA:

l) Venous drainage:

Anterior Posterior Superior Inferior Contra lateral

m) Other vascular anomalies / variations:

VI) CLASSIFICATION:

BARROW'S CLASSIFICATION

TYPE A - INTRACAVERNOUS ICA TO CAVERNOUS SINUS

TYPE B - DURAL ICA BRANCHES TO CAVERNOUS SINUS

TYPE C - DURAL ECA BRNCHES TO CAVERNOUS SINUS

TYPE D - DURAL ECA AND ICA BRANCHES TO CAVERNOUS SINUS

VII) MANAGEMENT

A) ANAESTHESIA:

B) PROCEDURE DONE:

CCF occlusion:

Time Taken:

a) Route for closure:

Venous:

IPS / SOV / ANGULAR VEIN

Arterial:

b) Material used for closure :

c) Types of coils:

d) Balloon used:

e) Amount of contrast used to inflate the balloon:

f) Guiding catheter:

g) Guidewire:

h) Microcatheter / Microwire:

i) Others:

VIII) <u>CHECK ANGIOGRAM</u>:

CCF completely secured:

Other findings:

IX) <u>INTRA PROCEDURAL COMPLICATION & MANAGEMENT:</u>

X) <u>POST PROCEDURE:</u>

Extubation

Cath lab Elective ventilation

Reason for elective ventilation:

Post procedural neurological status:

Post procedural medications:

Details of ICU stay:

XI) <u>FOLLOW UP VISUAL STATUS:</u>

XII) <u>ADDITIONAL COMMENTS:</u>

XIII) <u>PERFORMING RADIOLOGIST:</u>

ASSISTANT	**CONSULTANT**

RESULTS

RESULTS

We analyzed a total of 129 patients of CCF .Of these we encountered Type A fistulas in 70 patients (54.26%) , type B in nine patients (6.97%) , type C in eight patients (6.2%) and type D in forty two patients (32.55%). The following are the results of each type presented separately.

TYPE A

Type A CCF was noted in 70 patients out of which 50 (71.4%) were males and 20 (28.5 %) were females. The age range was from 6 years to 65 years (mean age 32.7 yrs). Duration of symptom onset was from immediate to our years (mean 3.3months).

Out of these 70 cases, 57(81.4%) were post traumatic, 1(1.4%) was iatrogenic while 12(17.4%) were spontaneous. The onset was acute in 11 (15.7%) and gradual in 59 patients (84.2%).Of the patients who presented with h/o trauma, 23(32.8%) had cranial fractures; two (2.8%) had associated ICH; 32 (57.1%) had associated LOC ,while 2 (3.5%) patients had associated vomiting.

Of these 70 patients, at presentation, swelling of eye was present in 64 (91.4%) & redness in 53 (75.7%), diplopia in 32 (45.7%), DOV in 12(17.1%) & visual loss in five patients (7.1%). Pain in eye was noted in 6 (8.5%), headache in 16 (22.8%), watering from eye in 5 (17.4%), ptosis in 3 (4.2 %), vertigo in one (1.4%) & tinnitus in 16 patients (22.8 %). Other associated features included hempiaresis in 4 patients (5.7 %) LMN facial in 4 patient (5.7 %) while one patient (1.4%) had pus in the anterior chamber.

On examination, proptosis was noted in 66 (94.2 %), redness in 61 (87.1%) & chemosis in 58 patients (82.8%).Lateral rectus palsy was noted in 32 (45.7%) with right 6th nerve palsy in 15 (46.8%) , left 6th nerve palsy in 17(53.1%), bilateral 6th nerve palsy in 1(1.4%) while multiple cranial nerve palsies (3rd, 4th, 6th) were noted in 15 patients(21.4 %).Other cranial nerve palsies were present in 13 patients (18.5%). EOM restriction was noted in 7 (10%) and vision could not be tested in 2 patients (2.8%) due to gross proptosis/pus in anterior chamber . Orbital bruit was present in 42 patients (34.2%) and additional bruit was noted in 14 patients (20%). Dysmorphic facies and features of connective tissue disorder were present in three patients (4.2%).

After angiography in 70 patients, threw were 71 CCFs. The CCF was unilateral in 69 patients (98.5%) and one patient (1.4%) it was bilateral. Amongst unilateral CCF it was on the right side in 38 (54.2 %) and left sided in 31 patients (44.2%) and in one it was bilateral (1.4%). The site of the fistula in the cavernous ICA was at C2 in three patients, C3 in twenty six patients, C4 in ten patients, C5 in two patients, at C2-C3 junction 4 patients, at C3-C4 junction in 15 patients and C4-C5 junction in ten patients .The venous drainage was to the SOV in 34 (48.5%), the IPS in 2 (2.8%),SOv & IPS in 30 (42.8%),IOV in 4 (5.7%), to the contralateral CS in 2(2.8%); while 13 (18.5%) showed cortical venous drainage, deep venous drainage was noted in 4 patients (5.7%).

Of the 70 patients with type A CCF who presented to our institute for endovascular management, 63 patients (90%) underwent embolisation .A combination of balloons, coils and balloons and coils was used. Of 63,eleven patients (15.7%) underwent ICA trapping of which ten were endovascular and had a surgical ligation. One (1.4%) showed spontaneous closure, one (1.4%) showed closure after attempting to pass a balloon and two (2.8%) were advised carotid compression. Three patients with EDS (4.2%) succumbed during catheterization for embolisation.

Of the total 63 CCFs that were embolised 43 patients(68.2%) showed total closure after the first attempt of embolisation,13 (20%)had near total closure while 7(11%) showed closure after multiple attempts . Two patients (3.5%) continue to have minimal residual fistula and are awaiting re-embolisation. Except for patients who had 1* optic atrophy at presentation, all other patients showed significant improvement - from total resolution of nerve palsies to minimal residual palsy. Of the 70 patients, four died .Of these; three had EDS while one succumbed to post procedural complications. There was permanent procedure related morbidity in 2 patients.

TYPE B

Type B CCF was noted in 9 patients out of which 5 (55.5%) were males and 4 (44.4%) were females .The age range was from 30 years to 67 years (mean age 49 years). Duration of symptoms was from 10 days to 17 months (mean - 4months 7 days).

Out of these 9 patients, 1(11.1%) was post traumatic, 8(18.8%) were spontaneous. The onset was acute in 2 patients (22.2%) and gradual in 7 patients (77.7%).

Of the 9 patients, at presentation, swelling of eye was present in all patients (100%), it was right sided in 6(66.6%) and on the left in 3(33.3%). Redness was noted in 9 (100%) patients, diplopia in 6(66.6%), DOV in 2(22.2%). None of the patient had visual loss. Pain in eye was noted in 2 (22.2%), headache in 3(33.3%) & tinnitus in 1 patients (11.1 %). None had the patients presented with fractures, LOC, vomiting or vertigo.

On examination, proptosis was noted in all 9 (100 %), redness in 9 (100%), chemosis in 8 patients (88.8%).Diplopia was noted in 6 (66.6%) with right 6th nerve palsy in 5 (55.5%) , left 6th nerve palsy in 1 (11.1%) while no patient had a bilateral 6th nerve palsy. Multiple cranial nerve palsies (3rd, 4th, 6th) were noted in 1 patient (11.1 %). Orbital bruit was present in 2 patients (22.2 %).

After angiography in these 9 patients we encountered 11 CCFs. The CCF was unilateral in 7 (77.7%), it was on the right in 6 (66.6%) and left in 1 patient (11.1%);

while two patients (22.2%) had a bilateral CCF. The venous drainage was to the SOV in 8 patients (88.8 %), the IPS in 2 (22.2%).

Of the total 9 patients six were advised carotid compression .One patient showed spontaneous occlusion, one underwent transvenous coiling while one patient had no venous access and hence could not be embolised.

At first follow up ,seven patients showed total resolution of the fistula while one showed minimal residual CCF , while one continues to be symptomatic (the patient with no venous access).We encountered no morbidity or mortality in these patients.

TYPE C

Type C CCF was noted in 8 patients out of which 2 (25%) were males and 6 (75 %) were females .The age range was from 24 years to 72 years (mean age 48.7 yrs). Duration of symptoms was from 15 days to 6 years (mean 19 months).

All eight type C CCFs were spontaneous in origin and gradual in onset.

Of the 8 patients, at presentation, swelling of eye was present in 7 patients (87.5%), being on the right in 2(25%) & left in 5 (62.5%) and it was bilateral in one .Redness was noted in all patients (100 %), diplopia in 6(75 %), DOV in 3(37.5 %).Pain in eye was noted in 4 (50 %), headache in 2 (25%), vomiting in 1(12.5 %) & tinnitus in 3 patients (37.5 %) .None of the patients had vertigo at presentation. Other associated features included photophobia in 1 patient (12.5%).

On examination, proptosis was noted in 7 (82.5%), redness in 8 (100%), chemosis in 7 patients (82.5%).Diplopia was noted in 6 (75%) with right 6^{th} nerve palsy in 1 (12.5%) , left 6^{th} nerve palsy in 3(37.5%), bilateral 6^{th} nerve palsy in 1(12.5%), while multiple cranial nerve palsies (3^{rd}, 4^{th}, 6^{th}) were noted in 1 patient(12.5 %). Orbital bruit was present in 5 patients (62.5%).

After angiography in eight patients we encountered 10 CCFs. The CCF was unilateral in 6 patients (75 %) and 2 patient (25%) had a bilateral CCF. Amongst unilateral CCF it was on the right side in 2 (25 %) and left sided in 4 patients (50%). Of the ECA feeders, the IMA was the commonest feeder, followed by MMA and ascending pharyngeal. The venous drainage was to the SOV in 4 patients (50%).

All 8 patients underwent embolisation. After the first embolisation, 2 patients showed total closure, 4 showed near total closure, while 2 showed minimal residual filling of the fistula. At follow up, seven patients (87.5%) showed total resolution of symptoms while one had progressed and underwent re-embolisation .He showed persistent symptoms and was advised transvenous embolisation.

TYPE D

Type D CCF was noted in 42 patients out of which 20 (47.6%) were males and 22 (52.3 %) were females .The age range was from 13 years to 72 years (mean age 48.8 years). Duration of symptoms was from 10 days to three years (mean 3 months).

Out of these 42 cases, 39 (92.8%) were spontaneous while 3 (7.1%) were post traumatic. The onset was gradual in 36 patients (85.7 %) & acute in 6 patients (14.2%).

Of the 42 patients, at presentation, swelling of eye was present in 33 (78.5%), it was on the right in 11(33.3%), on the left in 15(45.4%) and was bilateral in 7 patients (21.2%).Redness was noted in 35 (83.3%), diplopia in 29(69%), DOV in 11(26.1%) & one patient was blind in the left eye. Pain in eye was noted in 12 (28.5 %), headache in 19 (45.2%), vomiting in 4 (9.5%) & tinnitus in 8 patients (19%). None of the patients had vertigo at presentation. Other associated features included watering in 4 (9.5%), ptosis in 3 (7.4 %), left upper limb weakness in 1 patient (2.3 %). Of the patients who presented with h/o trauma no patient had cranial fractures or LOC. One patient (2.3%) had presented with ICH.

On examination, proptosis was noted in 33(78.5%), redness in 39(92.8%), & chemosis in 30 patients (71.4%). Diplopia was noted in 29 (69%) with right 6^{th} nerve palsy in 8 (19 %) , left 6^{th} nerve palsy in 13 (30%), bilateral 6^{th} nerve palsy in 4(9.5%) while multiple cranial nerve palsies (3^{rd}, 4^{th}, 6^{th}) were noted in 4 patients(9.5%). Third CN was noted in 5 patients (11.9%),other CN palsies were present in three patients (7.1%) .Orbital bruit was present in 5 patients (11.9%) and additional bruit was noted in 1 patient (2.3 %).

After angiography in 42 patients we encountered 68 CCFs. The CCF was unilateral in 16 (30.8%) & bilateral in 26 patients (61.9%). Amongst unilateral CCF it was on the right side in 8 (19 %) and left sided in 8 patients (19%). The venous drainage was to the SOV in 34 patients (80.9%), the IPS in 13 (30.9 %), SPS & IOV in 1 (2.3%), to the contralateral CS in 3 (7 %); while 1 (2.3%) showed cortical venous drainage, deep venous drainage was noted in 2 (4.7%).

Of the 34 patients who underwent embolisation, 26 (76.47%) underwent embolisation via arterial route, 3 (8.8%) underwent embolisation via the arterial and venous route while 5 (14.7%) underwent embolisation via the venous route only. Seven (16.6%) of 42 patients were advised carotid compression, while one patient died before embolisation.

Of those who underwent embolisation 17 (50%) showed total closure of the fistula after the first attempt,13 (38.2%)showed near total closure while four (11.7%) showed residual (3 of these underwent multiple sessions of embolisation) .

Three patients (7.1%) suffered permanent neurological morbidity .Two other patients (4.7%) suffered procedure related complication but it did not cause any neurological morbidity. One patient (2.3%) ([who had cortical venous reflux on angiogram) had a seizure just prior to being taken up for embolisation and later succumbed to SAH.

	TYPE A	TYPE B	TYPE C	TYPE D
NUMBER OF PATIENTS	70	9	8	42

Table 1.1: Total Number of Patients

Sex	TYPE A	TYPE B	TYPE C	TYPE D
MALE	50	5	2	20
FEMALE	20	4	6	22

Table 1.2: Sex Distribution

Age	TYPE A	TYPE B	TYPE C	TYPE D
0 - 10	4	0	0	0
11 - 20	8	0	0	1
21 - 30	20	1	2	4
31 - 40	18	2	2	7
41 - 50	13	1	0	8
51 - 60	4	4	3	13
61 - 70	3	1	0	6
71 - 80	0	0	1	3

Table 1.3: Age Distribution

Etiology	TYPE A	TYPE B	TYPE C	TYPE D
TRAUMA	57	1	0	3
SPONTANEOUS	12	8	8	39
IATROGENIC	1	0	0	0

Table 1.4: Etiology of CCF

Onset	TYPE A	TYPE B	TYPE C	TYPE D
ACUTE	11	2	0	6
GRADUAL	59	7	8	36

Table 1.5: Type of Onset

Presenting Symptoms	TYPE A	TYPE B	TYPE C	TYPE D
SWELLING OF EYE	64	9	7	33
REDNESS	53	9	9	35
DIPLOPIA	32	6	6	29
DOV	12	2	3	11
VISUAL LOSS	5	0	0	1
PAIN IN EYE	6	2	4	12
HA	16	3	2	19
TRAUMA	57	1	0	3
FRACTURES	23	0	0	0
ICH	2	0	0	1
LOC	32	0	0	0
VOMITING	2	0	1	4
WATERING	5	2	0	4
PTOSIS	3	0	0	3
TINNITUS	16	1	3	8
VERTIGO	1	0	0	0

Table 1.6: Presenting Symptoms

Duration	TYPE A	TYPE B	TYPE C	TYPE D
IMMEDIATE	1	0	0	0
< 1 WEEK	1	0	0	1
1 WK TO 1 MONTH	15	2	2	16
1M TO 1 YR	44	6	2	24
1 YR - 2 YR	6	1	2	0
2 YR - 3 YR	3	0	1	1
3 YR - 4 YR	0	0	0	0
4 YR - 5 YR	0	0	0	0
5 YR - 6 YR	0	0	1	0

Table 1.7: Duration of Symptoms

Signs of Examination	TYPE A	TYPE B	TYPE C	TYPE D
PROTOSIS	66	9	7	33
REDNESS	61	9	8	39
CHEMOSIS	58	8	7	30
LACRIMATION	5	2	0	4
LR PALSY	32	7	6	28
RT	15	5	1	6
LT	17	1	3	14
B/L	1	0	1	3
MULTIPLE CN PLASIES	15	1	1	6
OTHERS	13	1	0	0
NO EOM	7	0	0	0
CNBT	2	0	0	0
BRUIT	42	3	5	5
ADDITIONAL	14	0	0	1
ABNORMAL FACIES	3	0	0	0
PROTOSIS	66	9	7	33
REDNESS	61	9	8	39
CHEMOSIS	58	8	7	30
LACRIMATION	5	2	0	4

Table 1.8: Signs of Examination

Side	Type A	Type B	Type C	Type D
UNILATERAL	69	7	6	16
RIGHT	38	6	2	8
LEFT	31	1	4	8
BILATERAL	1	2	2	26

Table 1.9: Side of CCF on Angiogram

ILLUSTRATIVE CASES

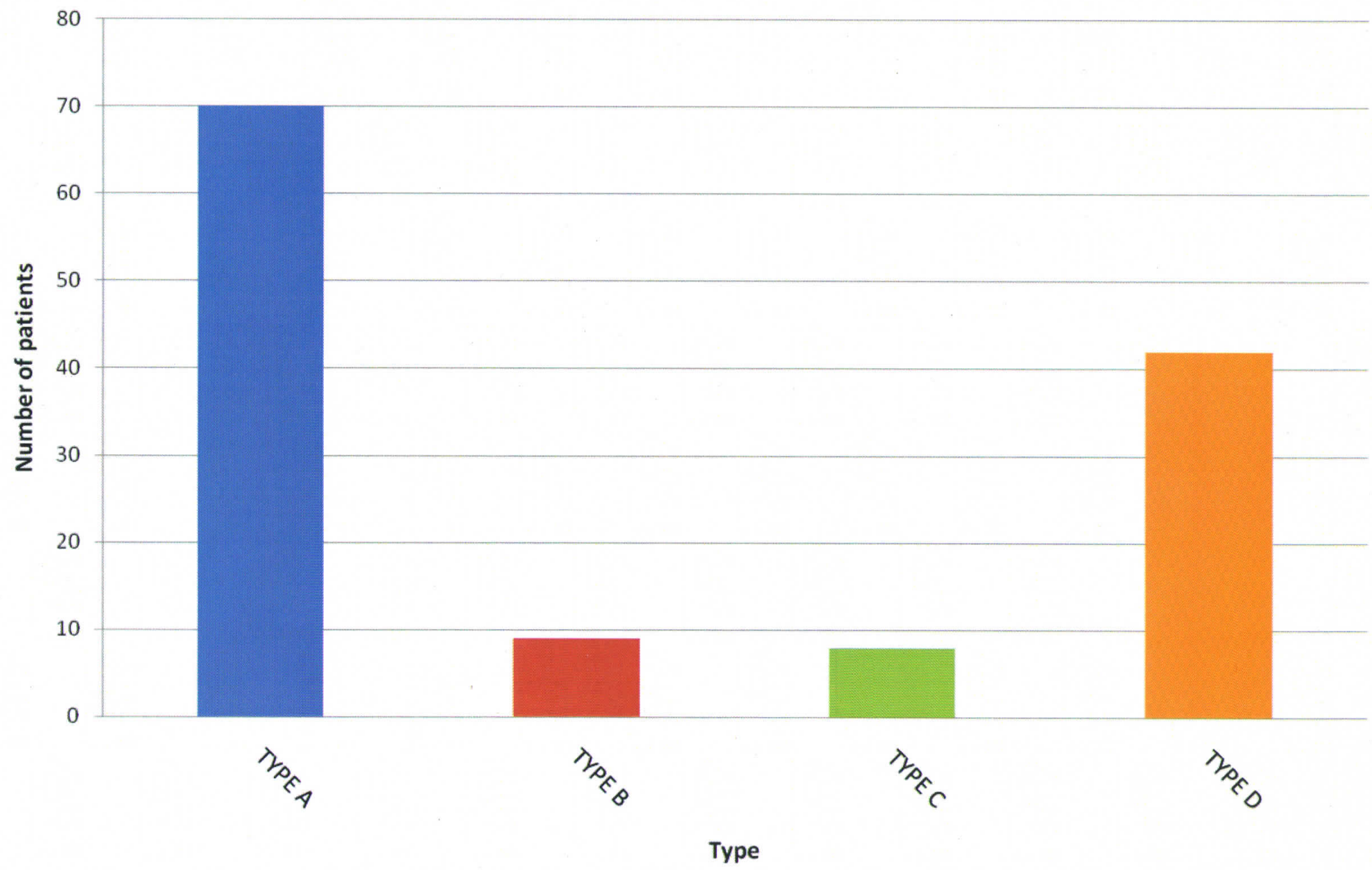


Figure 1 : Total Number of Patients

Figure 2 : Sex Distribution

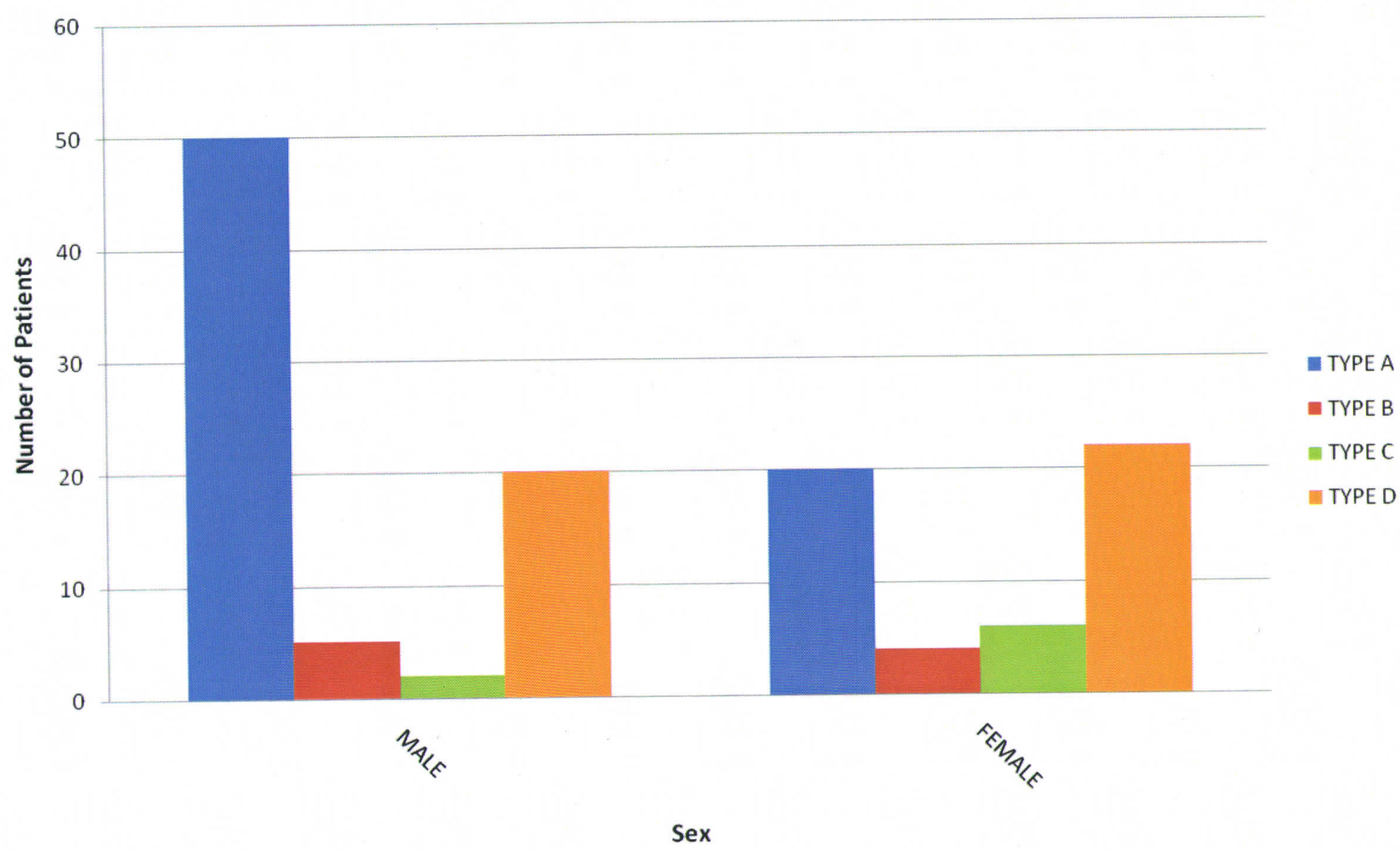


Figure 3 : Age Distribution

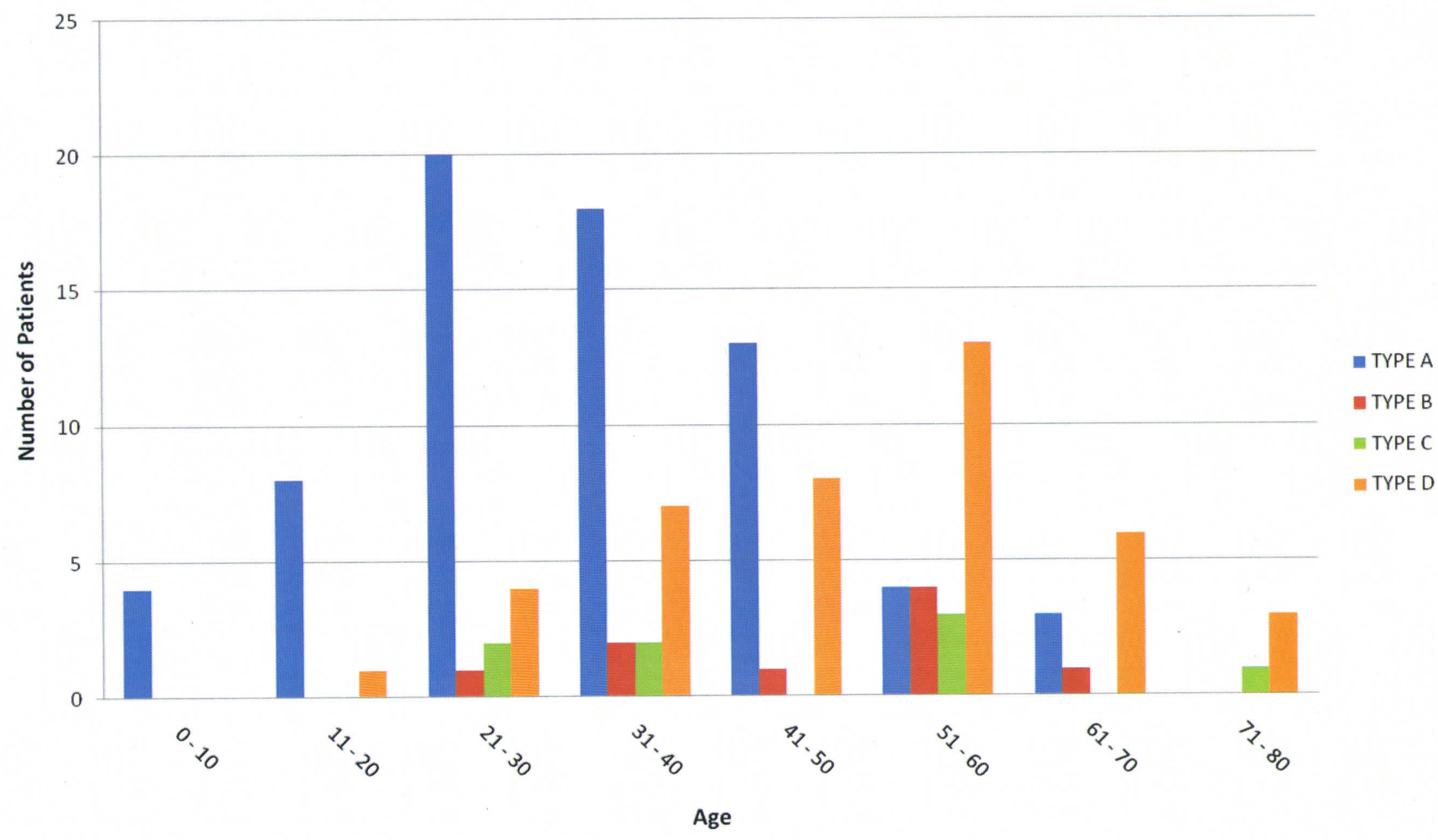


Figure 4 : Etiology of CCF

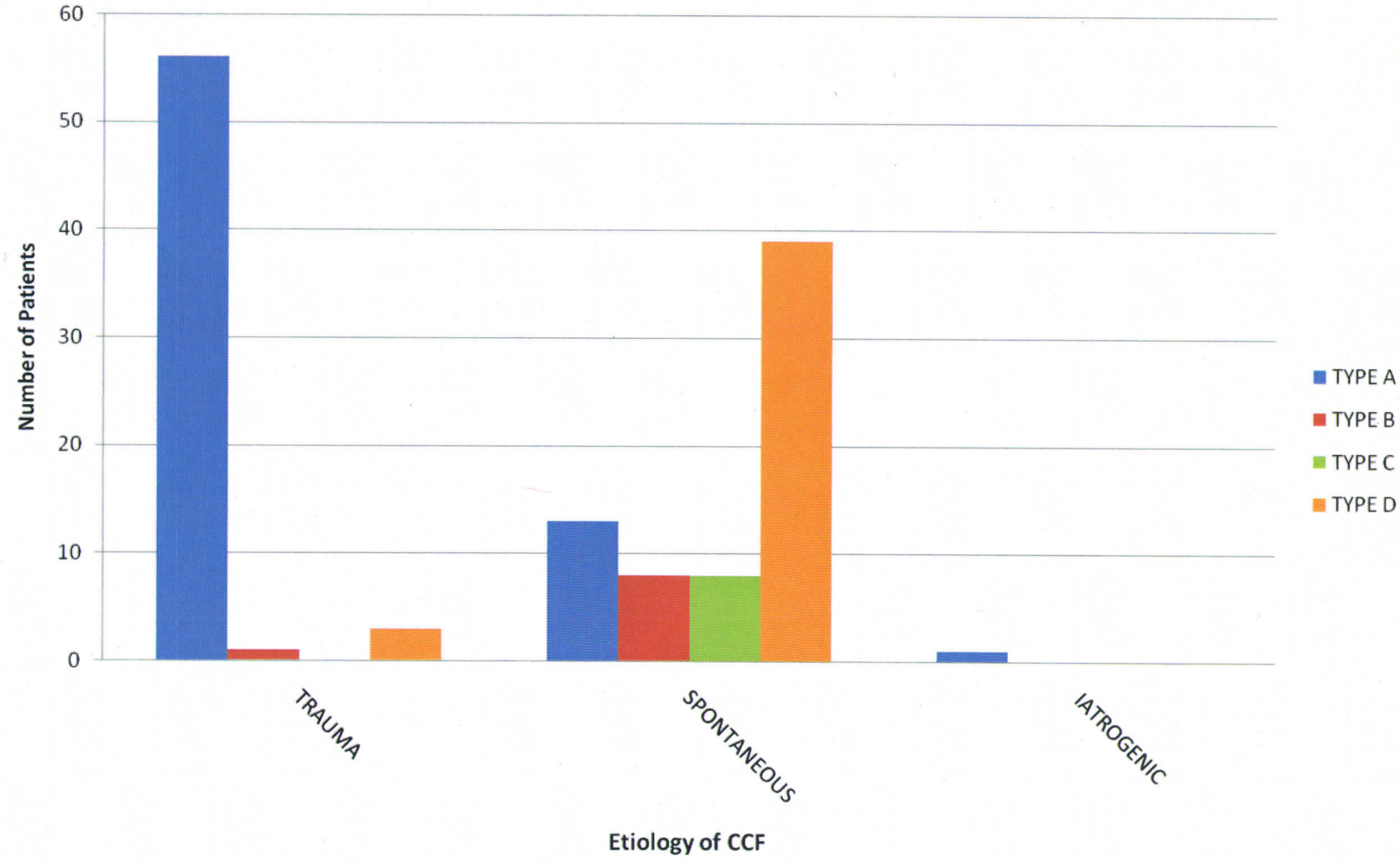
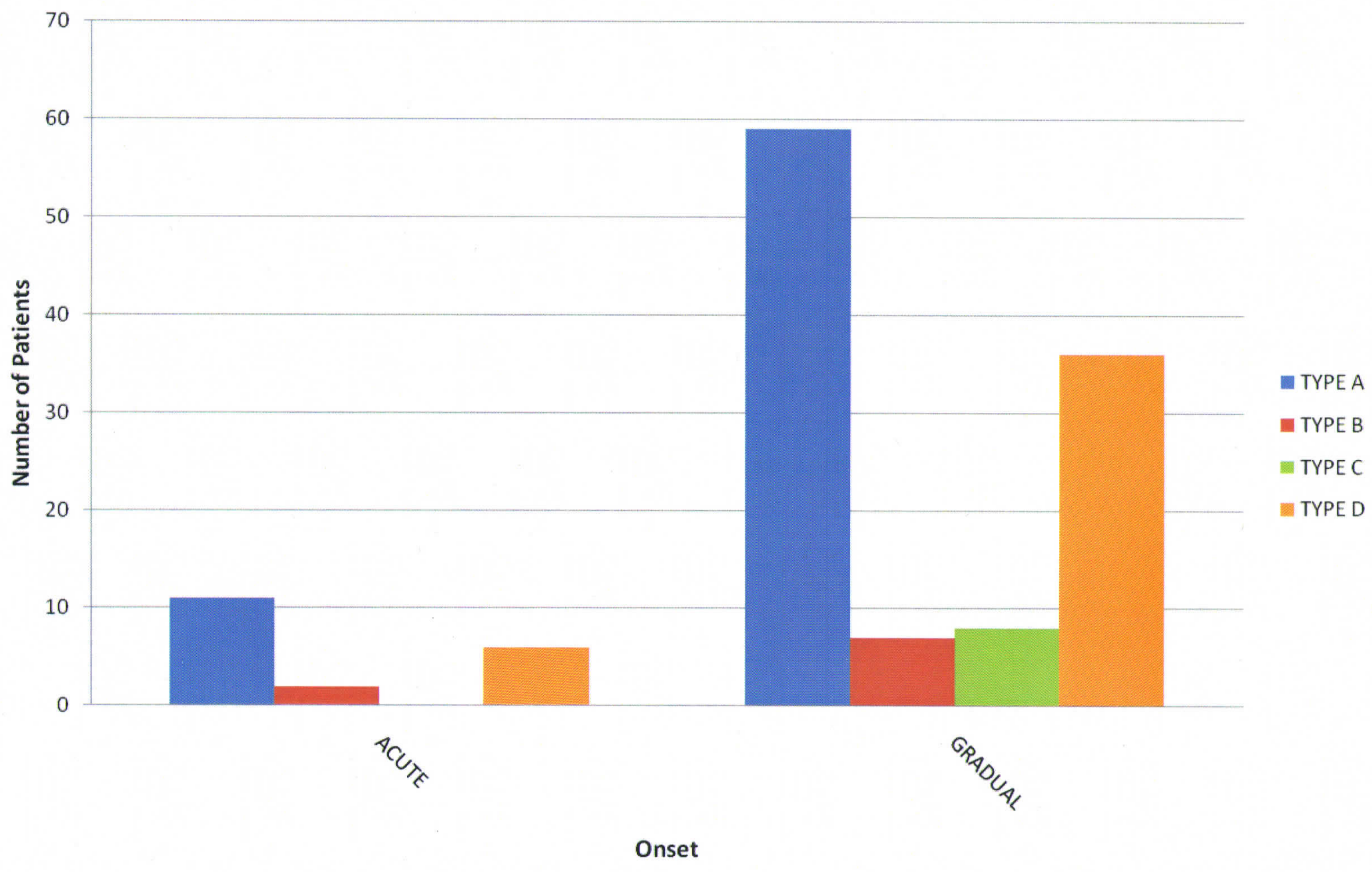


Figure 5 : Type of Onset



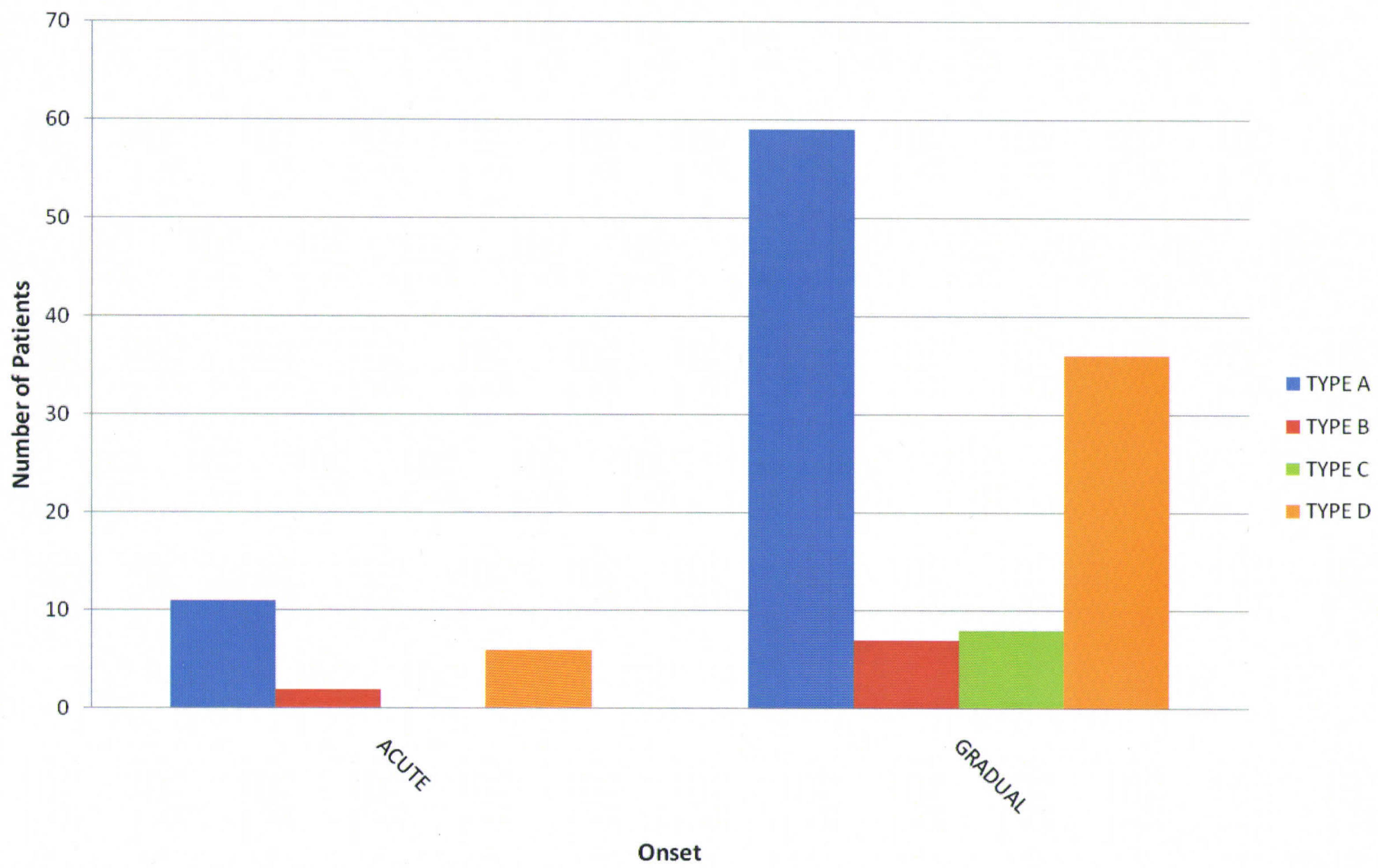


Figure 5 : Type of Onset

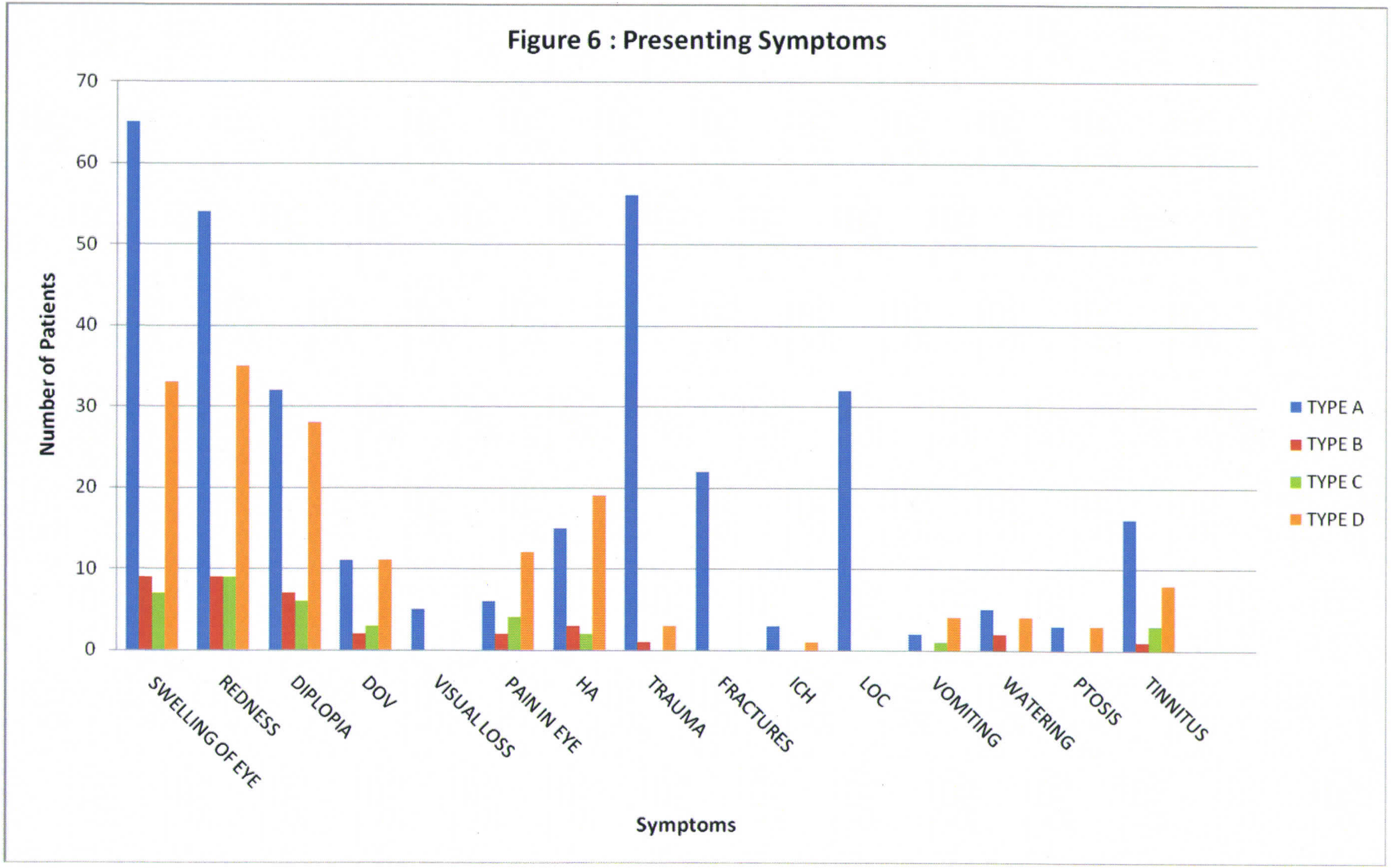


Figure 6 : Presenting Symptoms
Number of Patients
70
60
50
40
30
20
10
0
SWELLING OF EYE
REDNESS
DIPLOPIA
DOV
VISUAL LOSS
PAIN IN EYE
HA
TRAUMA
FRACTURES
ICH
LOC
VOMITING
WATERING
PTOSIS
TINNITUS
Symptoms
TYPE A
TYPE B
TYPE C
TYPE D

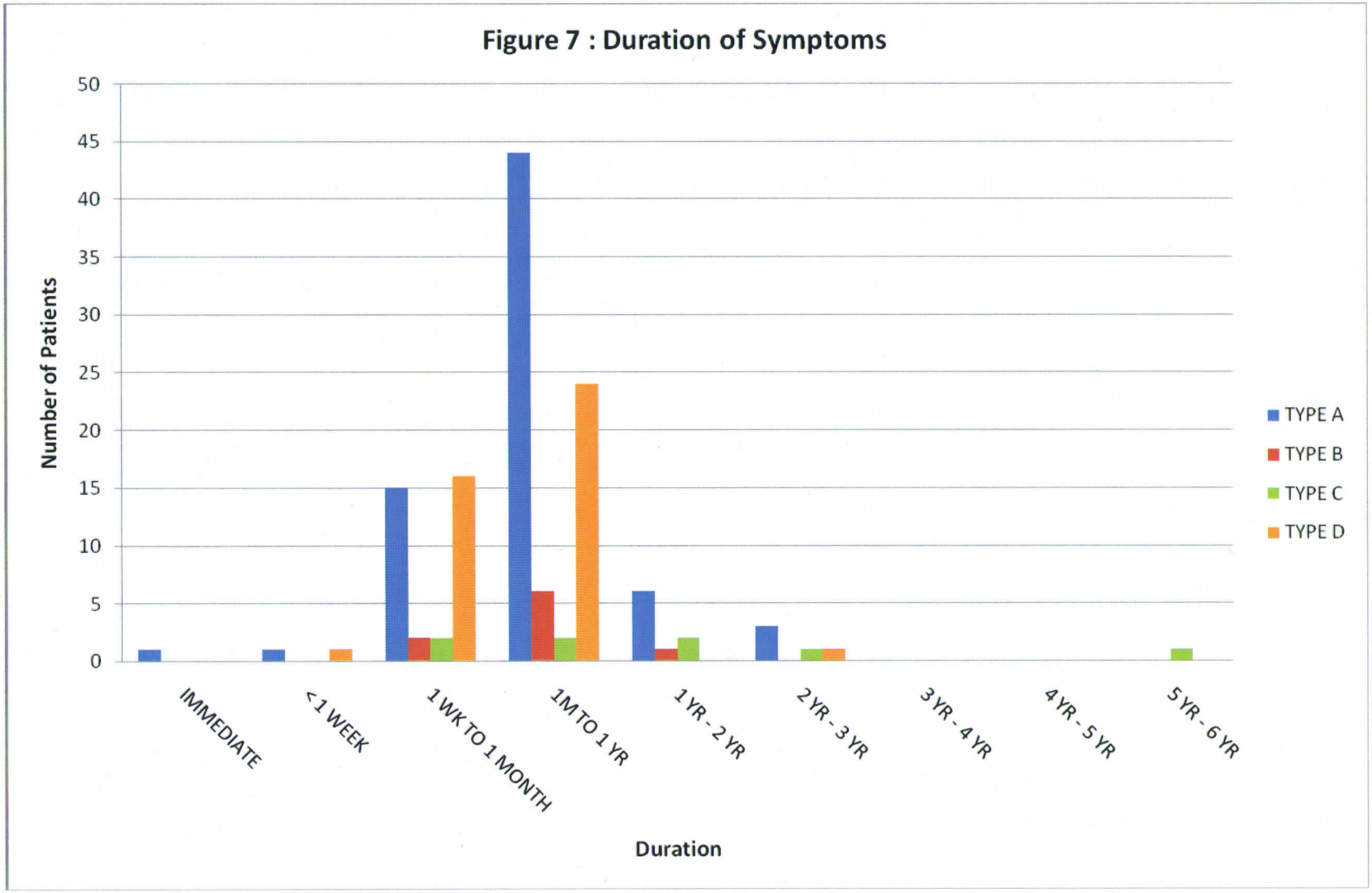


Figure 7 : Duration of Symptoms
Number of Patients
50
45
40
35
30
25
20
15
10
5
0
IMMEDIATE
<1 WEEK
1 WK TO 1 MONTH
1M TO 1 YR
1 YR - 2 YR
2 YR - 3 YR
3 YR - 4 YR
4 YR - 5 YR
5 YR - 6 YR
Duration
TYPE A
TYPE B
TYPE C
TYPE D

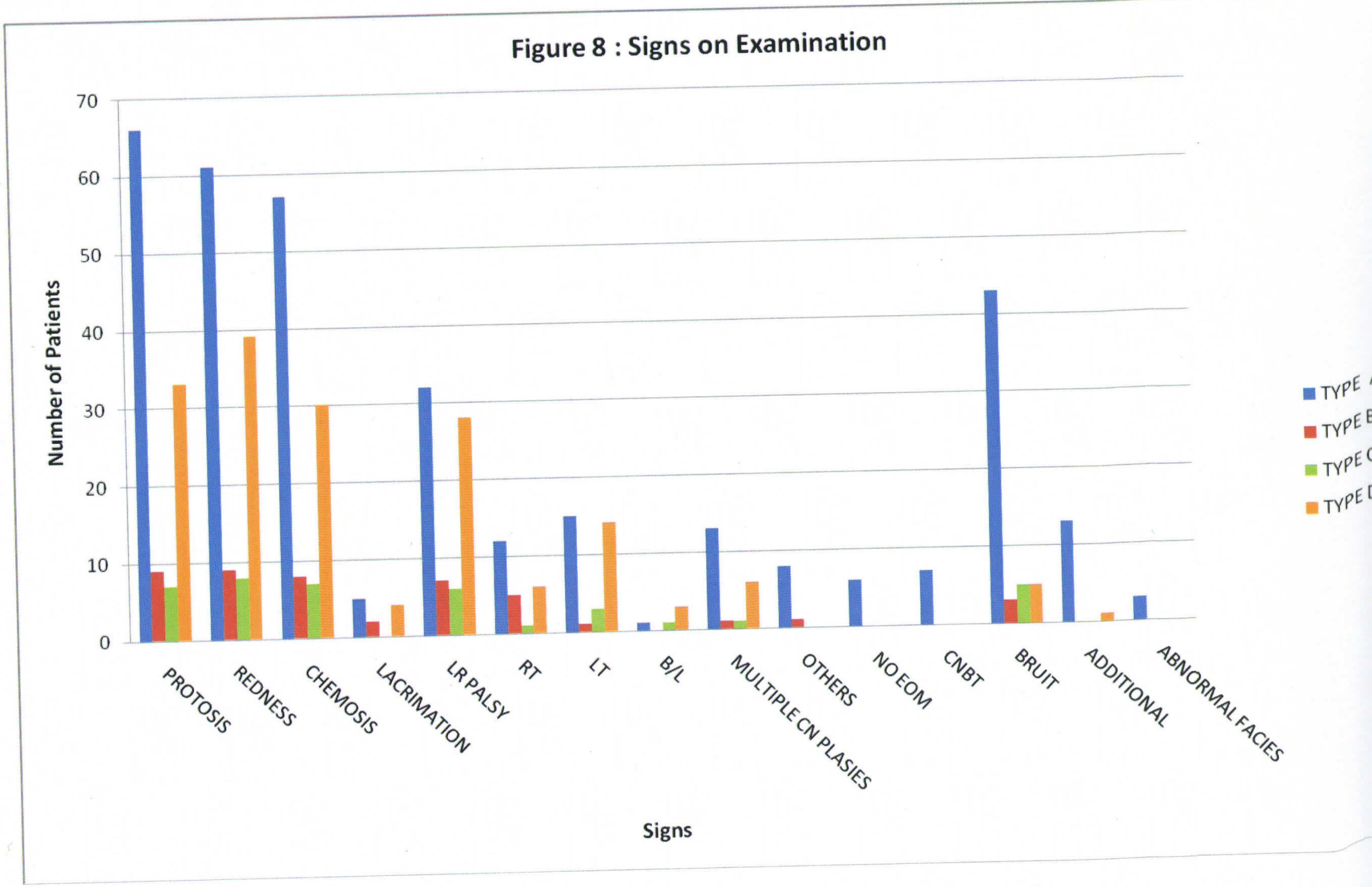
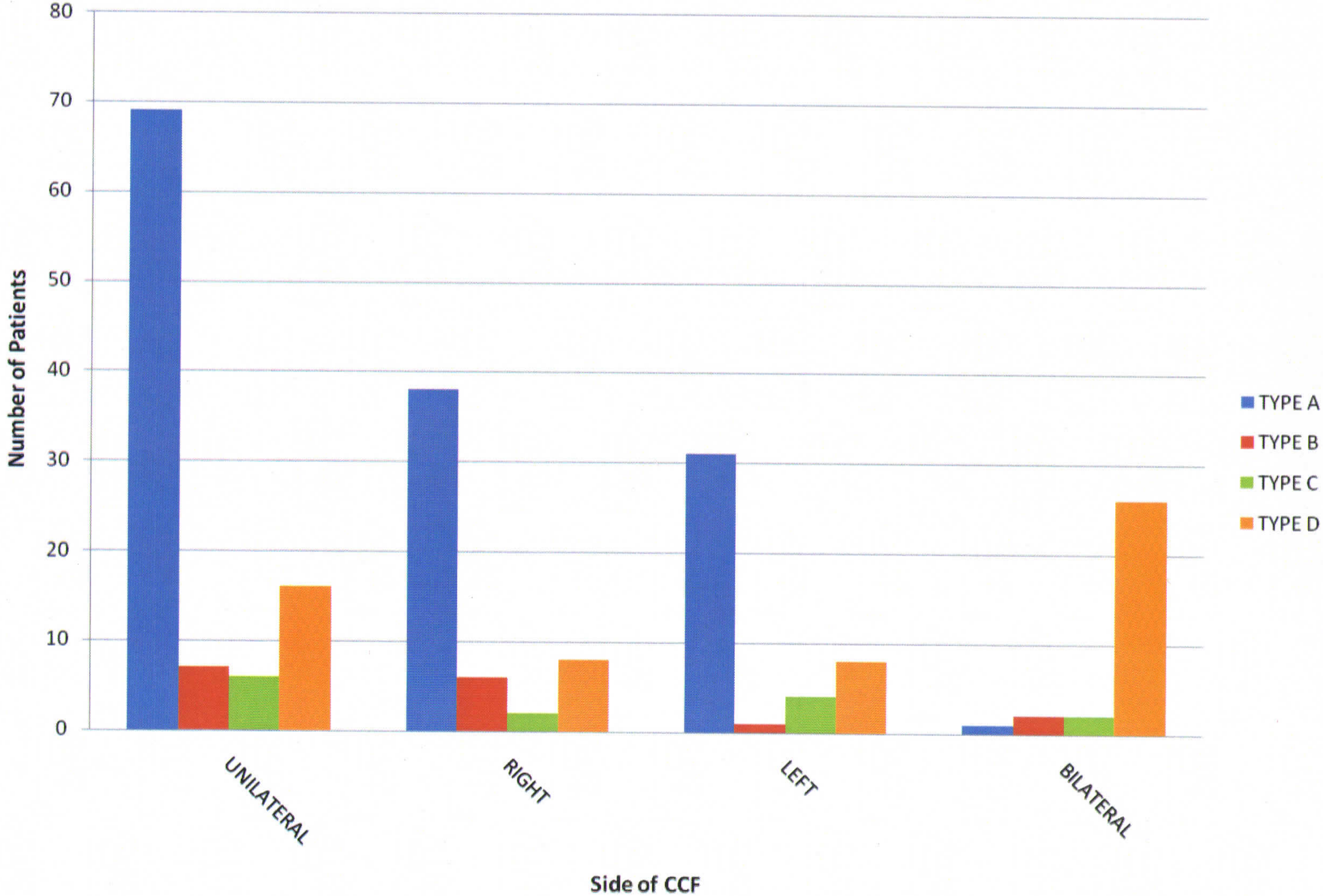
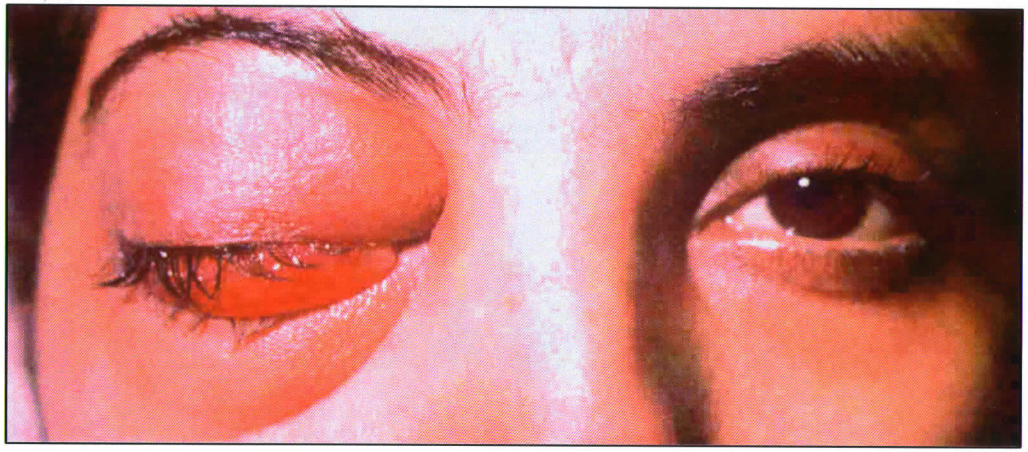


Figure 8 : Signs on Examination
Number of Patients
70
60
50
40
30
20
10
0
PROTOSIS
REDNESS
CHEMOSIS
LACRIMATION
LR PALSY
RT
LT
B/L
MULTIPLE CN PLASIES
OTHERS
NO EOM
CNBT
BRUIT
ADDITIONAL
ABNORMAL FACIES
Signs
TYPE A
TYPE B
TYPE C
TYPE D

Figure 9 : Side of CCF on Angiogram



PRE-EMBOLISATION



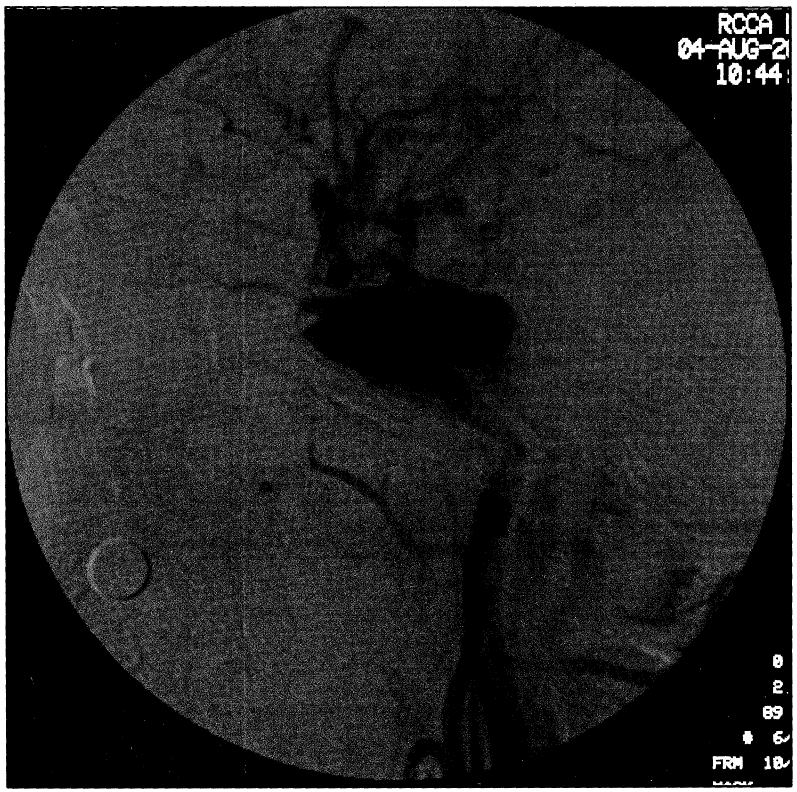
POST-EMBOLISATION

PRE-EMBOLISATION

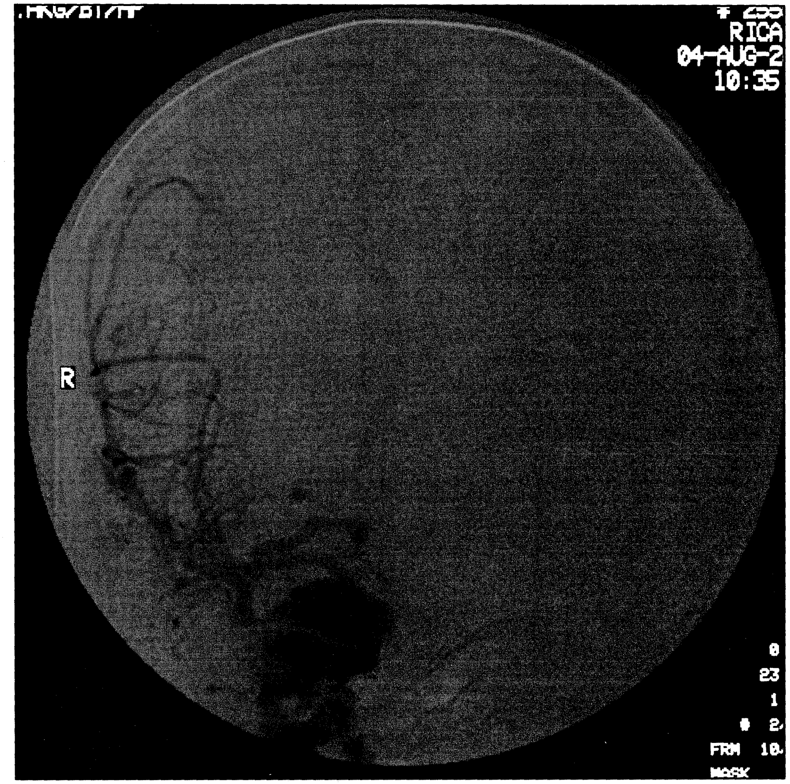
POST-EMBOLISATION

CASE-1, TYPE A CCF

PRE EMBOLISATION

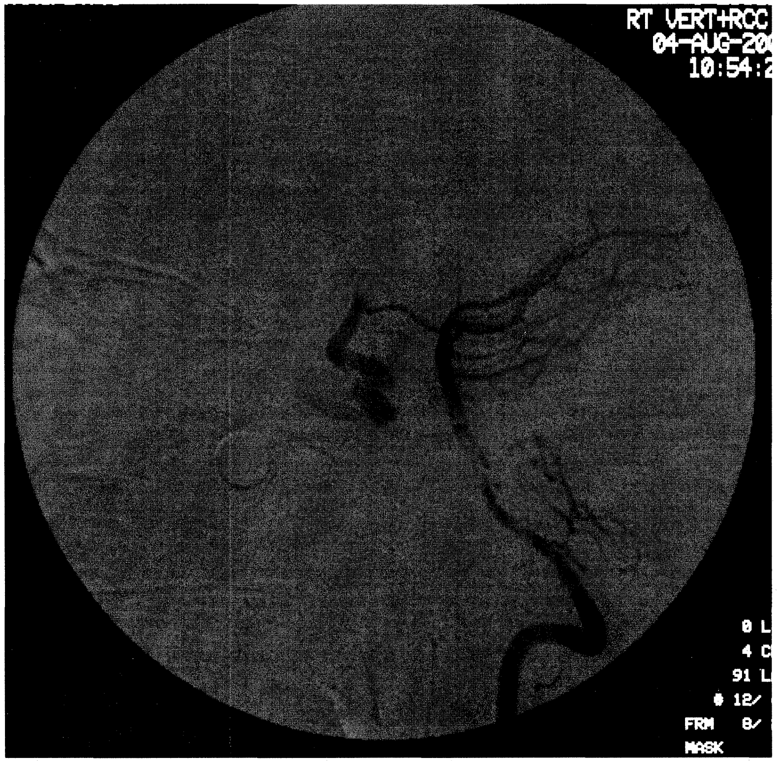


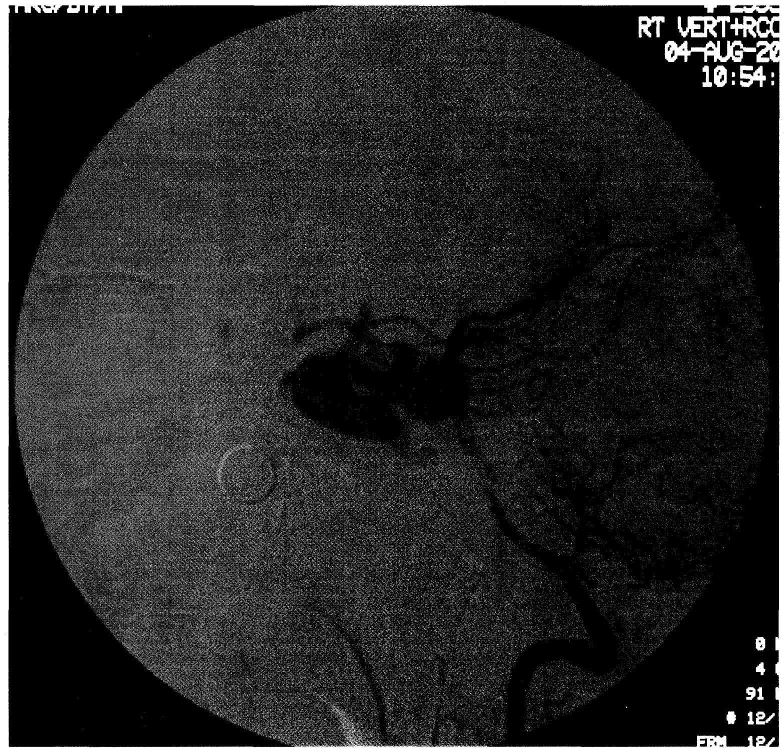
RCCA LATERAL VIEW



RICA AP VIEW

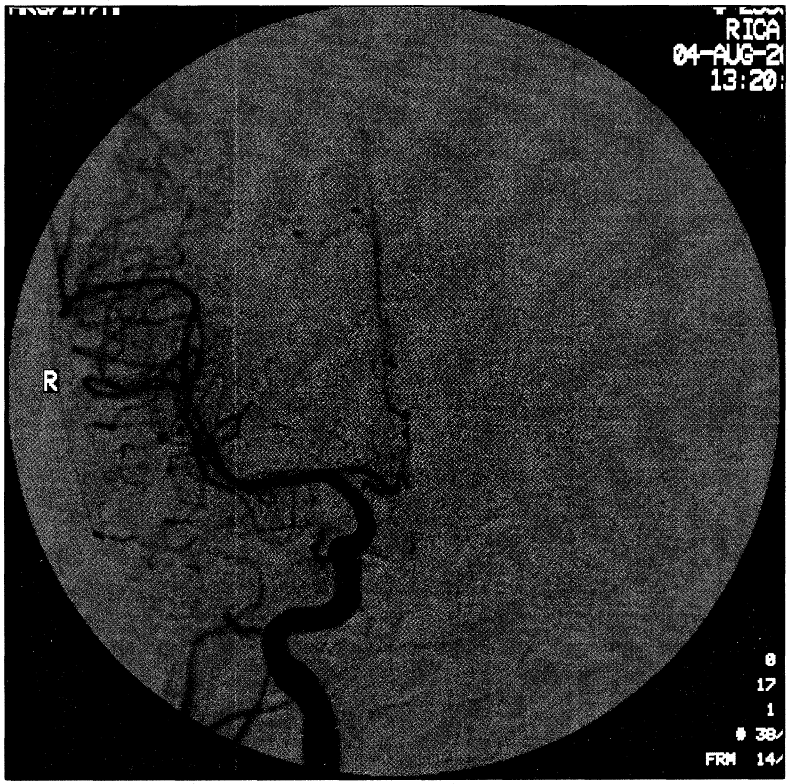
DEMONSTRATION OF FISTULA BY ALCOCK'S MANEUVER



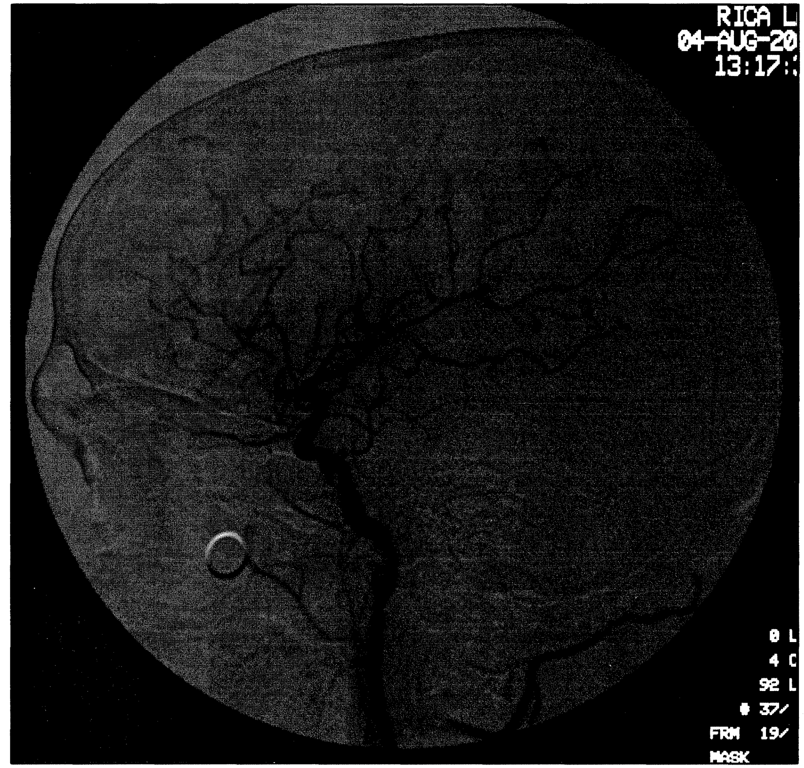


RIGHT VERTEBRAL ARTERY LATERAL VIEW

POST EMBOLISATION 2 BALLOONS



RICA AP VIEW



RICA LATERAL VIEW

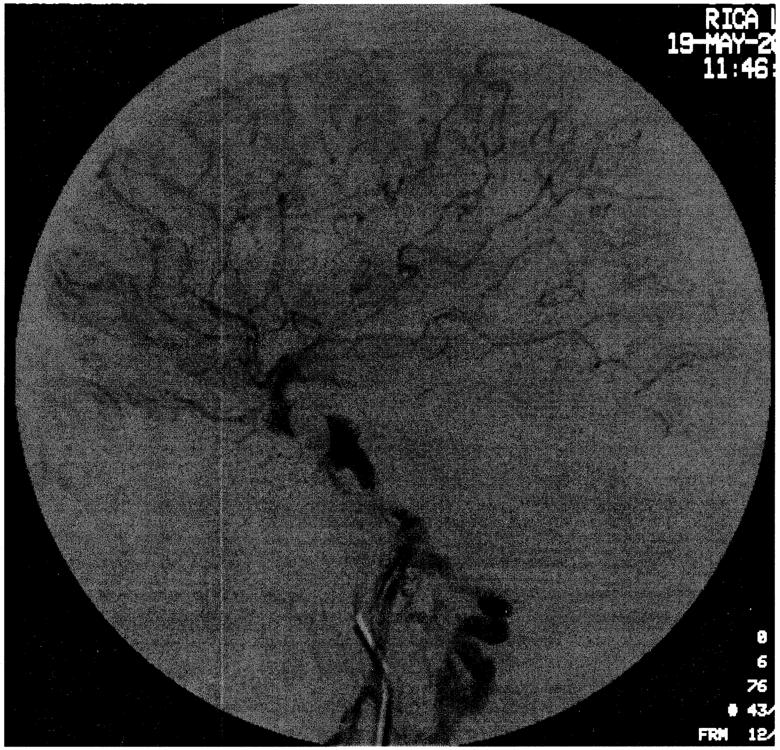
CASE-2, TYPE A ,U/L FISTULA ,B/L CS VENOUS DRAINAGE
B/L PROPTOSIS AT PRESENTATION

PREEMBOLISATION

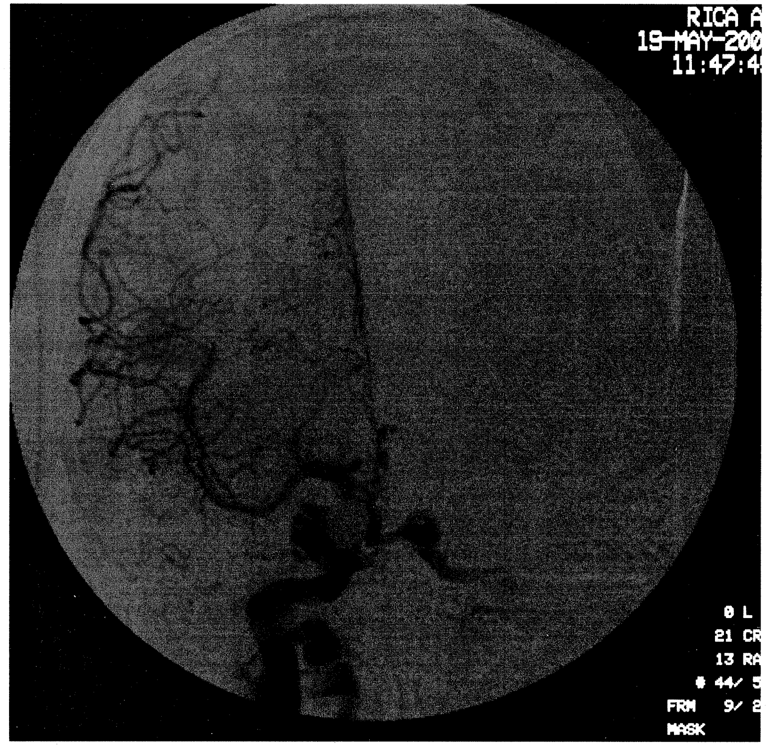
RICA LATERAL VIEW

RICA AP VIEW

POST EMBOLISATION

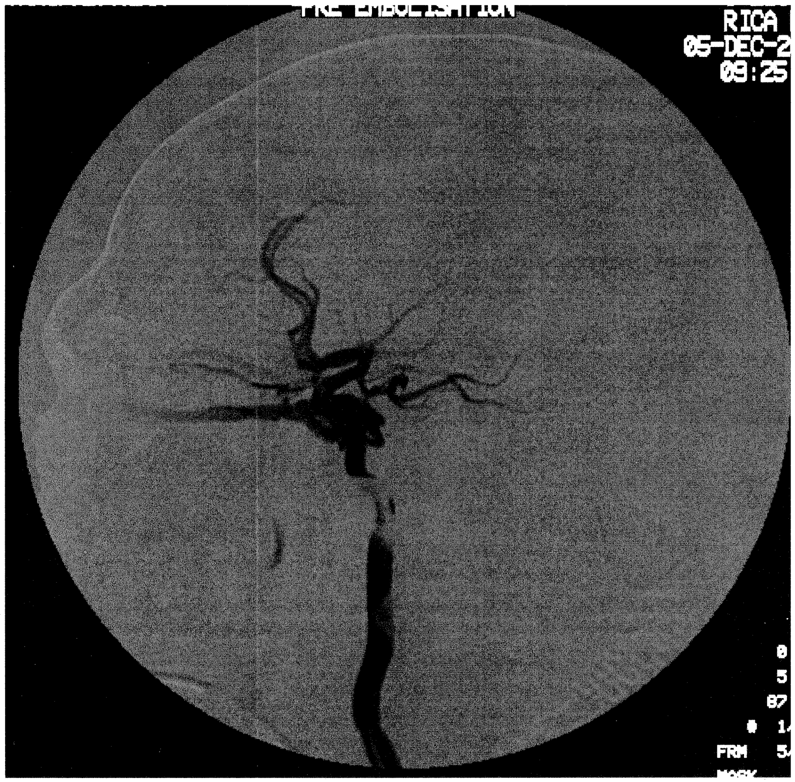


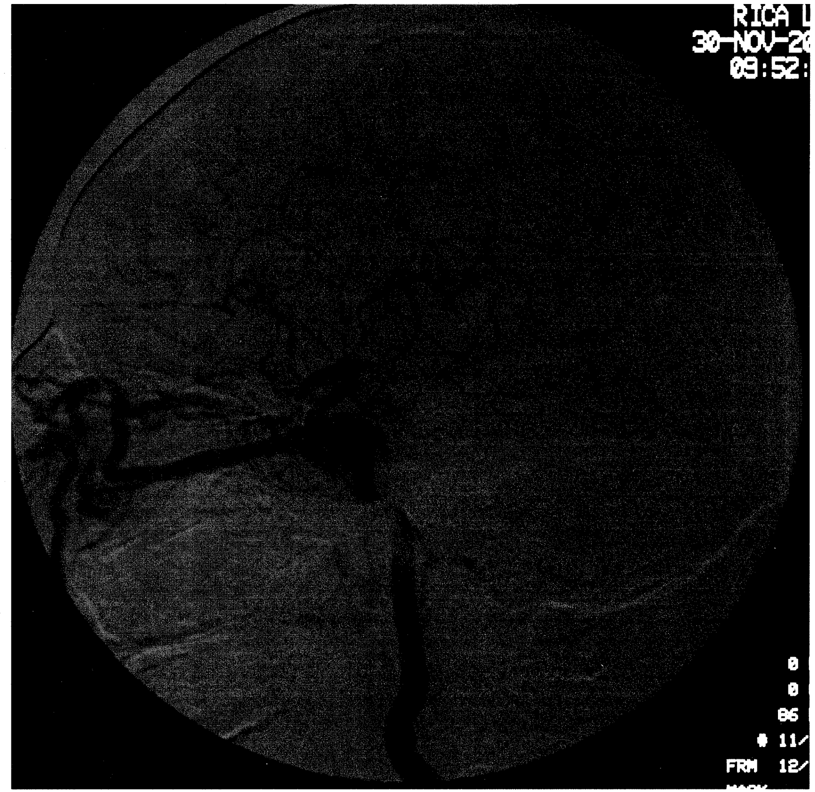
RICA LATERAL VIEW



RICA AP VIEW

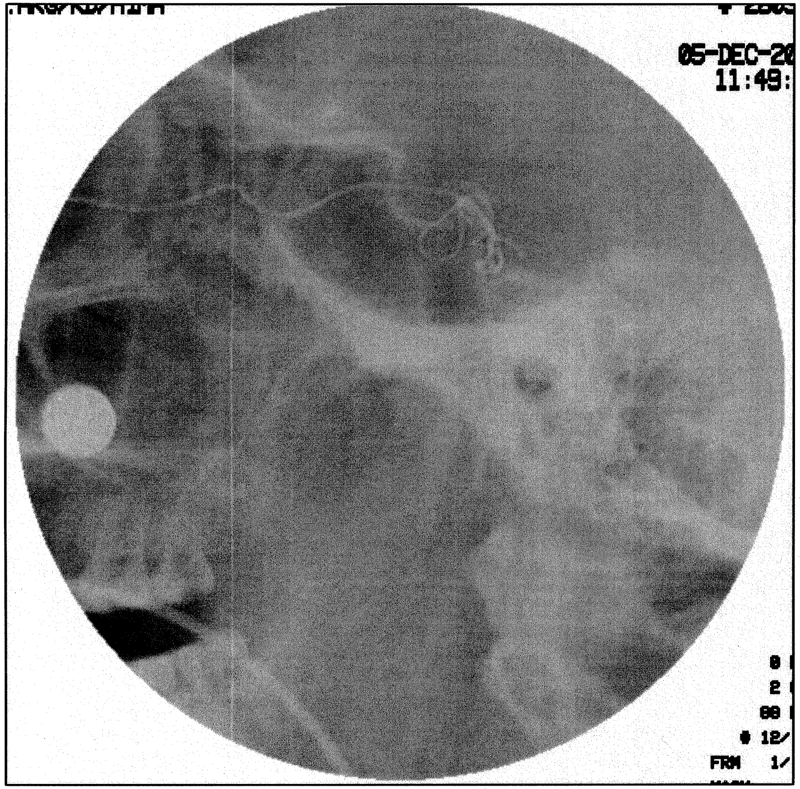
CASE-3, TYPE B CCF
PRE- EMBOLISATION





RICA LATERAL VIEW

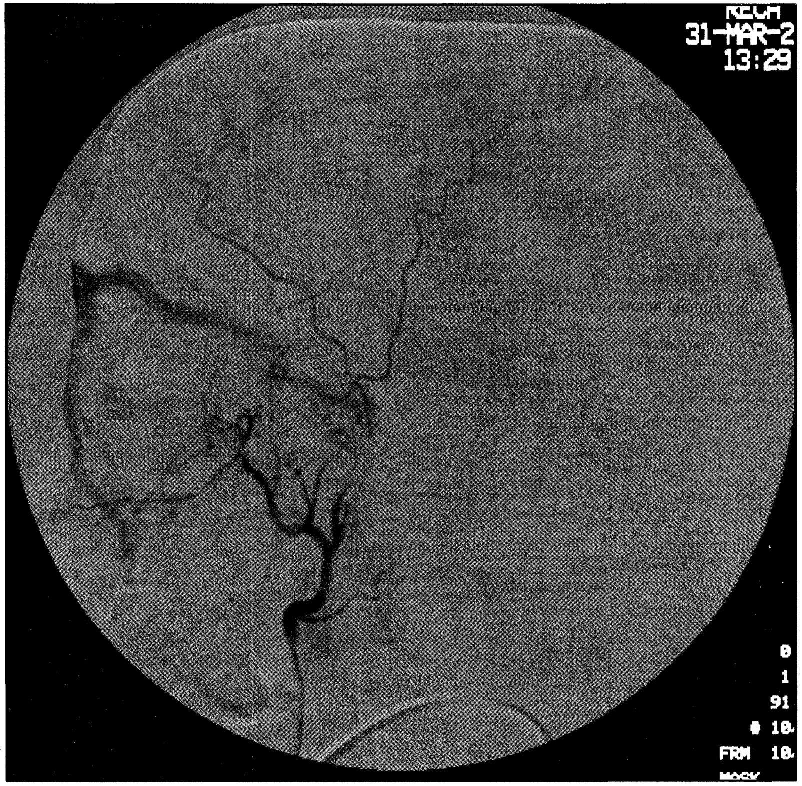
VENOUS ROUTE EMBOLISATION



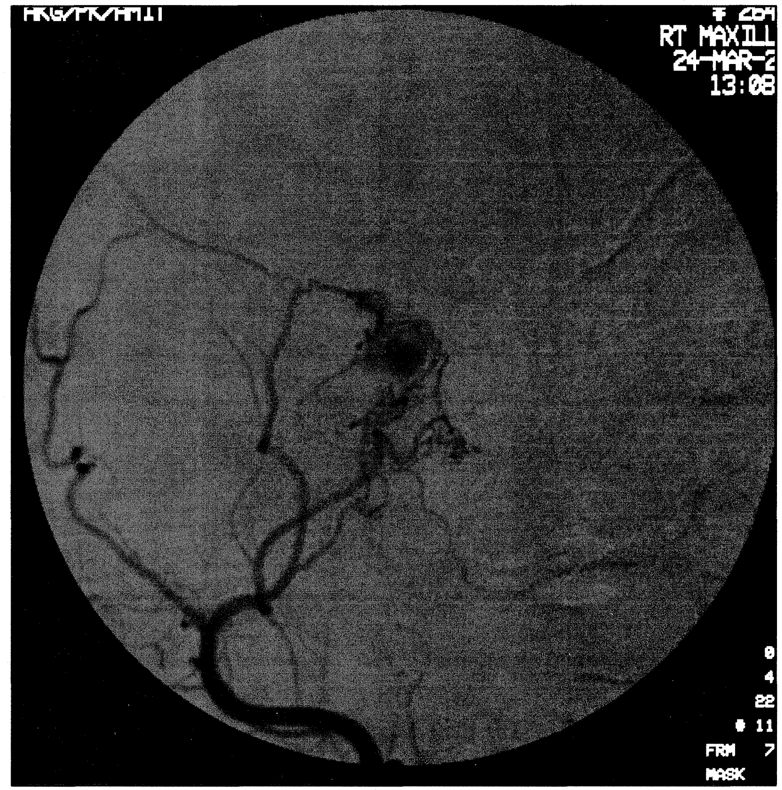
MICROWIRE VIA SOV ROUTE

POST EMBOLISATION
LCCA LATERALVIEW

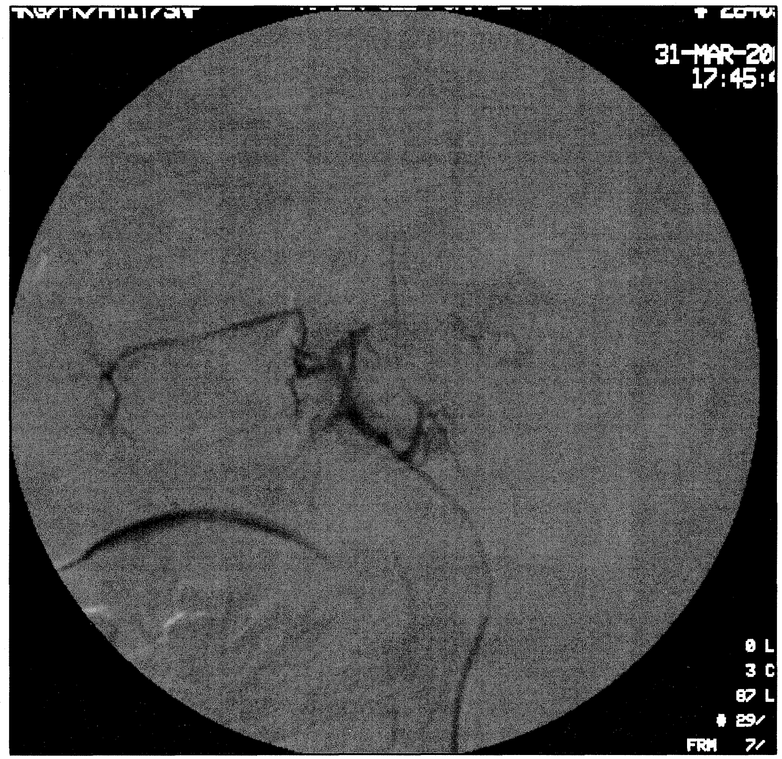
CASE-4,TYPE C CCF
PRE- EMBOLISATION



RIMA LATERAL VIEW



RIMA AP VIEW



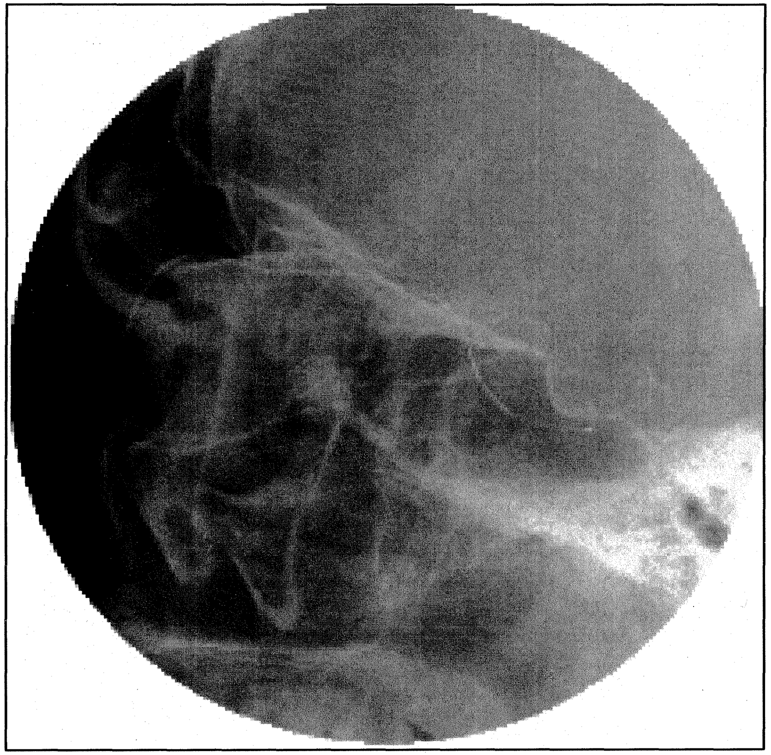
POST EMBOLISATION OF RIMA

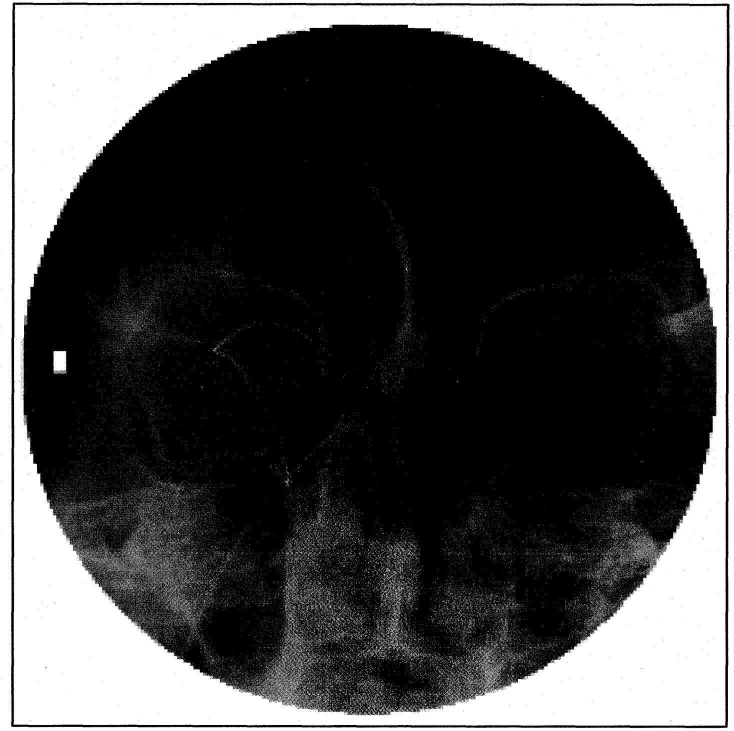
TYPE D CCF
PRE- EMBOLISATION

RICA LATERAL VIEW

RECA LATERAL VIEW

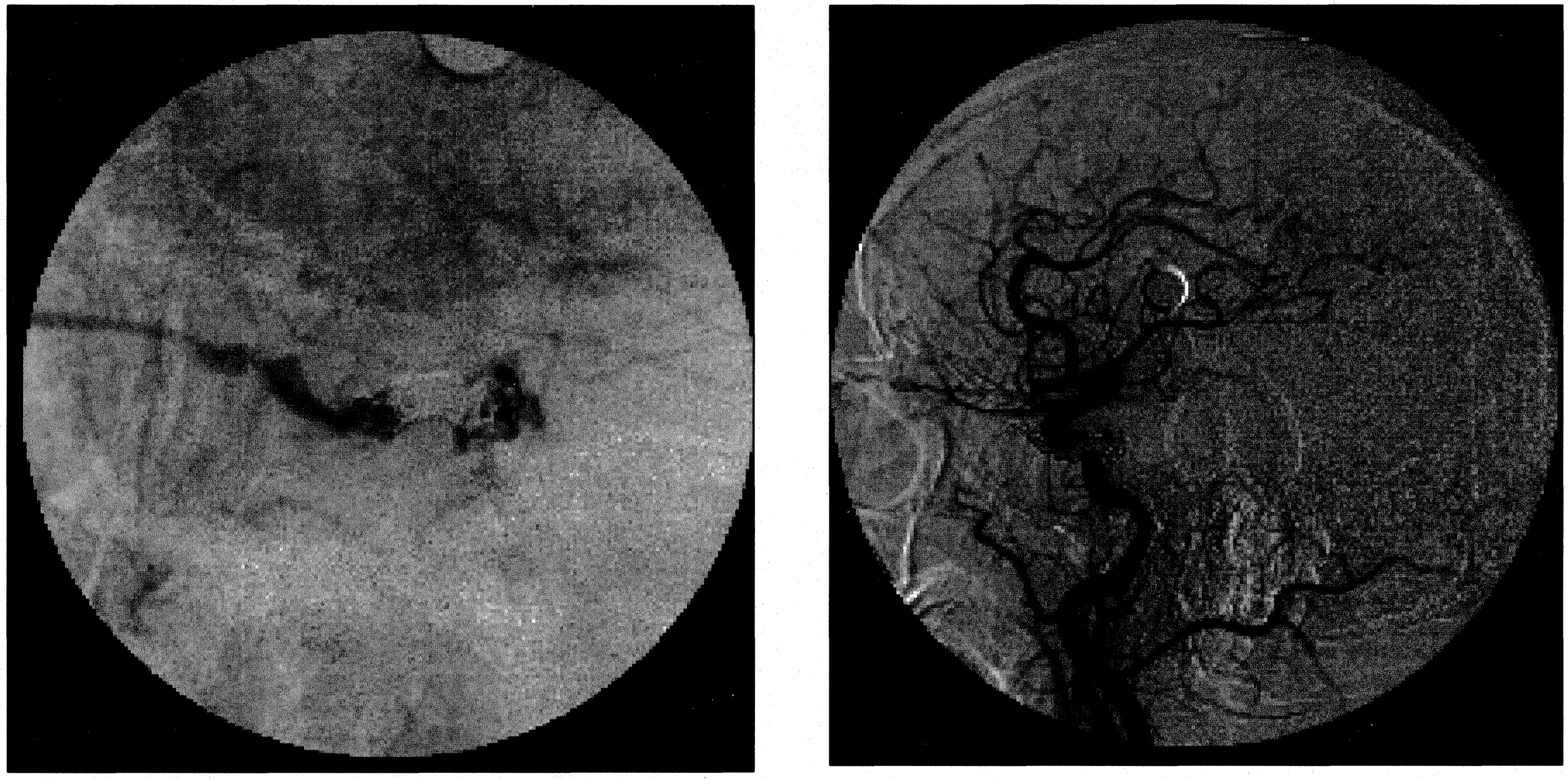
TRANSANGULAR VEIN CUT DOWN ACCESS TO CS





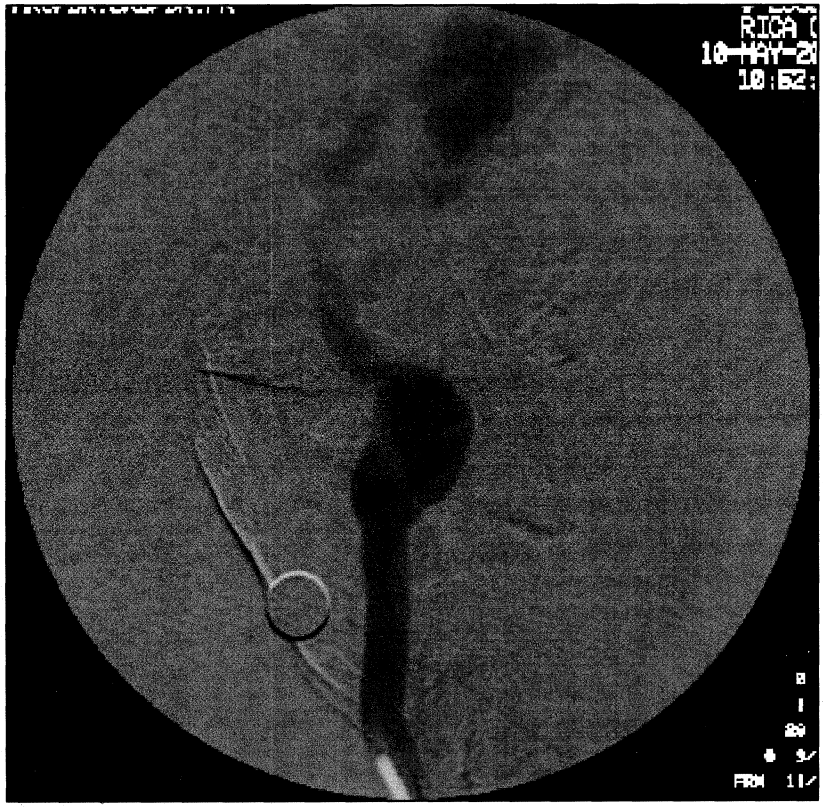
GUIDEWIRE IN SITU

LATERAL VIEW AP VIEW



POST COILING OF CAVERNOUS SINUS
RICA LATERAL VIEW

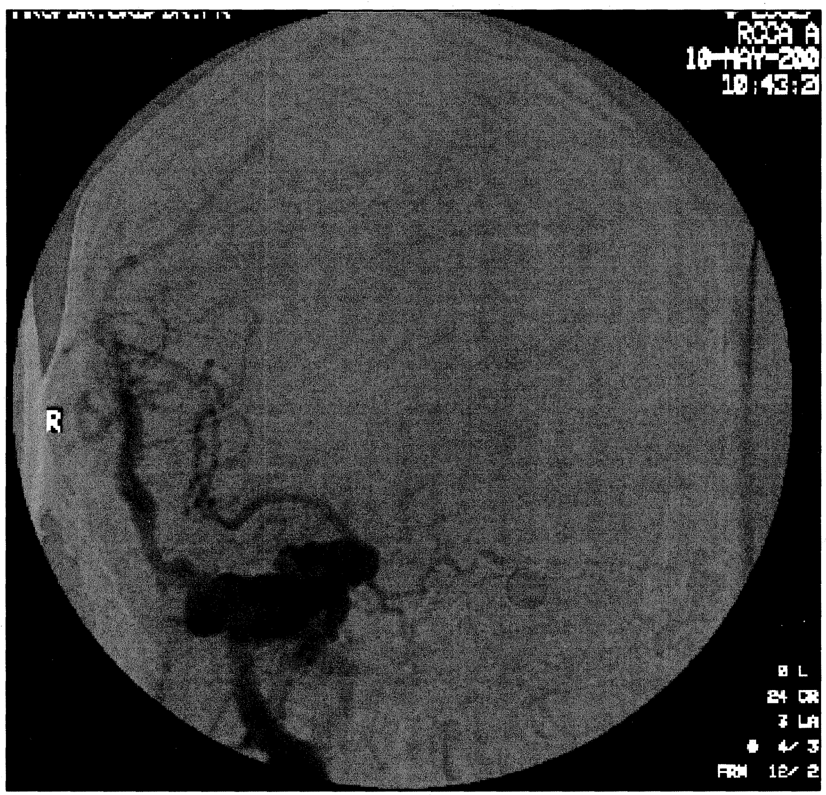
CASE-6 EHLER'S DANLOS SYNDROME



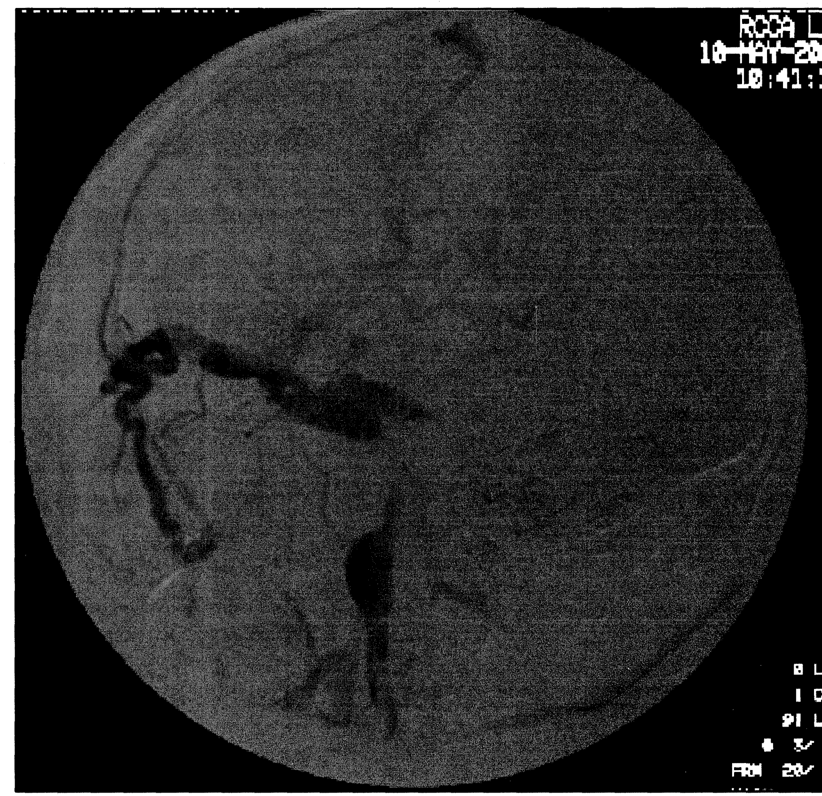
DYSPLASTIC RICA
WITH ANEURYSM

DYSPLASTIC LICA

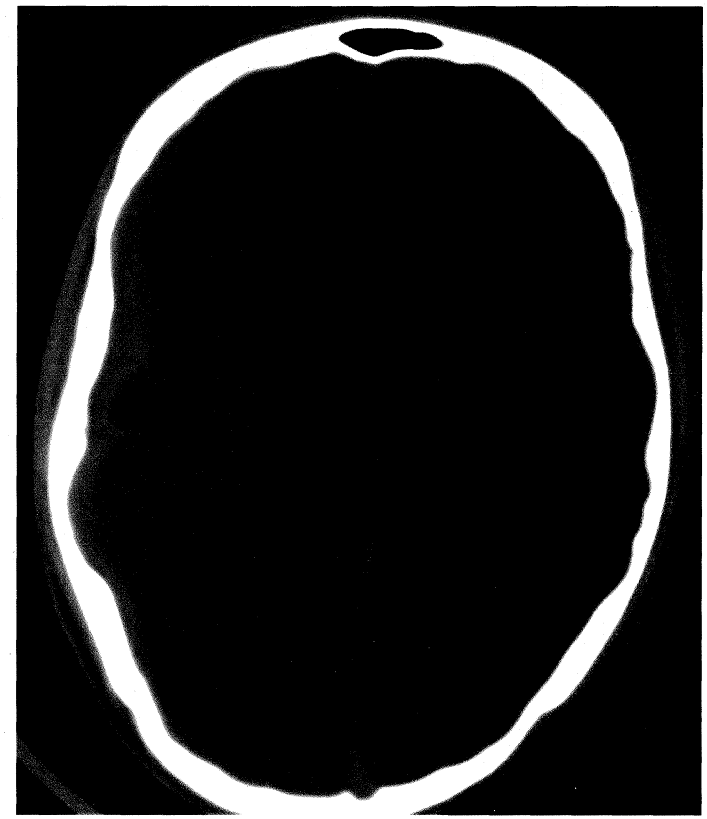
SPONTANEOUS CCF
PRE- EMBOLISATION



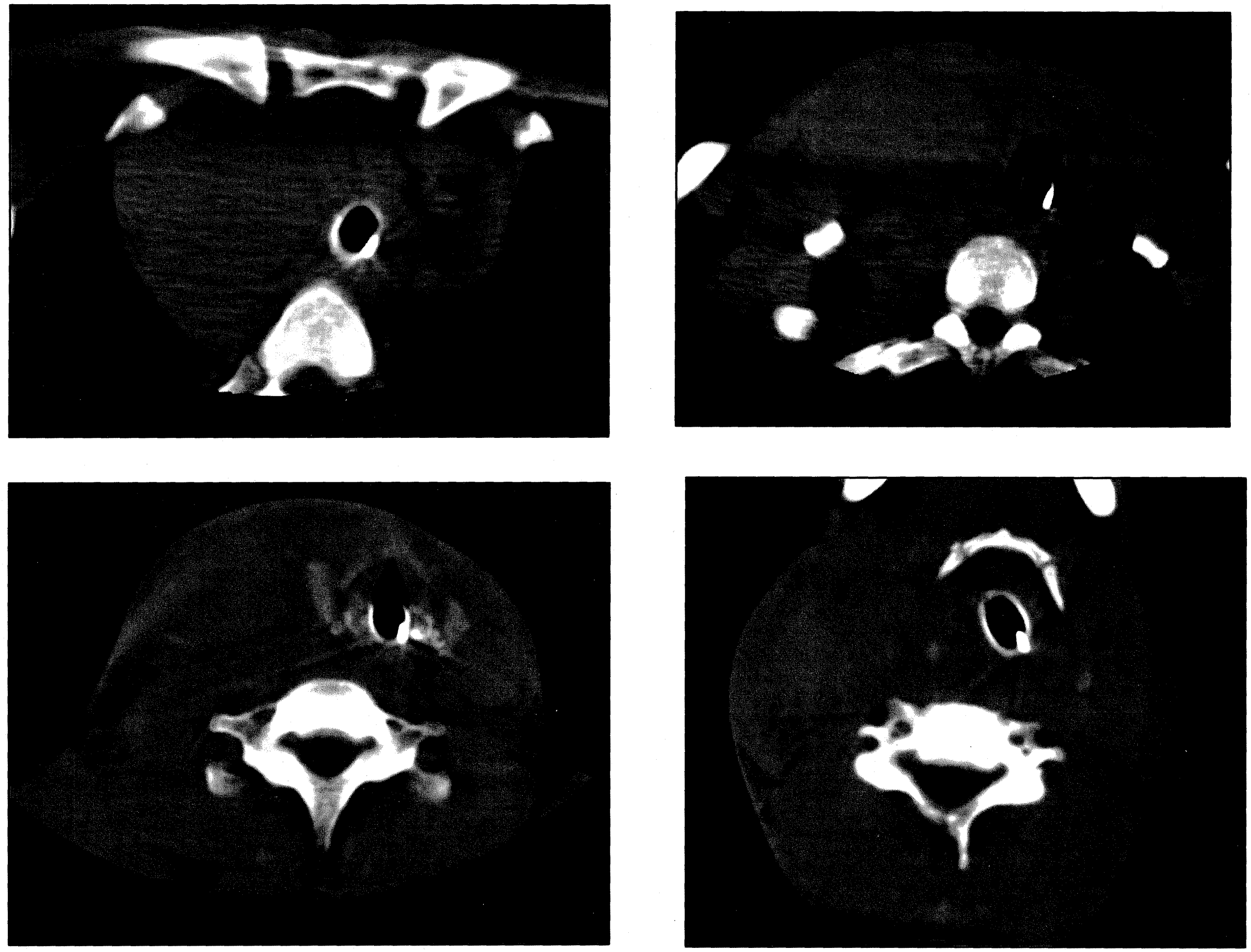
RICA AP VIEW



RICA LATERAL VIEW



SAH & RT SDH
WITH CEREBRAL EDEMA



CT SHOWS SPONTANEOUS RT CAROTID SHEATH HEMATOMA

CLINICAL PHOTOGRAPHS

PRE AND POST EMBOLISATION

DISCUSSION

DISCUSSION

CCFs are spontaneous or acquired connections between the branches of cavernous segment of ICA and the CS. According to Barrow et al, CCFs are classified into direct (type A) and indirect (types B–D) types .CCFs can be classified a) Pathogenetically into spontaneous or traumatic fistulas; b) Hemodynamically into high-flow or low-flow fistulas; and c) angiographically into direct or dural fistulas (4).

When the clinical diagnosis of CCF is established, adjunctive MR imaging or contrast-enhanced CT scanning is of little value, but maybe useful in establishing the degree of brain injury. CT is especially useful to identify skull fractures that may compromise the carotid artery lumen and create bony encroachment on the optic canal. The CT / MR show the dilated SOV, proptosis, bulging of the cavernous sinus and increased thickness of the extra ocular muscles .Dilated superficial veins, which represent an ominous sign of cortical venous drainage, can also be visualized. MRA can usually identify the presence of direct CCFs since the flow rate is usually rapid enough to allow detection; however the slower flow indirect CCF may be occult on MRA. Orbital doppler may also demonstrate all of these signs in addition to a reversal in the direction of the flow of blood in the SOV.

Noninvasive imaging is the corner stone of investigating patients presenting with connective tissue disorders / EDS .This prevents the risk of arterial injury such as rupture or dissection in these high risk patients. Once non-invasive imaging establishes the diagnosis, the best possible management for the lesion can be decided upon.

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

Cases where there has been prior treatment, particularly if it involved the sacrifice of a major feeder, the dural fistulas may recruit supply from one of the cervical arteries – either the ascending cervical arising from the thyrocervical trunk or the deep cervical arising from the costocervical trunk. These vessels also need to be studied in such situations. Each type of lesion, however presents its own sets of special considerations that should affect how the angiographic evaluation is conducted. Ideally, the initial angiographic work up should be sufficient to both establish a diagnosis and plan therapy.

ANGIOGRAPHIC CONSIDERATIONS PRIOR TO TREATMENT

The objectives of the initial angiogram are to:-

- Establish a diagnosis.
- Differentiate a direct CCF from an indirect CCF (DAVF).
- Determine the size and location of the fistula in the ICA in direct CCF.
- Identify any associated carotid cavernous aneurysm that may have ruptured.
- Identify all feeding arteries.
- Fully identify the venous drainage.
- Identify and confirm the patency of outflow pathways of the cavernous sinus.
- Identify high-risk features such as cortical venous drainage, pseudoaneurysm and CS varix.
- Identify associated vascular injuries in contralateral ICA and vertebral arteries (collateral circulation).

Venous drainage of the fistula

Two critical issues relate to the pattern of the venous drainage. First is the presence of cortical venous drainage from the fistula. This indicates the presence of intracranial venous hypertension and possibly impaired venous drainage. This constitutes a risk factor for stroke or hemorrhage. The second issue is whether the pathway of venous drainage would appear to permit retrograde catheterization for transvenous embolisation if indicated.

Once the fistula is demonstrated on angiography, with the help of the various maneuvers such as the Mehringer – Hieshima maneuver or the Alcock's maneuver, the treatment can be planned.

CURRENT APPROACHES TO TREATMENT

Transarterial embolisation

The efficacy and safety of detachable balloon occlusion of direct CCFs are well established. New approaches to and occlusion methods of CCFs s have been described, with increased focus on preserving ICA flow. These methods include the use of two-balloon techniques, use of GDC, permanent solidifying agents - Onyx, and even stents. Cyanoacrylates have the limitation of being less predictable in their behavior and are not recommended. Balloon embolisation from the venous side rarely works because the balloons are difficult to navigate against high pressure.

Transvenous embolisation

With the cavernous sinus, transvenous coil occlusion of fistula can be attempted in cases of dural CCFs or in cases where balloon embolisation has failed leading to failure of fistula obliteration or there is recurrence of fistula. To be effective, the occlusion must be at the point of the fistula. To perform the occlusion any further downstream might simply redirect the venous drainage and could increase the risk of hemorrhage.

There are various venous approaches to the CS and they include

- retrograde access via visible IPS.
- access via contralateral IPS.
- blind catheterization of non visualized IPS.
- catheterization of CS via EJV to FV to SOV.
- retrograde catheterization via pterygoid plexus – I/L or C/L.
- SOV cut down.
- Trans – orbital needle puncture of CS .
- Surgical exposure of CS.

TYPE A

For analyzing our cases we followed Barrow's classification. In our series, we had a total of 129 CCFs .Of these we encountered Type A fistulas in 70 (54.26%), type B in 9 (6.97%), type C in 8 (6.2%) and type D in 42 patients (32.55%). Barrows et al described treatment of CCFs in 14 patients , 2 were type A, 5 type B, one type C and six type D(4). In their report of 132 cases of CCFs, Debrun et al described 95 cases of type A, none of type B , 4 of type C and 28 of type D (34).

Type A CCF was noted in 70 patients out of which 50 (71.4%) were males and 20 (28.5 %) were females .The age range was from 6 years to 65 years (mean age 32.7 yrs). Duration of symptoms was from immediate to four years (mean 3.3 months).

Debrun described treatment of 54 traumatic CCFs .The youngest of their patient was a 5 year female and the oldest a 95 year female (17). Adam Lewis described 98 patients with 100 direct CCFs. Their patients were equally divided between males and females, ranging in age from 5 to 93 years (24). G.Bavinszki described six patients with direct CCFs, age ranging from 32 to 72 years (48).

After angiography in 70 patients we encountered 71 CCFs. The CCF was unilateral in 69 (98.5%) and bilateral in one patient(1.4%). Amongst unilateral CCF it was on the right side in 38 (54.2 %) and left sided in 31 patients (44.2%) The site of rent was at C2 in three patients, C3 in twenty six patients, C4 in ten patients, C5 in two patients, at C2-C3 junction 4 patients, at C3-C4 junction in 15 patients and C4-C5 junction in ten patients.

Amongst their 98 cases with 100 CCFs, Adam Lewis described 98 unilateral CCFs & one case of bilateral CCF while one patient had mirror image aneurysms that ruptured six years apart producing CCF (24). Yong described one case of bilateral CCF with bilateral carotid and vertebral dissection (23).

Out of these 70 cases, 57(81.4%) were post traumatic, 1(1.4%) was iatrogenic while 12(17.4%) were spontaneous. Debrun et al described 54 patients, 53 of these had known head injury & one had features of traumatic CCF though the patient did not recollect history of trauma (17) .In the series of 100 CCFs by Adam Lewis, 76 cases were of traumatic origin, 22 occurred after a ruptured aneurysm and 2 were iatrogenic (24).

Of the 70 patients, at presentation, swelling of eye was present in 64 (91.4%) & redness was noted in 53 (75.7%) patients, diplopia was noted in 32(45.7%), DOV in 12(17%) , visual loss in five patients (7.1%) and vision could not be tested in 2 patients (2.8%). Pain in eye was noted in 6 (8.5%), headache in 16 (22.8%), vomiting in 2(2.8%) & tinnitus in 16 patients (22.8 %).One of the patients had vertigo at presentation. Other associated features included ptosis in 3 (4.2 %), hempiaresis in 4 (5.7 %) patients, LMN facial in four patients (5.7%) while one patient had pus in the anterior chamber.

On examination, proptosis was noted in 66 (94.2 %), redness in 61 (87.1%), chemosis in 58 patients (82.8%).Lateral rectus palsy was noted in 32 (45.7%) with right 6th nerve palsy in 15 (46.8%) , left 6th nerve palsy in 17(53.1%), bilateral 6th nerve palsy in 1(1.4%) while multiple cranial nerve palsies (3rd, 4th, 6th) were noted in 15 patients (21.4 %).Other cranial nerve palsies were present in 13 patients (18.5%). EOM restriction was noted in 7 (10 %).Orbital bruit was present in 42 patients (60 %) and additional bruit was noted in 14 patients (20 %). Dysmorphic facies and features of connective tissue disorder were present in three patients (4.2%).

In the series by Adam Lewis the most common presenting symptom was orbital bruit noted in 78 patients, followed by proptosis in 71 patients, 54 had chemosis and 48 had isolated 6th nerve palsy .Headache and retro orbital pain was noted in 24 patients, opthalmoplegia in 23 patients, visual loss in 17patients (24).

Debrun et al have described the symptomatology of the patients and have correlated it to the angiographic features (17).

Of the 70 patients with type A CCF who presented to our institute for endovascular management, 63 patients (90%) underwent embolisation .For these 63, who underwent embolisation a combination of balloons only, balloons and coils and only coils was used one had stenting followed by coiling.Ten patients (14.2%) underwent ICA trapping, one (1.4%) showed spontaneous closure, one (1.4%) showed closure after attempting to pass a balloon and two (2.8%) were advised carotid compression. Three patients with EDS (4.2%) succumbed during catheterization for embolisation.

The spontaneous closure of direct CCFs is considered rare. Predisposing factors leading to spontaneous closure of direct CCFs are traumatic and iatrogenic ICA dissections, fistulas with a small ostium, partial balloon occlusion (>95%) closure, and attempts to place the balloon (24).

Of the patients who underwent balloon &/or coil embolisation (52 patients) , 43 (68.2%) patients showed fistula closure after the first embolisation . Seven patients (11%) had to undergo multiple attempts for closure .Of these 7 patients, recurrence in three was due to balloon deflation (this includes the patient with iatrogenic fistula), one due to deflation and displacement ,one due to repositioning of the balloon on detachment . Two other patients had minimal residual fistula after the first embolisation and later had to undergone a second embolisation .These seven patients had been embolised with only balloons in five patients & balloons and coils in two patients. Six patients demonstrated pseudoaneurysms. One of them underwent coiling of the pseudoaneurysm .

For embolising direct CCFs, balloons have been the mainstay as the embolising material with excellent results. The balloons were initially silicone and then latex. The balloons need to be inflated for which initially silicone was used followed by iodine, and then hyperosmolar contrast. Debrun (17) used balloons filled with silicone or iodine while Adam Lewis (24) used balloons of latex or silicone filled with hyperosmolar metrizamide. However considering the need CS packing for certain cases, coils were introduced as the embolising agents, after their use for coiling intracranial aneurysms had been established. G.Bavinszki has described the use of coils via transarterial and transvenous route in emolising the direct CCF in six patients (39).

Eleven patients (14.2%) had to undergo ICA trapping .Of these eleven patients , ten (12.8%) had undergone trapping of the fistula by the endovascular route and one (1.4%) via surgical route.

The reasons for endovascular trapping were varied. In two patients, the balloon could not be negotiated across the rent in ICA and hence the ICA had to be trapped. In the third case, the balloon slipped to the ICA after detachment partially occluding it, thus necessitating trapping. In the fourth case, the fistula persisted after first embolisation with balloons and hence it was decided to trap the ICA. This patient also showed recruitment of the ECA feeders on the second angiogram which were embolised with PVA with complete occlusion of the feeders. In two patients, the balloons caused extrinsic compression over the cavernous ICA after deployment - in one case after deployment of 9 balloons in the venous sac and in the other after 18 coils and 3 balloons. In another patient, the attempts at accessing the cavernous sinus via the arterial and venous route failed and hence ICA trapping was done to occlude the fistula. In the patient, the fistula the fistula was persistent and hence needed trapping. In the next patient, after deployment of 2 balloons , the catheter tip broke and could not be retrieved . The fistula had to be closed by trapping the ICA. One patient underwent surgical ligation of ICA after dissection during attempt for embolisation. One patient had ICA laceration during pituitary adenoma resection and had to undergo ICA trapping to occlude the fistula .

Of the ten patients who underwent endovascular trapping of the ICA, in seven cases the ICA was trapped from distal to proximal via the ICA itself. In two cases the ICA was trapped via PCom and in one case it was trapped by gaining access from the contralateral ICA and across Acom. We used a combination of various emolising materials to trap the ICA – balloons, GDC coils and fibre coils. Of these ten, 8 patients had undergone fistula trapping with complete closure of CCF at the first embolisation itself, while two had to undergo a second attempt for trapping with complete occlusion of the fistula at the second attempt.

Stuart C. Coley described ICA trapping in four patients with large high-flow shunts by using a combination of proximal balloon occlusion and distal coil embolization. They accessed ICA by retrograde catheterization of the distal parent vessel via the contralateral carotid or ipsilateral vertebral artery **(59).**

We had one case of iatrogenic CCF in which the ICA was trapped. He was a young male, and had undergone surgery for a pituitary adenoma. Intraoperatively, he sustained injury to the ICA with heavy bleeding. The bleeding was controlled with local insertion of muscle pieces, fat, and fascia which partially controlled the bleeding. An angiogram was done after the patient's clinical condition stabilized, two days later. A CCF was noted with fistula at the horizontal segment of cavernous ICA. Antegrade flow to the MCA was seen. Cross circulation however was poor. The fistula was occluded with 2 balloons with minimal sluggish flow across the fistula .Four days later, the patient had throbbing headache with increased proptosis, and recurrence of fistula was confirmed on doppler. It was due to deflation of one of the balloons. Subsequently the patient underwent a second attempt at fistula closure with fibre coils and latex balloons. Still the symptoms persisted and a check angiogram showed that the fistula persisted. It was then decided to trap the ICA in view of the persistence of fistula and symptoms. In this patient, since the cross circulation across Acom was poor, he suffered dense hemiplegia. However the occlusion of fistula was essential to save the patient's life.

Naci Kocer et al described a case in which iatrogenic injury to a patient's ICA with resultant CCF and massive hemorrhage was successfully managed with the emergency placement of an endovascular stent-graft (63).

Expedient treatment is of paramount importance in cases of iatrogenic CCFs as any delay may result in the patient's death .All treatment options should be thoroughly considered and discussed before a treatment decision is made. If intraoperative packing partially controls the hemorrhage and if a subsequent angiography reveals a lacerated ICA, occlusion of the ICA must then be performed with either surgical or endovascular means. A balloon occlusion test can be performed prior to ICA trapping. If the patient tolerates BOT, and then one can safely proceed with ICA trapping. However if it fails, skull-base bypass surgery may be attempted with a saphenous vein bypass or an ECA and/or MCA bypass (62, 76, and 77).

Overall the patency of the ICA was maintained in fifty six of our patients (80%) out of a total of 67 cases treated in all.

Higashida et al (75) reported preservation of the parent artery in 88% (*n* = 206) of the patients whom they treated using detachable balloons. Others describe a need for parent artery occlusion in as many as 20% of their cases (24, 34).

One of our patients, a 27 /M had sustained a road traffic accident .He had a LICA dissection of the cervical ICA and ipsilateral direct CCF. The fistula was accessed via the LICA itself and was closed completely by a single balloon. Following this the dissected segment was stented with a wallgraft. To cover the proximal segment of ICA completely another stent had to be deployed extending from the LICA to the LCCA .Check angiogram showed complete recanalisation of the ICA. However the left M1 segment showed occlusion which was thrombolysed with 4 lakh units of urokinase which established complete reopening. The patient had recurrence of fistula after 5 months which was embolised with 4 latex balloons with occlusion of the fistula. He had a second recurrence after fifteen days and again underwent embolisation with a silicone balloon with complete closure of fistula. There was no recurrence subsequently.

Though the occurrence of spontaneous arterial dissections and CCFs is known with collagen vascular disease like EDS, post traumatic occurrence of dissection and CCF in a patient without these diseases is very rare. Yong described a case of post traumatic CCF associated with bilateral carotid and vertebral artery dissection. The patient was managed conservatively (with heparin & warfarin).The CCF showed complete resolution at follow up angiography while the other arterial dissections showed significant improvement (23).

One of our patients had sustained a road traffic accident and had sustained major injuries. He had developed bilateral CCF immediately after trauma .He was managed elsewhere for his primary injuries, and was referred to our institute for management of his CCF. Angiography demonstrated bilateral direct high flow fistulae with preserved forward flow in the ICA. In the first attempt, bilateral fistulae were occluded with one balloon on either side that led to complete closure of the left fistula and had a minimal residual on right. In the second attempt stent assisted coiling was planned for the residual CCF on right. A Leo stent (4.5 X30mm) was deployed across the fistula but later the catheter could not negotiate beyond the balloon. Hence coiling could not be done .Also the venous route attempt failed. In the third attempt the right facial vein was punctured and both SOV and IOV were coiled with a total of 40 coils. Significant reduction of the fistula was achieved. A check angiogram a week later showed persistence of fistula on the right. This time, the fourth time, the intercavernous communication was coiled as also the right cavernous sinus with a total of 58 coils. This resulted in complete closure of the fistula .Bilateral CCF resolved .On follow up, the proptosis had completely resolved and the patient had regained his eyesight but had restricted eye movements with a left lower hemianopia,

Most traumatic CCFs are unilateral, and simultaneous bilateral traumatic CCFs are uncommon. .Over a period of 15 years, Luo CB et al described bilateral CCF in 5 men and 2 women with a mean age of 31 years out of a total of 252 consecutive patients with traumatic CCF. All patients underwent a single session of transarterial embolisation. After treatment, it was possible to preserve one or both ICAs in both patients.

Lee et al (78) reported successful treatment of a patient with a traumatic aneurysm of the supraclinoid internal carotid artery and an associated carotid cavernous fistula with a balloon-expandable coronary stent and coils The same technique was used by Ahn et al and Men et al (79, 80) in the treatment of traumatic CCFs. Redekop et al (57) reported 6 patients with traumatic vascular lesions of the skull base that were successfully treated using endovascular stents.

The use of stents can help the management of fistulas where there are multiple arterial lacerations. It helps preserve the ICA patency, which should be the goal of embolisation.

Fanny E. Morón described a total of 5 patients presenting with 6 high-flow type A CCF (one had a bilateral fistula) that were associated with severe laceration of the internal carotid artery. They treated all patients first with stenting of the injured segment of the internal carotid artery followed by transarterial [3/6] and/or transvenous [4/6] obliteration of the fistula with detachable platinum coils (81).

The use of stent-assisted coiling allows staged successful reconstruction of the damaged internal carotid artery and then obliteration of the fistula with preservation of the parent artery. Because most stents used for this application are balloon-expandable, the ability to carefully analyze the morphology of the fistula and to accurately measure the size of the ICA at the site of injury is critical because it allows correct choice of the size of the stent to be deployed. Improper choice of stent size may lead to an increased incidence of adverse events because undersizing with poor stent apposition may cause thrombus formation, whereas oversizing may result in arterial rupture (81).

Spontaneous CCF in patients with connective tissue disorders such as EDS remain a challenge to the endovascular surgeon. We had three patients who had EDS – two were females and one was a male. All three patients had arterial ruptures during catheterization for embolisation to which they subsequently succumbed - one had aortic rupture, one an iliac artery dissection and the third had carotid rupture and carotid sheath hematoma.

Describing one case of their own, R.Fox et al reviewed the literature on five patients with EDS having CCF. Angiographic investigation in these patients by direct puncture or arterial catheterization was complicated by local hemorrhage or arterial rupture in four and in two patients the carotid artery was noted to be friable during subsequent ligation. In one patient, the fistula was closed by transvenous balloon embolisation. However, all five patients died either during investigation or from arterial rupture occurring later at a site remote from the fistula (82).

A transarterial route has been successfully described in literature for embolisation of CCF in EDS. Debrun et al in 1996 described three cases of spontaneous CCF associated with EDS out of a series of 147 direct CCF (83).They also stated the difficulties encountered in the management of these patients.

Andrew A Kanner et al in 2000 described the first successful transvenous embolisation of a patient with EDS who had a spontaneous direct CCF. They approached the CCF via SOV and used regular and fibre coated GDC for embolisation. Considering the friability of the arterial and venous walls and the tortuousity of the vessels, any surgical or endovascular intervention carries a high risk of vessel perforation and rupture with associated morbidity & mortality. The authors recommend a transvenous approach via IPS or SOV for treatment of these patients , avoiding entry into the friable/dysplastic ICA .Manipulation of catheters should also be kept to a minimum .For embolisation GDC should be preferred over balloons. Coils offer less traumatic occlusion than balloons since they are softer, easier to control and exchangeable .They also recommend the use of fibre coated GDC for added thrombogenecity (84).

Hollands JK recently described a new approach they attempted to treat CCF in a ptient with EDS –IV. They surgically exposed the ipsilateral carotid arteries and the internal jugular vein and inserted two endovascular sheaths. The sheaths were then used for embolization access. The fistula was closed, with preservation of the carotid artery, using Guglielmi detachable coils deployed in the cavernous sinus from the arterial and venous sides. Rapid resolution of symptoms and signs followed in their patient which was sustained at 6-month follow-up (85).

Various other conditions eg. stenosis, dissections can coexist in patients who develop CCF .One of our patient had an associated persistent trigeminal artery.

COMPLICATIONS

We encountered various complications during the management of these patients. In four of our patients, the balloon had immediately deflated and migrated to the lung during deployment necessitating the placement of a second balloon .The catheter tip broke in three patients, and in one the broken tip could not be retrieved .The tip did not migrate distally and the patient subsequently underwent ICA trapping. In the other two cases, the catheter with the broken tip could be retrieved. In one case it was retrieved surgically and the CCF was subsequently embolised.

Three patients developed MCA infarcts .One patient who had undergone ICA trapping for iatrogenic fistula, developed a massive right. MCA infarct since the cross circulation was poor. However trapping the fistula was essential for saving the patient's life. The other patient had a recurrent fistula and in the last attempt to close the fistula, the balloon caused extrinsic compression over the ICA with no distal flow. Since the cross circulation was poor; he developed a massive MCA infarct and SDH and succumbed to it in next 24 hours. The third patient developed a thrombus in the ICA during catheter manipulation which was successfully lysed with urokinase .However the patient later developed right MCA infarct. On follow up, he recovered the power gradually. The same patient had pus in the anterior chamber at presentation and subsequently had to undergo enucleation of the eye.

Early on in their experience, Debrun et al (17) described the occurrence of symptomatic false aneurysms in five of their patients. Occulomotor nerve palsy occurred in 20% of the cases in whom the balloon was inflated with silicone. Other complications described by the authors occurred during surgical management of the fistula. They cautioned regarding the use of bucrylate either surgically or intra-arterially .In none of our cases of type A CCF, bucrylate has been used for treatment.

Adam Lewis et al described various complications in their series. Transient ischemic episodes occurred in three patients treated with latex detachable balloons which later resolved with either aspirin or heparin. Fistulas recurred in 4 patients due to shifting of detachable balloons. Four balloons deflated prematurely with fistula recurrence .There were no permanent CN palsies after the placement of detachable balloons. Two patients suffered neurological morbidity due to transient ischemia and another had cerebral infarction which the authors described to have occurred due to an embolus from the catheter before the routine use of systemic heparinisation for the procedures. Their permanent neurological complication rate was 4% including one cerebral infarction, one frontal ICH, one aseptic CS thrombosis with U/L vision loss, and one death related to MCA infarction after a balloon prolapsed into & occluded the ICA at the ostium of the fistula.

We had three patients with EDS who succumbed to the arterial dissections during catheterization for embolisation of their fistula ..

In the first case, a young female, the patient was taken for embolisation.During catheter manipulation , she complained of sever left sided chest pain . A check angiogram revealed perforation at the origin of the left CCA .The patient was taken for thoracotomy and was noted to have massive hemothorax. The patient succumbed before any surgical intervention could be done. Post mortem revealed the highly fragile arterial walls and cardiac chambers. The second patient patient, also a young female, during passage of the guide wire the patient complained of sudden severe pain in the lower abdomen and developed hypotension. At surgery, she was noted to have right iliofemoral dissection which was treated with placement of an iliofemoral prosthetic graft. The patient survived for 19 days ,but on the 20^{th} day had a seizure to which she succumbed. The third patient was a young male .During passage of the catheter into the ICA the patient suddenly developed left sided neck swelling and became tachypneic. An immediate CT revealed a large carotid sheath hematoma causing tracheal compression .A brain CT revealed SAH and SDH .

Elaborating the complications of endovascular management of CCF in patients of EDS they treated , Michael B. Horowitz et al have described the causes of death in two of their patients .In one patient after occlusion of the fistula , the patient developed a

LICA dissection (which was confirmed angiographically and patient managed conservatively) . Three days later, a follow-up arteriography was performed to view the ICA dissection. At the conclusion of the procedure, the patient vomited violently and became hypotensive and tachycardic. An abdominal CT showed diffuse retroperitoneal and abdominal bleeding. An immediate laparotomy revealed a ruptured splenic artery. The vessel was ligated, and hemostasis ultimately was achieved. After eighteen days of the procedure, the patient suddenly became unresponsive, with hypotension and tachycardia and succumbed to it. Postmortem examination revealed a ruptured cardiac posteromedial papillary muscle. The authors speculated that though the splenic artery and cardiac papillary muscle ruptures were unrelated to the procedure itself, Valsalva maneuvers during postanesthesia retching may have contributed to rupture of the splenic artery aneurysm.

In the second case, the patient had undergone successful transvenous embolisation of a left direct CCF. Subsequently over a time period the patient developed multiple spontaneous arteriovenous fistulae -a left VA fistula, a right direct CCF and a left VA fistula. The patient underwent stent assisted coiling for the left VA fistula and stenting for right VA fistula. After 20 minutes, while in the ICU, the patient became tachycardic and hypotensive. An immediate arteriogram showed a right iliac artery perforation with active retroperitoneal bleeding. A balloon was advanced across the bleeding site and inflated that arrested the bleeding. Stenting across the bleeding site was also carried out hoping that it would add strength to the vessel wall and allow for direct surgical repair. Surgical exposure of the iliac artery was accompanied by significant venous bleeding because of the fragility of the patient's tissues. The iliac artery ultimately ligated. During the exposure, it was noted that in addition to the initial vessel blowout, the proximal stent had also perforated the vessel. The patient ultimately suffered excessive blood loss in the ICU shortly after surgery and died (74).

Once a peripheral vessel ruptures, repair is difficult. However, in two of our patients surgical repair was contemplated . In 1987, Cikrit and Miles (86) reviewed the available cases in the English-language medical literature and found that aortic rupture or rupture of major vessels was uniformly fatal. Bergqvist (87) emphasized the frequent futility in trying to salvage EDS 4 patients once major vessel injuries occur and recommended that diagnostic procedures requiring arterial puncture be avoided except in extreme circumstances. When injuries do occur, he suggested nonsurgical therapy, if possible.

We feel that as described in the literature, the venous route should have been attempted in the last two cases; having had the experience in the first case .Also repeated passage of catheters in the arteries and veins should be minimized in all cases of EDS patients undergoing endovascular management.

INDIRECT CCFs

Indirect CCF includes Barrow's type B, C, D. They are usually spontaneous and low flow. Various authors have described the symptomatology of spontaneous CCF and dural fistulas.

Halbach et al described dural CCF in 30 patients. In their patients 23(77%) had proptosis, 14(47%) had bruit, 15 patients (50%) had diplopia from opthalmoplegia and 8 (27%) had diminishing visual acuity (53).

M.Theaudin et al studied 27 patients. The most common clinical symptoms and diagnosis were diplopia in 12 (45%), conjunctival injection in 11(41%), chemosis in 10(37%), proptosis in 10(37%) and visual deterioration in 8 (30%) patients (31).

TYPE B CCF

Type B CCF was noted in 9 patients out of which 5 (55.5%) were males and 4 (44.4%) were females .The age range was from 30 years to 67 years (mean age 49 years). Duration of symptoms was from 10 days to 17 months (mean - 4months 7 days). Out of these 9 patients, 1(11.1%) was post traumatic, 8(18.8%) were spontaneous. The onset was acute in 2 patients (22.2%) and gradual in 7 patients (77.7%).

Barrow described five patients with spontaneous type B CCF .There were 4 females, one male with age ranging from 53 to 81 years (4).

Of the 9 patients, at presentation, swelling of eye was present in all patients (100%), it was right sided in 6(66.6%) and on the left in 3(33.3%). Redness was noted in 9 (100 %) patients, diplopia was noted in 6(66.6%), DOV in 2(22.2%). Pain in eye was noted in 2 (22.2%), headache in 3(33.3%) & tinnitus in 1 patients (11.1 %) .

On examination, proptosis was noted in all 9 (100 %), redness in 9 (100%), chemosis in 8 patients (88.8%),lateral rectus palsy was noted in 6(66.6%) with right 6^{th} nerve palsy in 5 (55.5%) , left 6^{th} nerve palsy in 1 (11.1%) while no patient had a bilateral 6^{th} nerve palsy. Multiple cranial nerve palsies (3^{rd}, 4^{th}, 6^{th}) were noted in 1 patient (11.1 %). Orbital bruit was present in 2 patients (22.2 %).

After angiography in these 9 patients, there were 11 CCFs. The CCF was unilateral in 7 (77.7%), it was on the right in 6 (66.6%) and left in 1 patient (11.1%); while two patients (22.2%) had a bilateral CCF.

Of our nine patients with type B CCF, 6 (66.6%) were advised carotid compression and 5 (55.5 %) showed complete resolution of symptoms at follow up.

In the series of indirect CCF by Halbach et al, carotid compression therapy resulted in complete closure of fistula in 30% of the patients (53).

One of the 9 patients (11.1%) with type B, one showed spontaneous occlusion of the fistula. She had undergone a diagnostic angiogram and was awaiting embolisation. Subsequently, when the patient was taken for embolisation about 9 days later, the diagnostic angiogram showed obliteration of the CCF, thus consistent with a spontaneous closure.

Low flow dural CCFs have a relatively high incidence of spontaneous closure. Barrows (17) encountered spontaneous closure of spontaneous CCF in five of 14 patients (36%). This spontaneous cure can also be observed in bilateral spontaneous CCFs as described by Voigt et al (29).

A.M.vd Vliet et al described two cases of bilateral direct CCF, one of traumatic and one of spontaneous origin. Both the fistulas were slow flow and demonstrated spontaneous closure. They stressed the point that low flow spontaneous CCF, direct or indirect, can undergo spontaneous closure (55).

Seeger et al believed that a spontaneous cure of dural cavernous fistula in most cases is due to partial or complete thrombosis of the cavernous sinus and/or its tributaries (30).

One patient with type B CCF underwent transvenous embolisation. The route was from EJV, FV, AV and SOV to CS. A total of 17 GDC coils were deposited, which resulted in total closure of fistula. One of the patients could not undergo embolisation since there was no venous access.

On follow up 7 (77.7%) of the fistulas remained closed while one patient showed minimal residual symptoms and one continues to be symptomatic (the patient with no venous access). Of the 6 patients who had been advised carotid compression, the fistula closed in all six patients. There were no morbidities/mortality in these patients.

TYPE C CCF

Type C CCF was noted in 8 patients out of which 2 (25%) were males and 6(75 %) were females .The age range was from 24 years to 72 years (mean age 48.7 yrs).

Duration of symptoms was from 15 days to 6 years (mean 19 months). All eight type C CCFs were spontaneous in origin and gradual in onset.

One case of type C fistula described by Barrows was a 63 year female who had spontaneous closure of her CCF (4). Debrun described 4 cases of type C CCF in their series of 132 CCFs (34).

Of the 8 patients, at presentation, swelling of eye of eye was present in 7 patients (87.5%), being on the right in 2(25%), left in 5 (62.5%) and bilateral in one .Redness was noted in all patients (100 %), diplopia was noted in 6(75 %), DOV in 3(37.5 %).Pain in eye was noted in 4 (50 %), headache in 2 (25 %) , vomiting in 1(12.5 %) & tinnitus in 3 patients (37.5 %) and headache in two .None of the patients had vertigo at presentation. Other associated features included photophobia in 1 patient (12.5%).

On examination, proptosis was noted in 7 (82.5%), redness in 8 (100%), chemosis in 7 patients (82.5%).Lateral rectus palsy was noted in 6 (75%) with right 6th nerve palsy in 1 (12.5%) , left 6th nerve palsy in 3(37.5%), bilateral 6th nerve palsy in 1(12.5%) while multiple cranial nerve palsies (3rd, 4th, 6th) were noted in 1 patient (12.5 %). Orbital bruit was present in 5 patients (62.5%).

After angiography in eight patients there were 10 CCFs. The CCF was unilateral in 6 patients (75 %) and bilateral in 2 patients (25%). Amongst unilateral CCF it was on the right side in 2 (25 %) and left sided in 4 patients (50%).

All of our eight patients underwent embolisation of the fistula. Seven patients underwent transarterial embolisation of the feeders while one patient underwent transvenous embolisation via the IPS to CS route. Of the eight cases, after the first embolisation, 2 patients showed total closure, 4 showed near total closure, while 2 showed minimal residual filling of the fistula. Two of the patients had recurrence of symptoms, both underwent angiography for the delineation of the feeders. In one patient on identification of the feeders, the fistula was embolised with PVA and showed >90% angiographic occlusion and subsequent total closure. At subsequent follow up, seven patients (87.5%) showed total resolution of symptoms while one had progressed and underwent re-embolisation .He showed persistent symptoms at follow up and was advised transvenous embolisation. There were no morbidities or mortalities in these cases

TYPE D CCF

Type D CCF was noted in 42 patients out of which 20 (47.6%) were males and 22 (52.3 %) were females. The age range was from 13 years to 72 years (mean age 48.8 years). Duration of symptoms was from 10 days to three years (mean 3 months).

Out of these 42 patients, 39 (92.8%) were spontaneous while 3 (7.1%) were post traumatic. The onset was acute in 6 patients (14.2%) and gradual in 36 patients (85.7 %).Of the patients who presented with h/o trauma no patient had cranial fractures or loss of consciousness.

Halbach et al described 30 cases of indirect fistulas of the cavernous sinus out of a total of 158 cases of CCF. Of these 30, 22 were females and eight were males .The patient's age ranged from 22 to 75 years with a mean of 47 years (53).

M Théaudin et al described 27 cases of dural CCFs .There were 18 females and 9 males with age ranging from 32 to 80 years (31).

Simon Yu et al described 98 patients with dural CCF .Of these, 73 were females and 25 were males (average age 57 .6 years). Twenty patients had bilateral CCF, while 42 had right and 36 had left dural CCF (48).

Of the 42 patients, at presentation, swelling of eye was present in 33 (78.5%), it was on the right in 11(33.3%) , on the left in 15(45.4%) and was bilateral in 7 patients (21.2%) .Redness was noted in 35 (83.3%) patients, diplopia in 29(69%), DOV in 11(26.1%), one patient was blind in the left eye. Pain in eye was noted in 12 (28.5 %), headache in 19 (45.2%), vomiting in 4 (9.5%) & tinnitus in 8 patients (19%). Other associated features included watering in 4 (9.5%) ptosis in 3 (7.4 %) & left upper limb weakness in 1 patient (2.3 %).One patient (2.3%) presented with cerebellar hemorrhage.

On examination, proptosis was noted in 33 (78.5%), redness in 39 (92.8%), chemosis in 30 patients (71.4%).Lateral rectus palsy was noted in 29 (69%) with right 6^{th} nerve palsy in 8 (19%) , left 6^{th} nerve palsy in 13 (30%), bilateral 6^{th} nerve palsy in 4(9.5%) while multiple cranial nerve palsies (3^{rd}, 4^{th}, 6^{th}) were noted in 4 patients(9.5 %),only third nerve palsy in 5 (11.9%)& other nerve palsies in three (7.1%). Orbital bruit was present in 5 patients (11.9%) and additional bruit was noted in 1 patient (2.3 %).

In the series of 30 cases by Halbach et al, the presenting symptoms were proptosis in 23(77%),bruit in 14 (4&%), diplopia in 15 (50%) and diminishing visual acuity in 8 (27%) (53).

In the series of 27 cases by M.Theaudin et al , most common clinical symptoms at diagnosis were diplopia in 12 (45%), conjunctival injection in 11(41%) , chemosis in 10 (37%), proptosis in 10 (37%) and visual deterioration in 8 (30%) patients (31).

After angiography in 42 patients we encountered 68 CCFs. The CCF was bilateral in 26 patients (61.9%) and unilateral in 16 (30.8%). Amongst unilateral CCF it was on the right side in 8 (19 %) and left sided in 8 patients (19%).

Of the 42 patients, 34 underwent embolisation .Of these, twenty five embolization were via the arterial route only, nine via venous route only, and 1 via both arterial and venous route. Seven patients were advised carotid compression .One patient had a seizure just prior to the procedure. On CT, she was found to have perimesencephalic venous drainage with associated venous pouch .She suffered a cerebellar hemorrhage and SAH, to which she succumbed (no embolisation was carried out).

Of the 34 patients who underwent embolisation for type D CCF, one patient had undergone trapping of ICA (for a type A fistula) elsewhere 2 years ago and had now presented with recurrence of fistula, this time a dural CCF. The cavernous sinus was accessed via the PCom and occluded with GDC coils. Minimal residual blush was noted from the ECA branches .The symptoms persisted and an angiogram done 4 days later demonstrated recurrence of fistula due to coil compaction .The cavernous sinus was further packed with coils and ECA feeders embolised with PVA with near total closure of the fistula. 3 months later, due to persistent symptoms, a repeat angiogram was done that revealed feeders from the ECA with recruitment of ophthalmic artery feeders. The ECA was embolised with hydrogel particles with total obliteration of the blush. A follow up was embolised with hydrogel particles with total obliteration of the blush. A follow up TCD one month later demonstrated SOV thrombosis. There was no recurrence of fistula subsequently.

Seven (16.6%) patients were advised only carotid compression. Two (4.7%) of these , and one who had undergone arterial route embolisation had a recurrence of fistula demonstrated on angiogram .However these patients were diabetic and had poorly controlled diabetes at the time of recurrence. These patients were advised strict diabetic control and to continue with carotid compression. Follow up angiogram in these patients after diabetic control demonstrated complete resolution of fistula. It should be noted that patients who had been advised only carotid compression had recurrence of their fistula when their diabetis flared up. This is an important observation .Hence we advice that all patients of indirect CCF, who have concurrent diabetes, should have a very strict control of their blood sugar. Failing this, the CCF might recur.

In the series by Halbach et al, carotid compression therapy resulted in complete closure of fistula in 30% of the patients treated (53).

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

Miller et al have described the treatment of 12 CCFs via the SOV approach. All their patients were successfully treated either by advancement of detachable balloon catheter to the CS with subsequent inflation and detachment (11patients) or by introduction of multiple coils into the fistula (1 patient) .All patients had complete fistula closure and resolution of symptoms and signs after occlusion of the CCF (47) .

Derang et al have described the SOV approach and have successfully treated 21 cases of CCF (44).

There were two other patients who had undergone arterial and venous route embolisation together and showed complete obliteration of the fistula. In one of these patients, we had accessed the CS via the contralateral thrombosed IPS.

Bendorf G described four cases in which they accessed the CS via the ipsilateral thrombosed IPS (42).

Of the 34 patients who underwent embolisation, after first embolisation ,17showd total closure ,13 near total ,while 4 had residual (of these 4,3 underwent multiple attempts). These 17 patients were advised concurrent carotid compression and on follow up they showed total resolution of symptoms. One patient who had undergone arterial embolisation showed partial obliteration and is awaiting venous route embolisation. Of the 5 patients who underwent venous route embolisation of their CCF,in four, the patent

IPS was chosen for entering the CS. In one case we accessed the CS via the thrombosed IPS.

Of the patients who underwent repeat embolisation, one underwent three more sessions of embolisation. This patient had recruitment of OA feeders. The IMA and OA feeders were embolised with bucrylate with total angiographic closure of the fistula.The patient later developed CRA occlusion with total visual loss.

One patient had a post traumatic type D fistula for which she underwent both arterial and venous route embolisation (PVA embolisation for ECA feeders and CS coiling) at the first attempt with complete closure of the fistula. However the symptoms recurred one month later and the patient underwent further coiling of the cavernous sinus with total closure. A follow up angiogram showed no recurrence of fistula after two years.

COMPLICATIONS

Complications occurred in 5(11.9%) patients who underwent treatment for CCF type D. These included CRA occlusion resulting in total blindness in one patient. This was following bucrylate injection into the re-recruited IMA and OA feeders.

Embolic infarcts occurred in two patients in the ACA and MCA territory one had infarcts in the ACA territory and the motor cortex & the other had watershed infarcts in bilateral ACA-MCA territory. Both these patients had major neurological deficits, which improved subsequently; however, the patients remained debilitated over a period of time.

Two patients who underwent particle embolisation of the ECA feeders, had dissection of the IMA at the conclusion of the procedure. However no neurological deficits resulted.

Halbach encountered three transient and one permanent complication .In one patient liquid adhesive glued a catheter tip into the distal IMA .One patient had a transient increase in proptosis from occlusion of venous outflow of the orbit .One patient had not undergone selective angiography and suffered a transient decrease in vision when the unrecognized meningolacrimal artery was embolised with PVA particles. The fourth patient suffered a stroke with residual mild hempiaresis when thrombus was dislodged from catheter after removal from a direct carotid puncture(53).

M.Theaudin et al in their series of 27 patients with dural CCFs had one patient who had a cerebral hemorrhage after transvenous embolisation of the fistula with leptomeningeal drainage (31).

CONCLUSIONS

Conclusions

CCFs are one of the vascular conditions where endovascular management has become the present day treatment of choice .

The features of proptosis, redness, chemosis, diplopia, diminution of vision are the commonest symptoms and should alert the physician of a possibility of CCF .The symptomatology of direct CCF is more pronounced while that of indirect CCF may be subtle. A timely clinical evaluation followed by angiogram can direct the early management of these lesions. It is essential to identify high risk patients to provide emergent management with a view to preserve the vision as best as possible.

Non invasive modalities should be the first line imaging for patients with connective tissue disorder like Ehler's Danlos syndrome. Management should be very cautious with minimal vascular injuries.

The fistulas (direct) close totally with latex balloons. In cases where balloons fail to close the fistula completely or there is recurrence, use of coils needs to be considered. A combination of the various embolising materials can be chosen from. The CS packing can be done with varied types of coils as per the angiographic features of the given case.

The goal of endovascular management should be obliteration of the fistula and preservation of the ICA patency. Stents – bare or covered can be very beneficial especially for preserving the parent artery while closing the fistula in difficult cases.

Given the present day expertise, technology, machines and available materials endovascular management of CCF has extremely good results.

The mortality or morbidity associated with endovascular management is very low in the present day. Previously encountered morbidities were due to inexpertise and technical limitations.

A detailed clinical evaluation, imaging work up, angiographic analysis and timely follow up for all patients with carotico-cavernous fistulas is recommended.

REFERENCES

1- Seldinger SI.
Catheter replacement of the needle in percutaneous arteriography; a new technique.
Acta Radiol 1953; 39:368-76.

2- Dotter CT, Judkins MP.
Transluminal treatment of atherosclerotic obstructions: description of a new technique and preliminary report of its applications.
Circulation 1964; 30: 654–670.

3- Serbeninko FA
Balloon cathetrisation and occlusion of major cerebral vessels.
J Neurosurgery 1974; 41: 125-145.

4- Daniel L. Barrow, Robert H. Spector, Braun, Jeffrey A. Landman, Suzie C. Tindall and George T. Tindall .
Classification and treatment of spontaneous carotid-cavernous sinus fistulas.
J Neurosurgery 1985 ;62: 248-256.

5- Van V. Halbach, Randall T. Higashida, Grant B. Hieshima, Carl W. Hardin, Peter J. Yang
Transvenous embolization of direct carotid cavernous fistulas.
AJNR 1988 ;9:741-747.

6- Halbach VV , Higashida RT , Larsen DW et al
Treatment of dural arteriovenous fistulas , In Maciunas RJ , editor .Endovascular neurologic intervention .
Park Ridge : American association of neurologic surgeons , 1955 ;217-246 .

7- Ciesielski T, Stefanowicz E, Brzozowski E
A case of post-traumatic carotid-cavernous fistula in a 9-year-old girl.
Neurol Neurochir Pol1984; 18 :65 –67.

8- Jorgensen JS, Guthoff R
Opthalmoscopic findings in Carotico cavernous fistula : an analysis of 20 patients .
G raefes Arch Clin Exp Opthalm 1988 ;226 : 34 -6 .

9- Sanders MD , Hoyt WF
Hypoxic ocular sequelae of carotid –cavernous fistulae .study of the causes of visual failure before and after neurosurgical treatment in a series of 25 cases .
Br J Opthalm 1969 ;53:82-97.

10- Wilms G
Unilateral double carotid cavernous fistula treated with detachable balloons
AJNR 1990 ;11;517.

11- Donald W Larsen
Traumatic vascular injuries and their management
Neuroimaging clinics of North America , 12 , 2002, 249-269.

12- Takahashi M, Killeffer F, Wilson G ,
Iatrogenic carotid cavernous fistulas : Case report .
J Neurosurgery 1969 ;30 :498- 500 .

13- Flandroy P, Lenelle J, Collington J.
Carotid cavernous fistula associated with Fogarty catheter angioplasty .
AJNR Am J Neuroradiol 1988 ,9,1242 .

14- Barker WF, Stern WE ,Krayenbuhl h et al
Carotid endarterectomy complicated by carotid cavernous sinus fistula
Annals of Surgery 1968 ,167 ,568-72 .

15- Teitelbaum GP, Bernstein K, Choi S,et al
Endovascular management of biopsy related posterior inferior cerebellar artery pseudoaneurysm.
Surgical Neurology 1995 ,43, 357-9.

16- Paul Philips
Carotid cavernous fistulas
Neurosurgery Clinics of North America ,1999,vol 10,653-667.

17- Gerard Debrun, Pierre Lacour, Fernando Vinuela, Allan Fox, Charles G. Drake, and Jean P. Caron .
Treatment of 54 traumatic carotid-cavernous fistulas.
J Neurosurgery1981 ; 55 :678-692.

18- Donald J Prolo and John W Hanberry .
Intraluminal occlusion of a carotid cavernous sinus fistula with a balloon catheter.
Neurosurgery ,1971 : vol 35; Aug , 237-242 .

19- Parkinson D, Downs AR , Whytehead LL, Syslak WB
Carotid cavernous fistula : Direct repair with preservation of carotid
Surgery 1974 ; 76 :882 – 889 .

20- Mullan S.
Treatment of Carotico cavernous fistula by cavernous sinus occlusion
J Neurosurg 1979 ,50:131-144

21- Debrun G, Lacour P , Caron JP et al
Detachable balloon and calibrated leak balloon techniques in the treatment of cerebral vascular lesions
J Neurosurgery ,1978 ;49: 635-649.

22- Adam I. Lewis, Thomas A. Tomsick, John M. Tew, Jr.
Management of 100 consecutive direct carotid-cavernous fistulas: Results of treatment with detachable balloons.
Neurosurgery 1995:36 ,No 2;239-245 .

23- R.L. Young and N.S. Heran
Traumatic carotid cavernous fistula with bilateral carotid artery and vertebral artery dissections.
Acta neurochirugica (wien) ,2005;147 :1109- 1113.

24- Adam I. Lewis, Thomas A. Tomsick, John M. Tew, Jr.
Management of 100 consecutive direct carotid-cavernous fistulas: Results of treatment with detachable balloons.
Neurosurgery 1995:36 ,No 2;239-245 .

25- Wesley A.King et al
Venous rupture during transvenous approach to a carotid cavernous fistula.
J Neurosurg 1989 ,71 ; 133-137.

26- I Wanke, A Doerfler, D Stolke, M Forsting
Carotid cavernous fistula due to a ruptured intracavernous aneurysm of the internal carotid artery: treatment with selective endovascular occlusion of the aneurysm Trans-arterial.
J Neurology Neurosurgery Psychiatry ,2001; 71:784-787 .

27- Goto K, Hieshima GB ,Higashida RT ,Halbach VV , Bentson JR , Mehringer CM , Pribram HF
Treatment of direct Carotico cavernous sinus fistulae – Various therapeutic approaches and results in 148 cases
Acta Radiologica (suppl) 1986 ; 369 :576-579 .

27a- W.J. van Rooija, M. Sluzewskia and G.N. Beute
Ruptured Cavernous Sinus Aneurysms Causing Carotid Cavernous Fistula: Incidence, Clinical Presentation, Treatment, and Outcome
American Journal of Neuroradiology 27:185-189, January 2006

28- Fernando Vinuela, M.D., Allan J. Fox, M.D., Gerard M. Debrun, M.D., Sydney J. Peerless, M.D., and Charles G. Drake, M.D
Spontaneous carotid-cavernous fistulas: clinical, radiological, and therapeutic considerations.
JNeurosurgery 1984; vol 60 , May :976- 984

29- Voigt K , Sauerm , Dichgans J :
Spontaneous occlusion of a bilateral Carotico cavernous fistula studied by carotid angiography .
Neuroradiology 1971 ,2: 207-211 .

30- Seeger JF ,Gabrielsen TO, Giannotta S et al
Carotico cavernous sinus fistulas and venous thrombosis
AJNR , 1980 ;1: 141-148 .

31- M Theaudin, J.P. Saint-Maurice, R Chapot, K Vahedi, M Mazighi, C Vignal, G Saliou, C Stapf, M.G. Bousser, E Houdart
Diagnosis and treatment of dural carotid-cavernous fistulas: a consecutive series of 27 patients.
J Neurology Neurosurgery Psychiatry ,2007; 78:174-179 .

32- Tomio Ohta, Shuro Nishimura, Haruhiko Kikuchi, and Mitsuo Toyama,
Closure of carotid-cavernous fistula with polyurethane foam embolus.
J Neurosurgery ,1973 ,vol 38: Jan ;107- 112 .

33- S. Mullan
Carotid cavernous aneurysms and fistulae.
J Neurosurgery ,1974 :41:657-670.

34- Gerard M. Debrun, M.D., Fernando Vinuela, M.D., Allan J. Fox, M.D., Kenneth R. Davis, M.D., and Hyo S. Ahn. M.D.
Indications for Treatment and Classification of 132 Carotid-Cavernous Fistulas.
J Neurosurgery ,1988 ,22 : 285-289 .

35- D. Lo, J.N. Vallee, A. Bitar, R. Guillevin, L. Lejean, R. Van Effenterre and J. Chiras.
Endovascular management of carotid-cavernous fistula combined with ipsilateral internal carotid artery occlusion due to gunshot: contra-laternal arterial approach.
Acta Neurichirugica (Wien),2004,146:403-406.

36- Van V. Halbach, Randall T. Higashida, Stanley L. Barnwell, Christopher F. Dowd, Grant B. Hieshima
Transarterial platinum coil embolization of carotid-cavernous fistulas.
AJNR , 1991,12,429-433 .

37- Hosobuchi Y.
Carotid cavernous fistula :In Wilson C,Stein BM,eds,Intracranial arteriovenous malformations .
Baltimore: Williams and Wilkins 1984:246-258.

38- Guido Guglielmi, Fernando Vinuela, Francesco Briganti, Gary Duckwiler, Carotid-Cavernous Fistula caused by a ruptured intracavernous aneurysm: endovascular treatment by electrothrombosis with detachable coils. *Neurosurgery 1992;31:591-597 .*

39- G Bavinszki ,M Killer et al
Treatment of post-traumatic carotico-cavernous fistulae using electrolytically detachable coils: technical aspects and preliminary experience.
Neuroradiology 1995 ,39 :81-85.

40- G. Bavinzski, M. Killer, A. Gruber, B. Richling
Treatment of post-traumatic carotico-cavernous fistulae using electrolytically detachable coils: technical aspects and preliminary experience.
Neuroradiology 1997 ,39:81-85 .

41- Van V. Halbach, Randall T. Higashida, Grant B. Hieshima, Carl W. Hardin, Peter J. Yang
Transvenous embolization of direct carotid cavernous fistulas.
AJNR 1988 ;9:741-747.

42- G. Benndorf, A. Bender, R. Lehmann and W. Lanksch
Transvenous occlusion of dural cavernous sinus fistulas through the thrombosed inferior petrosal sinus: report of four cases and review of the literature.
Surgical Neurology, 2000 ,54: 42-54 .

43- Yamashita K, Taki W, Nishi S, Sadato A , Nakhara I ,Kikuchi H ,Yonekawa Y
Transvenous embolisation of dural Carotico cavernous fistulae : Technical considerations
Neuroradiology 1993 ;35:475-479 .

44- Jiao Derang, Huang Ying, Sun Reifa, Wei Qiming, Fan Yimu, Sung Guoxiang, Cui Shimin, Xiao Lihua, Wang Shi and Chen Zunhua .
Treatment of carotid-cavernous sinus fistulas retrograde via the superior ophthalmic vein (SOV).
Surgical neurology ,1999 ;52:286-293.

45- Eskridge JM ,
Interventional neuroradiology
Radiology, 1989 ;172 :991-1006 .

46- Ulfacker R ,Lima S , Rebas G , Piske RL,
Carotid cavernous fistulas : Embolisation through the superior ophthalmic vein approach
Radiology 1986 ;159 :175-179 .

47- Miller NR ,Monsein LH, Debrun GM , Tamorgo KJ , Nauta HJW
Treatment of carotid cavernous sinus fistulas using a the superior ophthalmic vein approach
J Neurosurgery 1995 ; 83 :838-842 .

48- Simon C.H. Yu, Harold K.M. Cheng, George K.C. Wong, Chi M. Chan, , James Y.L. Cheung, Wai S. Poon .
Transvenous embolization of dural carotid-cavernous fistulae with transfacial catheterization through the superior ophthalmic vein.
Mention journal

49- G. Benndorf, A. Bender, A. Campi, H. Menneking, W.R. Lanksch.
Treatment of cavernous sinus dural arteriovenous fistula by deep orbital puncture of the superior ophthalmic vein.
Neuroradiology , 2000 , October ,499 -502 .

50- C. Venturi, S. Bracco, A. Cerase, P. Gennari, F. Lore, E. Polito, A.E. Casasco .
Endovascular treatment of a cavernous sinus dural arteriovenous fistula by transvenous embolisation through the superior ophthalmic vein via cannulation of a frontal vein.
Neuroradiology 2003;45:574-578 .

51- Richard J. Bellon, Amon Y. Liu, John R. Adler, Jr. and Alexander M. Norbash,
Percutaneous transfemoral embolization of an indirect carotid-cavernous fistula with cortical venous access to the cavernous sinus.
J Neurosurgery 1999 ; 90: 959-963 .

52- Van V. Halbach, Grant B. Hieshima, Randall T. Higashida, Murray Reicher
Carotid cavernous fistulae: indications for urgent treatment.
AJR , 1987 ;419:587-593.

53- Van V Halbach , Randall T Higsahida , Grant B. Hieshima ,Murray Reicher, David Norman , T H Newton
Dural fistulas involving the cavernous sinus: results of treatment in 30 patients.
Radiology 1987 ;163 :437-442 .

54- Charles G.H. West,
Bilateral carotid-cavernous fistulaeA review
Surgical Neurology vol 13 , 1980 , 85 – 90.

55- A.M. vd Vliet, H.T. Rwiza, H.O.M. Thijssen, H.F.C.M. Brands, J.L. Merx and A. Keyser
Bilateral direct carotid-cavernous fistulas of traumatic and spontaneous origin: two case reports.
Neuroradiology ,1987 ;29 :565-569.

56- S. Kumar, R.V. Phadke, B. Mazumdar, S. Roy and R.B. Gujral
Double traumatic caroticocavernous fistula and its treatment by detachable balloons.
Neuroradiology 1992 ;34:532-533.

57- Gary Redekop, Thomas Marotta, and Alain Weill,
Treatment of traumatic aneurysms and arteriovenous fistulas of the skull base by using endovascular stents.
J Neurosurgery ,2001 ;95 :412-419.

58- Anoop Madan, Asim Mujic, Katie Daniels, Andrew Hunn, John Liddell, and Jeffrey V. Rosenfeld.
Traumatic carotid artery – cavernous sinus fistula treated with a covered stent.
L Neurosurgery ,2006 ;104 : 969-973 .

59- Stuart C. Coley Hament Pandya, Tim J. Hodgson, Martin A. Jeffree and Neil P. Deasy
Endovascular Trapping of Traumatic Carotid-Cavernous Fistulae
American Journal of Neuroradiology 24:1785-1788, October 2003.

60- Gossman MD, Berlin AJ, Weinstein MA, et al.
Spontaneous direct carotid-cavernous fistula in childhood.
Ophthalmology Plastic Reconstructive Surgery 1993; 9 :62 –65.

61- Ansaar T. Rai, Jennifer A. Sivak-Callcott, Chris Larzo and Gary D. Marano
Direct Carotid Cavernous Fistula in Infancy: Presentation and Treatment
American Journal of Neuroradiology 25:1083-1085, June-July 2004.

62- Raymond J, Hardy J, Czepko R, Roy D.
Arterial injuries in transsphenoidal surgery for pituitary adenoma; the role of angiography and endovascular treatment.
AJNR 1997; 18 :655 –665.

63- Naci Kocer, Osman Kizilkilic, Sait Albayram, Ibrahim Adaletli, Fatih Kantarci and Civan Islak
Treatment of Iatrogenic Internal Carotid Artery Laceration and Carotid Cavernous Fistula with Endovascular Stent-Graft Placement
American Journal of Neuroradiology 23:442-446, March 2002.

64- Pigott TJ, Holland IM, Punt JA.
Carotico-cavernous fistula after trans-sphenoidal hypophysectomy.
Br J Neurosurg1989; 3 :613 –616.

65- Ahuja A, Guterman LR, Hopkins LN.
Carotid cavernous fistula and false aneurysm of the cavernous carotid artery: complications of transsphenoidal surgery.
Neurosurgery1992; 31 :774 –778.

66- van Meckeren J.
Heel-en-geneekonstige..
Amesterdam: C. Commelijn; 1868:170–172.

67- North KN, Whiteman DAH, Pepin MG, Byers PH.
Cerebrovascular complications in Ehlers-Danlos syndrome type IV.
Annals of Neurology , 1995;38:960-964.

68- Debrun GM, Aletich VA, Miller NR, Dekeiser RJW.
Three cases of spontaneous direct carotid cavernous fistulas associated with Ehlers-Danlos syndrome type 4.
Surgical Neurology 1996;46:247-252.

69- Graf CJ.
Spontaneous carotid cavernous fistula.
Archieves of Neurology , 1965;13:662-672.

70- Halbach VV, Higashida RT, Dowd CF, Barnwell SL, Hieshema GB.
Treatment of carotid-cavernous fistulas associated with Ehlers-Danlos syndrome.
Neurosurgery 1990;26:1021-1027.

71- Freeman RK, Swegle J, Sise MJ.
The surgical complications of Ehlers-Danlos syndrome.
The American Surgeon 1996;62:869-873.

72- Schievink WI, Piepgras DG, Earnest F, Gordon H.
Spontaneous carotid-cavernous fistulae in Ehlers-Danlos syndrome type IV.
J Neurosurg 1991;74:991-998.

73- Schoolman A, Kepes JJ.
Bilateral spontaneous carotid cavernous fistulae in Ehlers-Danlos syndrome.
J Neurosurgery 1967;26:82-86.

74- Michael B. Horowitz, Phillip D. Purdy, R. James Valentine and Kevin Morrill
Remote Vascular Catastrophes after Neurovascular Interventional Therapy for Type 4 Ehlers-Danlos Syndrome
American Journal of Neuroradiology 21:974-976 (5 2000).

75- Higashida RT, Halbach VV, Tsai FY, et al.
Interventional neurovascular treatment of traumatic and vertebral lesions: results in 234 cases.
AJR Am J Roentgenol 1989;153:577–582.

76- The EC/IC Bypass Study Group.
Failure of extracranial-intracranial arterial bypass to reduce the risk of ischemic stroke: results of an international randomized trial.
*N Engl J Med*1985; 313:1191 –1200.

77- Macdonald S, Gan J, McKay AJ, Edwards RD.
Endovascular treatment of acute carotid blow-out syndrome.
*J Vasc Interv Radiol*2000; 11 :1184 –1188.

78- Lee CY, Yim MB, Kim IM, Son EI, Kim DW.
Traumatic aneurysm of the supraclinoid internal carotid artery and an associated carotid-cavernous fistula: vascular reconstruction performed using intravascular implantation of stents and coils—case report.
J Neurosurg 2004; 100:115–119.

79- Ahn JY, Lee BH, Joo JY.
Stent-assisted Guglielmi detachable coils embolization for the treatment of a traumatic carotid cavernous fistula.
J Clin Neurosci 2003;10:96–98.

80- Men S, Ozturk H, Hekimoglu B.
Traumatic carotid-cavernous fistula treated by combined transarterial and transvenous coil embolization and associated cavernous internal carotid artery dissection treated with stent placement: case report.
J Neurosurg 2003;99:584–586.

81- Fanny E. Morón, Richard P. Klucznik, Michel E. Mawad and Charles M. Strother
Endovascular Treatment of High-Flow Carotid Cavernous Fistulas by Stent-Assisted Coil Placement
American Journal of Neuroradiology 26:1399-1404, June-July 2005.

82- R.Fox, F.M.Pope ,P.Narcisi
Spontaneous Carotico- cavernous fistula in Ehler's Danlos syndrome
J Neurology Neurosurgery Psychiatry ,1998;51:984-986.

83- Gerald M.Debrun,Victor A. Aletich ,Neil R.Miller
Three Cases Of Spontaneous Direct Caroticocavernous Fistulas Associated With Ehler's Danlos Syndrome IV
Surgical Neurology 1996;46:247-52.

84- Andrew A. Kanner , Shimon Maimon, Zvi H.Rappaport
Treatment Of Spontaneous Caroticocavernous Fistula In Ehler's Danlos Syndrome By Transvenous Occlusion With Guglielmi Detachable Coils .
J Neurosurg , 2000 ,93;689-692 .

85- Hollands JK, Santarius T, Kirkpatrick PJ, Higgins JN
Treatment of a direct carotid-cavernous fistula in a patient with type IV Ehlers-Danlos syndrome: a novel approach.
Neuroradiology. 2006 May 6.

86- Cikrit DF, Miles IH, Silver D.
Spontaneous arterial perforation: the Ehlers-Danlos spector.
J Vasc Surg 1987;5:248-25.

87- Bergqvist D.
Ehlers-Danlos type IV syndrome. A review from a vascular point of view.
Eur J Surg 1996;16:163-170.

ABBREVIATIONS

ABBREVIATIONS

B/L	: Bilateral
Br.Asthama	: Bronchial Asthma
C/L	: Contralateral
CAD	: Coronary Artery Disease
CCF	: Carotico-cavernous Fistula
CS	: Cavernous Sinus
CVR	: Cortical venous reflux
DCCF	: Dural Carotico Cavernous Fistula
DM	: Diabetes Mellitus
DOV	: Diminution of vision
ECA	: External Carotid artery
EDS	: Ehler's Danlos Syndrome
EOM	: Extraocular Movements
HT	: Hypertension
I/L	: Ipsilateral
ICA	: Internal Carotid Artery
ICH	: Intracranial Hemorrhage
ICU	: Intensive Care Unit
IJV	: Internal Jugular Vein
IMA	: Internal Maxillary Artery
IOV	: Inferior Ophthalmic vein
IPS	: Inferior Petrosal Sinus
ISIR	: Imaging Sciences and Interventional Radiology
MI	: Myocardial Infarction
MMA	: Middle Meningeal Artery
SCTIMST	: Sree Chitra Tirunal Institute of Medical Sciences & Technology

SOV	: Superior Opthalmic vein
T.B.	: Tuberculosis
U/L	: Unilateral
VA	: Vertebral Artery

MASTER CHART

S/N	DATE	N	A/S	H/NO	T	ET	S/P	O	T	LO C	F	S/E	Du	HA	Vo	P	D/V	Di	R	Ve	Ti	Wa	Af	O/E	P	R	C	6
1	6/5/1989	RS	33/M	8902998	A	T	LT	G	Y	N	N	Y	6M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y	
2	4/8/1989	J	24/M	8905702	A	T	RT	G	Y	N	N	Y	1.5Y	N	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y	
3	19/1/1990	AK	18/M	8908370	A	T	LT	G	Y	Y	N	Y	2Y	Y	N	N	Y	Y	Y	N	Y	N	N		Y	Y	Y	
4	30/4/1990	FK	17 / F	9003386	A	T	LT	G	Y	Y	Y	Y	2Y	N	N	N	Y	Y	N	N	Y	N	N		Y	Y	Y	
5	21/5/1990	R	9/M	9003963	A	T	LT	G	Y	Y	Y	Y	4M	Y	N	N	N	Y	Y	N	N	N	N		Y	Y	Y	
6	13/5/1991	B	32/F	9103845	A	T	RT	G	Y	N	N	N	3M	N	N	N	N	Y	N	N	N	N	N		Y	N	N	
7	16/8/1991	G	27/M	9106980	A	T	LT	G	Y	Y	N	Y	7M	N	N	N	Y	N	N	N	N	N	N		Y	N	Y	
8	16/8/1991	S	45/M	9106435	A	T	B/L	G	Y	Y	N	Y	45D	Y	N	N	N	Y	N	N	N	N	N		Y	Y	Y	
9	19/3/1992	A	35/F	9201993	A	T	RT	G	Y	Y	Y	Y	1M	N	N	N	VL	N	Y	N	N	N	N		N	Y	Y	
10	20/3/1992	S	22/M	9202064	A	T	LT	G	Y	Y	N	Y	6M	N	N	N	N	Y	Y	N	N	N	N		N	N	Y	
11	18/4/1992	D	62/F	9202656	A	T	RT	G	Y	N	N	Y	3M	Y	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y	
12	30/10/1992	A	53/M	9207733	A	T	RT	G	Y	Y	Y	Y	4W	N	N	N	N	N	Y	N	N	N	N		Y	N	N	
13	29/1/1992	K	65/F	9300338	A	T	RT	G	Y	N	N	Y	1M	Y	N	N	N	N	N	N	N	N	N		Y	Y	Y	
14	21/4/1992	R	28/F	9202322	A	S	LT	G	N	N	N	Y	1M	Y	N	N	N	N	Y	N	N	N	SS		Y	Y	Y	
15	4/8/1992	SF	48/ M	8808018	A	T	LT	G	Y	N	N	Y	2W	Y	Y	N	Y	N	N	N	N	N	N		Y	Y	Y	
16	21/2/1994	RL	27/F	9401460	A	S	RT	G	N	N	N	Y	4M	N	N	N	N	N	Y	N	N	N	N		Y	Y	N	
17	26/10/1994	R	36/M	9408206	A	T	RT	A	Y	Y	Y	Y	1M	N	N	N	Y	N	Y	N	N	N	Pus		Y	Y	Y	
18	20/12/1994	R	39/M	9406481	A	S	LT	G	N	N	N	Y	10M	N	N	Y	N	Y	Y	N	N	Y	N		Y	Y	Y	
19	8/4/1995	S	48/M	9502601	A	T	RT	A	Y	N	N	Y	2M	N	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y	
20	26/10/1995	MC	39/F	9503985	A	S	RT	A	N	N	N	Y	9M	Y	N	N	N	N	N	N	Y	N	N		Y	Y	Y	
21	2/12/1995	SS	53/F	9509517	A	S	LT	G	N	N	N	N	3M	Y	N	N	Y	Y	N	N	Y	N	N		N	N	N	

MN P	ON P	B	AB	Di	VA RE	VA LE	VF	OA	N/E	L/G A	S/A	S/R	SO V	IPS	AVD	R/E	R ECA	R ICA	MAT R	E/O R	L ECA	L ICA	MAT L	E/O L	E1	FU	C
N	N	Y	Y	Y	N	N	N	N	1	LA	LT	C3	Y	Y	N	A	N	N	N	N	N	N	1B	NT	NT	T	
N	N	Y	Y	Y	N	N	N	N	1	LA	RT	C3	Y	Y	N	A	N	N	2B	NT	N	N	N	N	NT	T	
N	N	Y	N	Y	N	C	PC	N	3	LA	LT	C3	Y	Y	N	A	N	N	N	N	N	N	1B	NIL	NIL	MA, T	
Y	N	Y	N	Y	N	C	N	N	1	LA	LT	C4	Y	Y	N	A	N	N	N	N	N	N	1B	NT	NT	T	
N	N	Y	Y	Y	N	N	N	N	1	LA	LT	C4	Y	Y	N	A	N	N	N	N	N	N	1B	T	T	T	
N	N	N	N	Y	N	N	N	N	1	LA	RT	C3-C4	Y	N	IOV	A	N	N	1B	T	N	N	N	N	T	T	
N	N	Y	N	N	N	N	N	N	1	LA	LT	C4	Y	N	N	A	N	N	N	N	N	N	1B	T	T	T	
N	Y	Y	N	Y	N	N	N	N	1	LA	RT	C3	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T	
Y	Y	N	N	N	NIL	NIL	NIL	N	1	LA	RT	C3	N	N	Y	A	N	N	1B	T	N	N	N	N	T	T	
N	N	Y	N	Y	N	N	N	N	3	LA	LT	C3-C4	N	Y	N	A	N	N	N	N	N	N	1B	NIL	Res	MA T	
N	N	Y	N	Y	N	N	N	N	1	LA	RT	C4-C5	Y	N	CVR	A	N	N	1B	T	N	N	N	N	T	T	
N	Y	N	N	N	C	N	C	Y	1	LA	RT	C4	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T	
Y	N	Y	N	Y	N	N	N	N	1	LA	RT	C3-C4	Y	N	N	A	N	N	N	N	N	N	N	N	T	Tr	
N	N	Y	Y	N	N	N	N	N	N	LA	LT	C3	Y	N	N	N	N	N	N	N	N	N	N	N	D	D	
N	N	Y	Y	N	N	C	N	Y	N	LA	LT	C3-C4	Y	N	N	A	N	N	N	N	N	N	1B	T	T	T	
N	N	N	N	N	N	N	N	N	1	LA	RT	C3	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T	
N	N	N	N	T0	C	C	N	N	1	LA	RT	C3-C4	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T	
N	N	N	N	N	N	N	N	N	1	LA	LT	C4-C5	Y	Y	N	A	N	N	2B	T	N	N	N	N	T	T	
N	T0	N	Y	T0	C	C	PC	N	1	LA	RT	C3-C4	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T	
N	N	Y	N	N	N	N	N	N	1	LA	RT	C5	Y	N	N	X	N	N	1B	T	N	N	N	N	T	T	
N	N	N	Y	Y	N	N	N	N	1	LA	LT	C3-C4	Y	Y	N	Sx	N	N	N	N	N	N	N	N	T	Tr	

22	29/2/1996	AKD	32/M	9601561	A	T	RT	A	Y	N	N	Y	10 D	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y
23	8/7/1996	S	9/F	9603816	A	S	LT	G	N	N	N	Y	2Y	N	N	N	N	Y	N	N	N	N	N		Y	Y	N
24	12/8/1996	N	48/F	9606004	A	T	LT	G	Y	N	Y	Y	2W	N	N	N	N	N	Y	N	N	N	N		Y	Y	N
25	4/7/1997	VJ	41/M	9704913	A	T	LT	G	Y	Y	N	Y	2M	N	N	N	N	Y	Y	Y	N	N	N		Y	Y	Y
26	12/12/1997	S	23/M	9710038	A	T	RT	G	Y	Y	N	Y	9M	N	N	N	Y	N	Y	N	N	N	N		Y	Y	Y
27	15/1/1998	J	6/M	9800448	A	T	LT	G	Y	N	N	Y	2M	N	N	N	N	N	N	N	N	N	N		Y	N	N
28	9/3/1998	PP	42/M	9801750	A	T	RT	G	Y	N	N	Y	3M	N	N	N	N	Y	Y	N	N	Y	N		Y	Y	Y
29	29/5/1998	J	39/M	9804199	A	T	LT	G	Y	Y	N	Y	2M	N	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y
30	3/9/1998	PR	22/M	9807159	A	T	RT	G	Y	N	Y	Y	1 M	N	N	N	VL	N	Y	N	N	N	N		Y	Y	Y
31	4/11/1998	V	19/M	9807701	A	T	LT	G	Y	N	N	Y	3M	N	N	N	N	N	Y	N	N	N	N		Y	Y	N
32	3/12/1998	UK	27/M	9808347	A	T	RT	G	Y	N	Y	Y	2.5 M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	N
33	1/3/1999	KM	20/M	9809527	A	T	RT	G	Y	Y	Y	Y	25D	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y
34	4/3/1999	NS	44/F	9901353	A	S	B/L	G	N	N	N	Y	1.5Y	N	N	N	Y	N	N	N	N	N	N		Y	N	N
35	4/6/1999	GM	56/M	9904008	A	T	RT	G	Y	Y	Y	N	1W	N	N	N	N	N	N	N	Y	N	N		Y	Y	Y
36	21/6/1999	S	40/F	9904284	A	T	LT	G	N	N	N	N	3M	N	N	N	Y	Y	Y	N	N	N	N		Y	Y	Y
37	26/7/1999	S	23/M	9907440	A	T	RT	G	Y	Y	Y	Y	3M	N	N	N	VL	N	Y	N	N	N	N		Y	Y	Y
38	31/8/1999	S	50/F	9906163	A	T	RT	G	Y	N	N	Y	2M	N	N	N	N	Y	Y	N	Y	N	Pt		Y	Y	Y
39	28/2/2000	J	26/M	181458	A	T	LT	G	Y	Y	N	Y	1.5M	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y
40	22/7/2000	DP	19/F	185223	A	S	LT	G	N	N	N	Y	3M	N	N	N	N	N	Y	N	N	N	EDS		Y	Y	Y
41	2/8/2001	MK	27/M	183862	A	T	LT	G	Y	N	N	Y	4 M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
42	21/8/2001	S	42/M	195317	A	T	RT	G	Y	Y	N	Y	4M	N	N	N	N	N	Y	N	Y	N	N		Y	N	Y
43	20/9/2001	B	32/M	193773	A	T	LT	G	Y	Y	Y	Y	15 D	N	N	N	Y	N	N	N	N	N	N		Y	Y	Y

N	N	Y	N	N	N	N	N	N	1	LA	RT	C3	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T
N	N	Y	N	Y	N	N	N	N	1	LA	LT	C3	Y	N	N	A	N	N	2B	T	N	N	N	N	T	T
N	N	N	N	N	N	N	N	N	2	LA	LT	C3	Y	Y	N	A	N	N	N	N	N	N	N	N	E0	T
N	Y	Y	Y	Y	N	N	N	N	1	LA	LT	C3	Y	Y	N	A	N	N	N	N	N	N	2B	T	T	T
N	N	Y	N	N	N	N	NIL	Y	1	LA	RT	C2-C3	Y	Y	N	A	N	N	4B	NT	N	N	N	N	NT	T
N	N	Y	N	N	N	N	N	N	1	GA	LT	C3	Y	Y	N	A	N	N	N	N	N	N	1B	T	T	T
N	N	Y	N	Y	N	N	N	N	1	LA	RT	C2	Y	Y	N	A	N	N	2B	T	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	2	LA	LT	C3-C4	Y	Y	N	A	N	N	N	N	N	N	2B	MR	MR	T
Y	N	N	N	N	C	N	N	Y	1	GA	RT	C3	Y	N	N	A	N	N	Co	Mo R	N	N	N	MoR	Res	T
N	N	Y	N	Y	N	N	N	N	1	LA	LT	C4	Y	Y	N	A	N	N	N	N	N	Y	Co	MR	MA	T
N	N	N	Y	Y	N	N	N	N	1	LA	RT	C4-C5	N	N	CVR	A	N	N	1B	NT	N	N	N	N	NT	T
N	N	Y	N	N	C	N	C	N	1	LA	RT	C4	Y	Y	IOV, CVR	A	N	N	2B	T	N	N	N	N	MA	T
N	N	Y	N	Y	C	C	PC	Y	1	LA	RT	C3	Y	Y	N	A	N	N	3B	NT	N	N	N	N	NT	T
Y	N	Y	Y	Y	N	N	N	N	1	LA	LT	C4-C5	Y	Y	N	A	N	N	N	N	N	N	1B	T	T	T
Y	N	Y	Y	Y	C	C	C	N	2	LA	LT	C3-C4	Y	Y	N	A	N	N	1B	T	N	N	N	N	T	Rec
N	N	Y	N	VL	NIL	N	NIL	Y	1	LA	RT	C4	Y	Y	DVD	A	N	N	1B	T	N	N	N	N	T	T
N	Y	Y	N	Y	C	C	C	N	1	LA	RT	C2	Y	N	N	A	N	N	1B	T	N	N	N	N	T	T
N	7	N	N	N	N	N	N	N	1	LA	LT	C3	Y	N	N	A	N	N	N	N	N	N	B	N	T	Tr
N	N	Y	Y	Y	N	N	N	N	N	LA	LT	C4-C5	Y	Y	N	N	N	N	N	N	N	N	X	N	D	D
N	N	Y	N	Y	N	N	N	N	3	LA	LT	C3-C4	Y	Y	N	A	N	N	N	N	N	Y	1B,Gf	N	NT	Rec, MA
N	N	Y	N	Y	N	N	N	N	1	LA	RT	C3	Y	Y	N	A	N	N	1B	T	N	N	N	N	T	T
N	N	Y	N	Y	N	C	NIL	Y	1	LA	LT	C3	Y	N	N	A	N	N	N	N	N	Y	2B	T	T	T

44	24/10/2001	V	20/M	197506	A	T	LT	A	Y	Y	Y	Y	1M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y	[illegible]
45	17/4/2002	UB	36/F	201697	A	S	LT	G	N	N	N	Y	10 M	N	N	Y	N	N	N	N	N	N	N		Y	Y	Y	
46	14/8/2002	BT	34/M	206694	A	T	LT	G	Y	Y	Y	N	4Y	N	N	N	N	N	Y	N	N	N	Pt		Y	Y	Y	[illegible]
47	28/12/2002	KD	35/M	210976	A	T	B/L	G	Y	N	N	Y	6M	N	N	Y	N	N	Y	N	N	N	N		Y	Y	Y	[illegible]
48	18/2/2003	PS	47/M	212572	A	T	LT	G	Y	N	N	Y	4M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y	[illegible]
49	2/5/2003	S	34/F	214973	A	S	LT	G	N	N	N	Y	6M	Y	N	N	N	Y	Y	N	N	N	EDS		Y	Y	Y	
50	1/1/2004	C	60/F	222717	A	T	LT	G	Y	N	N	Y	10M	Y	N	Y	N	Y	N	N	N	Y	Pt		Y	Y	Y	
51	13/3/2004	GA	65/F	225441	A	S	RT	G	N	N	N	N	1.5 M	N	N	N	N	Y	Y	N	N	Y	Pt		N	Y	Y	
52	19/4/2004	K	23/M	226893	A	T	RT	G	Y	Y	Y	Y	3Y	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y	
53	19/5/2004	BK	33/M	221644	A	T	RT	G	Y	Y	Y	Y	7M	N	N	N	N	N	Y	N	Y	N	N		Y	Y	Y	
54	1/7/2004	D	22/M	229030	A	T	LT	A	Y	Y	Y	Y	1M	Y	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y	
55	16/10/2004	M	45/F	232231	A	S	LT	A	N	N	N	Y	1M	Y	Y	Y	N	Y	Y	N	Y	N	N		Y	Y	Y	
56	27/12/2004	SS	7/M	235212	A	T	RT	A	Y	N	N	Y	4M	N	N	N	N	N	Y	N	N	N	EDS		Y	Y	N	
57	18/2/2005	DK	41/M	237130	A	T	RT	G	Y	Y	Y	Y	2M	Y	N	N	N	Y	Y	N	N	N	N		Y	Y	Y	
58	18/3/2005	RG	44/M	238374	A	T	RT	G	Y	Y	N	Y	3M	N	N	N	N	N	Y	N	N	N	Hp		Y	Y	Y	
59	13/5/2005	RC	31/M	240244	A	T	B/L	A	Y	Y	Y	Y	1.5M	N	N	N	T0	T0	Y	N	N	N	G Pr		Y	Y	Y	
60	22/8/2005	SK	26/M	229666	A	T	RT	G	Y	Y	Y	Y	2Y	Y	N	N	Y	N	Y	N	Y	N	N		Y	Y	Y	
61	25/1/2006	RJ	28/M	249331	A	T	RT	G	Y	Y	Y	Y	1.5Y	Y	N	Y	N	N	Y	N	N	N	N		Y	Y	Y	
62	3/2/2006	SK	20/M	249787	A	T	LT	G	Y	Y	Y	Y	12 D	N	N	N	Y	Y	Y	N	N	N	N		Y	Y	Y	
63	6/2/2006	SK	35/M	249862	A	T	RT	G	Y	Y	Y	Y	1M	N	N	N	VL	N	Y	N	N	N	N		Y	Y	Y	
64	15/2/2006	JVT	27/M	250040	A	T	RT	G	Y	Y	Y	Y	1Y	N	N	N	VL	N	Y	N	N	N	N		Y	Y	Y	

N	Y	N	N	Y	N	N	N	N	2	LA	LT	C4-C5	Y	Y	CVR	A	N	N	1B, 4Co	MR	N	N	N	N	Res	Tr
N	N	Y	N	N	N	N	N	N	N	LA	LT	C2-C3	Y	N	N	A	N	N	N	N	N	Y	1B	T	T	T
N	Y	Y	Y	N	N	NIL	NIL	Y	1	LA	LT	C2-C3	N	N	Y	A	N	N	N	N	N	N	1B	NT	T	Tr
Y	N	Y	N	N	N	N	N	N	1	LA	RT	C3	Y	N	N	A	N	N	1B	T	N	N	N	T	T	T
N	N	N	N	Y	N	N	N	N	1	LA	LT	C3-C4	Y	N	CVR	A	N	N	N	N	N	N	1B	T	T	T
Y	N	Y	N	Y	N	N	N	N	1	LA	LT	C3	Y	Y	N	A	N	N	1B, 2Co	T	N	N	N	N	T	T
Y	N	Y	N	Y	N	N	PC	N	N	LA	LT	C3	Y	N	N	N	N	N	CC	N	N	N	N	N	Res	Res
Y	N	N	N	Y	N	N	N	N	1	GA	RT	C2- C3	Y	Y	N	A	N	N	11Co	T	N	N	N	N	T	Tr
Y	N	N	N	N	N	N	T0	N	1	LA	RT	C3	Y	Y	N	A	N	N	3B	T	N	N	N	N	T	T
N	N	Y	N	N	N	N	N	N	1	LA	RT	C4	Y	Y	BVR, CVR	A	N	N	2B	T	N	N	N	N	T	T
Y	N	N	N	N	N	N	N	N	1	LA	LT	LT C4	Y	Y	CVR. DVD	A	N	N	N	N	N	Y	2B	T	T	Tr
Y	N	Y	N	Y	N	N	N	N	1	LA	LT	C3	Y	Y	N	A	N	N	N	N	N	N	1B	T	T	T
N	N	N	N	N	N	N	N	N	1	LA	RT	C3-C4	Y	N	N	A	N	N	1B	T	N	N	X	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	RT	C3	Y	Y	N	A	N	N	1B	T	N	N	X	N	T	T
N	7	Y	N	N	N	N	N	N	1	LA	RT	C3-C4	Y	N	N	A	N	N	16Co	T	N	N	X	N	NT	Tr
N	Y	Y	N	T0	T0	T0	T0	N	1	LA	B/L	C2	Y	N	IOV	A&V	N	N	1B	MR	N	Y	1B	T	Res	MA, T
Y	N	Y	N	Y	C	N	NIL	Y	2	LA	RT	C3	Y	N	N	A	N	N	1B	MR	N	N	N	N	NT	T
N	N	Y	N	N	N	N	N	N	1	LA	RT	C4-C5	Y	N	CVR	A	N	N	2B	T	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	LT	C3	Y	N	IOV	A	N	N	N	N	N	N	6c	T	T	Tr
Y	7	N	Y	N	C	N	C	N	1	LA	RT	C4	Y	Y	CVR	A	N	N	2B	T	N	N	N	N	T	T
N	7	N	N	N	NIL	N	N	N	2	GA	RT	C3- C4	Y	Y	N	A	N	N	B.Co	N	N	N	N	N	RES	Tr

65	19/5/2006	S	14/M	249085	A	T	RT	G	Y	Y	N	Y	2M	N	N	N	N	Y	N	N	N	N	N		Y	N	N
66	20/5/2006	PK	24/M	253279	A	S	RT	G	X	N	N	Y	5 M	N	N	N	N	N	Y	N	N	N	EDS		Y	Y	Y
67	30/5/2006	PRT	25/M	253696	A	T	RT	G	Y	N	N	Y	4M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
68	4/8/2006	B	29/M	255925	A	T	RT	G	Y	N	N	Y	7 M	N	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y
69	1/4/2007	R	38/M	265334	A	T	RT	G	Y	N	N	Y	18M	N	N	N	N	N	Y	N	N	Y	N		Y	Y	Y
70	7/8/2000	S	28/M	184707	A	Ia	RE	A	Y	N	N	Y	1D	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y
71	7/3/1991	AJ	55/F	8909853	B	S	RT	G	N	N	N	Y	3M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
72	8/2/1999	PJ	40/M	9808352	B	S	RT	A	N	N	N	Y	4M	N	N	N	Y	N	Y	N	N	N	N		Y	Y	Y
73	20/5/2002	AB	51/M	202819	B	S	RT	G	N	N	N	Y	6M	N	N	Y	N	Y	Y	N	N	N	N		Y	Y	Y
74	29/4/2003	S	30/F	125829	B	S	RT	G	N	N	N	Y	1M	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y
75	20/6/2003	AK	47/M	216654	B	S	LT	G	N	N	N	Y	2M	Y	N	N	N	Y	Y	N	N	Y	N		Y	Y	Y
76	19/1/2004	SD	60/F	223576	B	S	LT	A	N	N	N	Y	10 D	Y	N	Y	Y	Y	Y	N	N	N	N		Y	Y	Y
77	14/3/2006	P	67/M	251130	B	S	LT	G	N	N	N	Y	2M	N	N	N	N	N	Y	N	N	N	N		Y	Y	N
78	29/11/2006	S	37/M	260366	B	PT	RT	G	Y	N	N	Y	17 M	Y	N	N	N	Y	Y	N	Y	Y	N		Y	Y	Y
79	3/4/2007	A	54/F	265094	B	S	RT	G	N	N	N	Y	3M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
80	19/6/1992	T	24/F	9102474	C	S	LT	G	N	N	N	Y	1.5Y	N	N	N	N	N	Y	N	Y	N	N		Y	Y	Y
81	31/11/1994	M	59/M	9309076	C	S	RT	G	N	N	N	Y	6YR	N	N	Y	Y	N	Y	N	N	N	N		Y	Y	Y
82	5/7/1997	HK	43/F	9503768	C	S	B/L	G	N	N	N	Y	3Y	Y	Y	N	N	Y	Y	N	Y	N	N		Y	Y	Y
83	20/11/2002	DSH	57/F	209222	C	S	RT	G	N	N	N	Y	2M	N	N	Y	N	Y	Y	N	Y	Y	N		Y	Y	Y
84	29/11/2002	RV	60/F	210164	C	S	LT	G	N	N	N	Y	1.5Y	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
85	8/10/2003	RTS	46/F	219745	C	S	LT	G	N	N	N	Y	1 M	N	N	N	N	Y	Y	N	N	N	PP		Y	Y	Y
86	4/12/2006	Md H	72/M	260238	C	S	LT	G	N	N	N	Y	5M	N	N	Y	Y	Y	Y	N	N	N	N		Y	Y	Y

N	N	N	N	Y	N	N	N	N	1	LA	RT	C4-C5	Y	N	CVR	A	N	N	1B	T	N	N	N	N	NT	Res
N	N	Y	N	N	N	N	N	N	1	LA	RT	C3	Y	N	CVR	N	N	N	2B	N	N	N	N	N	D	X
N	N	N	N	Y	N	N	N	N	1	LA	RT	C4-C5	Y	Y	CVR	A & V	N	N	B,Co	T	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	RT	C4-C5	N	Y	CVR, DVD	A	N	N	1B	T	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	RT	C 5	Y	X	SPS	A	N	N	4B	T	N	N	N	N	T	T
N	N	Y	N	N	N	N	N	N	2	LA	RT	C3	Y	N	N	A	N	N	2B	T	N	N	N	N	Rec	Tr
N	N	N	N	Y	N	N	N	N	N	N	RT	N	N	N	N	N	N	Y	CC	N	N	N	N	N	T	T
N	N	N	N	N	N	N	N	N	N	N	RT	N	Y	N	N	N	N	Y	CC	N	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	N	N	RT	N	Y	N	N	N	N	Y	CC	N	N	N	N	N	T	T
N	N	N	N	N	N	N	N	N	N	N	RT	N	Y	N	N	N	N	Y	CC	N	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	N	N	B/L	N	Y	N	N	N	N	Y	CC	N	N	Y	CC	N	MR	MR
Y	N	Y	N	Y	N	N	N	N	N	N	LT	N	Y	Y	N	N	N	N	N	N	N	Y	B	N	T	T
N	N	N	N	N	N	N	N	N	N	N	B/L	N	Y	N	N	N	N	Y	CC	N	N	Y	CC	N	T	T
N	N	Y	N	Y	N	N	N	N	1	LA	RT	N	Y	Y	N	V	N	Y	Co	T	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	A	X	RT	N	Y	N	N	X	N	Y	N	N	N	N	N	N	S	S
N	N	Y	N	N	N	N	N	N	1	LA	LT	N	N	N	N	A	N	N	N	N	2	N	B	T	T	T
N	N	Y	N	N	N	N	C	N	1	LA	RT	N	Y	N	N	A	2	N	Hg	NT	N	N	N	N	NT	T
N	N	N	N	Y	N	N	N	N	2	LA	B/L	N	N	N	N	A	1	N	Hg	50%	1,3	N	Hg	NT	NT	T
N	N	Y	N	Y	C	C	N	N	1	LA	RT	N	Y	N	N	V	1,3	N	Co	T	N	N	Hg	T	T	T
N	N	Y	N	Y	N	N	C	N	1	LA	LT	N	Y	N	N	A	N	N	N	N	1	N	PVA	NT	NT	T
N	N	N	N	Y	N	N	N	N	1	LA	LT	N	Y	N	N	A	N	N	N	N	1	N	PVA	Res	MR	R
N	N	Y	N	Y	N	N	N	N	1	LA	LT	N	N	N	N	A	N	N	N	N	1,2,3	N	PVA+ Co	MR	MR	T

87	30/7/2007	SKC	29/F	268914	C	S	LT	G	N	N	N	X	15D	Y	N	Y	Y	Y	Y	N	N	N	N		N	Y	N
88	18/1/1993	K	52/M	9300336	D	S	LT	G	N	N	N	Y	2W	Y	Y	N	N	Y	N	N	N	N	N		Y	Y	Y
89	28/11/1995	RD	42/M	9509106	D	S	LT	G	N	N	N	Y	4M	N	N	N	Y	N	N	N	N	N	Pt		Y	Y	Y
90	13/12/1995	RES	28/M	9508993	D	S	LT	G	N	N	N	Y	1M	Y	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
91	5/11/1996	RV	39/M	9608488	D	S	RT	G	N	N	N	Y	3M	N	N	Y	Y	Y	Y	N	Y	N	N		Y	Y	Y
92	20/1/1997	RS	29/M	9700565	D	S	RT	G	N	N	N	Y	1M	N	N	N	Y	N	Y	N	N	N	N		Y	Y	Y
93	21/10/1997	VC	51/M	9708189	D	S	RT	G	N	N	N	Y	6M	N	N	N	Y	Y	Y	N	N	N	N		Y	N	Y
94	31/3/1998	BB	49/M	9802523	D	S	RT	G	N	N	N	X	1M	Y	N	N	Y	N	N	N	N	N	P		N	N	N
95	26/11/1998	J	56/F	9809315	D	S	RT	G	N	N	N	Y	3M	Y	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
96	8/11/1999	T	51/M	9901092	D	S	LT	G	N	N	N	X	15D	N	N	Y	N	N	Y	N	N	N	N		N	Y	N
97	12/3/2000	K	55/F	181527	D	S	LT	G	N	N	N	Y	3M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
98	31/5/2000	M	22/M	183819	D	T	LT	A	Y	N	N	Y	1M	Y	N	Y	C	N	Y	N	N	N	N		Y	Y	Y
99	31/8/2000	J	51/M	185601	D	S	LT	G	N	N	N	Y	8M	N	N	N	N	N	Y	N	N	N	N		Y	Y	N
100	21/12/2000	R	39/M	189232	D	S	B/L	A	N	N	N	N	2 M	Y	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y
101	20/2/2002	K	37/F	200928	D	S	RT	G	N	N	N	Y	1M	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y
102	31/5/2002	SJ	55/F	204219	D	S	LT	G	N	N	N	Y	2M	Y	Y	Y	Y	Y	Y	N	N	N	N		Y	Y	Y
103	20/6/2002	N	56/F	204389	D	S	B/L	G	N	N	N	Y	2M	N	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y
104	29/10/2002	S	71/M	121759	D	S	B/L	G	N	N	N	Y	2M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y
105	23/05/2003	A	45/F	211232	D	S	LT	G	N	N	N	Y	1.5 M	N	N	N	N	Y	Y	N	N	N	W		Y	Y	Y
106	20/9/2003	S	50/F	219546	D	S	LT	A	N	N	N	Y	2M	N	N	Y	N	Y	Y	N	N	N	PEV		Y	Y	Y
107	3/2/2004	KG	70/F	9710647	D	S	RT	G	N	N	N	N	1M	N	N	Y	N	Y	Y	N	N	N	N		N	Y	N
108	23/4/2004	RJ	45/F	225747	D	T	RT	A	Y	N	N	Y	2D	Y	N	N	N	Y	Y	N	Y	N	N		Y	Y	Y

N	N	N	N	Y	N	N	N	N	1	LA	B/L	N	Y	N	N	A	1,2	N	N	N	1,2	N	PVA	NT	NT	T
N	N	N	N	Y	N	N	N	N	2	LA	B/L	N	Y	N	N	A	2	N	Hg	T	1,3	4	N	N	T	T
N	L-3	N	N	N	N	N	N	N	1	LA	LT	N	Y	N	N	A	N	N	N	1	N	4	PVA	50%	Res	T
N	R-3	N	N	Y	N	N	N	N	2	LA	RT	N	Y	N	N	A	1	4	Hg	NT	N	N	N	N	NT	T
Y	N	Y	N	Y	C	C	N	N	1	LA	B/L	N	N	N	N	A	1	4	Hg,Gf	NT	1	4	Hg,Gf	NT	NT	T
N	N	Y	N	N	N	N	N	N	2	LA	RT	N	Y	N	N	A	1	5	Hg,Gf	NT	N	N	N	N	NT	Rec, MA
N	N	N	N	Y	N	N	N	N	1	LA	B/L	N	Y	N	N	A	1	4	Hg	T	N	4	N	N	T	T
N	R-3	N	N	N	C	C	N	N	1	LA	B/L	N	N	Y	C/L	A	2,3	4	Hg	NT	1,3	4	N	N	NT	T
N	N	N	N	Y	C	C	N	N	1	LA	B/L	N	Y	N	N	A	1	4	PVA	NT	N	4	N	N	NT	T
N	N	N	N	N	N	N	N	N	1	LA	LT	N	Y	N	N	A	N	N	N	N	1,3	4	Hg	NT	NT	T
N	N	N	N	Y	N	N	N	N	1	LA	LT	N	Y	N	N	A	N	N	N	N	1	4	Bu	T	T	T
N	N	Y	N	N	N	N	C	N	3	GA	LT	N	Y	Y	DVD	A	N	N	N	N	1,2	4	Co	Res	MR	MA, T
N	N	N	N	N	N	N	N	N	N	N	B/L	N	Y	N	N	CC	1	4	CC	N	1	4	CC	N	T	Rec
Y	N	Y	N	Y	N	N	N	N	1	LA	B/L	N	N	Y	N	V	1,2,3	4	N	N	2,3	4	Co	T	T	T
N	N	N	N	N	N	N	N	N	1	LA	RT	N	Y	N	N	A	1	4	PVA	T	N	4	N	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	LT	N	Y	Y	N	A	N	4	N	N	1,3	4	Co	T	T	T
N	N	N	N	N	N	N	N	N	N	LA	B/L	N	Y	Y	N	CC	1	4	CC	N	1	4	CC	N	T	T
N	N	N	N	Y	N	N	N	N	N	X	B/L	N	Y	N	N	CC	1,3	4	CC	1,3	4	CC	N	N	T	T
N	7	N	N	Y	N	N	N	N	N	X	B/L	N	N	N	C/L	CC	1	4	CC	N	1	4	CC	N	T	Rec, T
N	N	N	N	Y	N	N	N	N	1	LA	B/L	N	Y	N	N	CC	1	4	CC	N	1	4	CC	N	T	T
Y	N	N	N	Y	N	N	N	N	N	X	B/L	N	N	Y	N	CC	1	4	CC	N	1	4	CC	N	T	T
Y	N	N	N	Y	C	C	N	N	2	GA	RT	N	Y	Y	N	V	1	4	PVA, Co	T	N	4	N	N	T	MA

109	5/5/2004	K	64/F	227483	D	S	B/L	A	N	N	N	Y	3W	Y	N	N	N	Y	Y	N	N	N	N		Y	Y	N	B
110	21/7/2004	HD	53/M	229893	D	S	LT	G	N	N	N	Y	15 D	N	N	N	N	N	Y	N	N	N	N		Y	Y	Y	[illegible]
111	15/10/2004	R	63/M	232740	D	S	LT	G	N	N	N	N	10 D	N	N	N	Y	Y	N	N	N	N	N		N	Y	Y	B
112	14/12/2004	KP	60/M	234706	D	S	RT	G	N	N	N	Y	2 M	N	N	N	N	Y	Y	N	N	N	B		Y	Y	N	B
113	25/1/2005	A	13/M	235650	D	S	RT	G	N	N	N	Y	2 M	N	N	N	N	N	Y	N	N	N	N		Y	Y	N	
114	27/1/2005	Y	68/F	234752	D	S	LT	G	N	N	N	Y	2W	N	N	N	N	Y	N	N	N	N	W		Y	N	N	L
115	31/3/2005	C	72/M	238648	D	S	RT	A	N	N	N	Y	3W	N	N	N	N	Y	Y	N	N	N	N		Y	Y	Y	[illegible]
116	1/4/2005	B	55/F	238770	D	S	LT	G	N	N	N	Y	5M	Y	N	Y	Y	Y	Y	N	N	N	N		Y	Y	Y	[illegible]
117	10/6/2005	L	62/F	241243	D	S	B/L	G	N	N	N	Y	1M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	N	[illegible]
118	18/8/2005	MT	34/F	243749	D	S	LT	G	N	N	N	Y	3Y	Y	Y	Y	N	N	Y	N	N	N	W		Y	Y	Y	[illegible]
119	22/3/2006	J	35/F	251549	D	S	RT	G	N	N	N	Y	1.5 M	Y	N	N	Y	Y	Y	N	Y	N	LW		Y	Y	Y	[illegible]
120	19/4/2006	M	61/F	252298	D	S	LT	G	N	N	N	Y	3W	Y	N	Y	Y	Y	Y	N	N	N	N		Y	Y	Y	[illegible]
121	27/10/2006	SP	44/M	249278	D	S	LT	G	N	N	N	N	2 M	Y	N	N	N	Y	Y	N	Y	N	N		N	Y	N	
122	16/12/2006	S	53/M	261446	D	S	B/L	G	N	N	N	Y	1M	N	N	N	N	Y	Y	N	N	N	Pt		Y	Y	Y	
123	17/2/2007	RG	55/F	261613	D	S	LT	G	N	N	N	Y	4M	N	N	N	N	Y	Y	N	N	N	N		Y	Y	N	
124	24/3/2007	V	50/F	264053	D	S	B/L	G	N	N	N	Y	8M	N	N	Y	N	N	Y	N	Y	N	W		Y	Y	Y	
125	29/3/2007	KL	73/F	262250	D	S	LT	G	N	N	N	N	3 M	Y	N	Y	Y	Y	Y	N	N	N	N		N	Y	Y	
126	22/6/2007	N	38/F	264769	D	S	RT	G	Y	N	N	N	10 D	Y	Y	Y	N	N	Y	N	Y	N	N		N	Y	Y	
127	18/7/2007	S	28/F	267485	D	S	B/L	G	N	N	N	Y	4M	Y	N	N	N	Y	N	N	N	N	N		Y	Y	Y	
128	27/7/2007	PK	41/F	265998	D	S	RT	G	N	N	N	N	4 M	Y	N	N	N	Y	N	N	N	N	N		N	Y	Y	
129	23/8/2007	B	37/M	270568	D	S	RT	G	N	N	N	Y	2M	Y	N	N	N	N	Y	N	N	N	B		Y	Y	N	

N	L-3	N	N	Y	N	N	N	N	1	GA	B/L	N	Y	Y	N	V	1,2	4	PVA	NT	1,2,3	X	Co	T	T	T
Y	N	N	N	Y	N	N	N	N	1	LA	LT	N	Y	N	N	V	1,2	4	N	N	1,2	4	Co	T	T	T
N	N	N	N	Y	C	C	PC	N	1	LA	B/L	N	Y	N	N	A	1,2	4	PVA	80%	1,2	4	PVA	80%	RES	T
N	N	N	N	Y	C	C	N	N	1	LA	RT	N	Y	N	N	A	1,2	4	Co	T	1,2	4	Co	T	T	T
N	N	N	N	N	N	N	N	N	1	LA	RT	N	Y	Y	N	A	2	4	PVA	T	N	N	N	N	T	T
N	N	N	N	Y	N	N	N	N	2	LA	B/L	N	Y	N	N	A	1,3	4	PVA	T	1,3	4	PVA	T	T	T
N	N	N	N	Y	N	N	N	N	2	GA	RT	N	Y	N	N	A.V	1,3	4	Gf	T	N	N	N	N	NT	MA
N	L-4	N	N	Y	C	C	N	N	1	LA	B/L	N	Y	N	N	A	1,3	4	PVA	T	1,3	N	PVA	T	T	T
N	N	N	N	Y	N	N	N	N	1	LA	B/L	N	Y	Y	N	V	1,2	N	N	N	1,3	4	Co	T	T	T
N	N	N	N	N	N	N	N	N	N	N	B/L	N	N	N	CVR	N	N	4	N	N	1,2	4	D	N	D	D
N	N	N	N	Y	N	N	PC	N	1	LA	B/L	N	Y	Y	N	A	1,2,3	4	PVA	NT	1,2,3	4	PVA	NT	NT	T
N	N	N	N	Y	N	NT	NT	N	1	LA	B/L	N	Y	N	N	A	1	4	PVA	MR	1	4	PVA	MR	NT	Rec, T
N	N	N	N	Y	C	C	N	N	1	LA	LT	N	N	N	DVD	A	N	N	N	N	1	4	N	N	Res	MA
N	R-3	N	N	Y	N	N	N	N	N	N	B/L	N	N	N	N	CC	N	4	CC	N	1	4	CC	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	LT	N	Y	N	N	A	N	N	N	N	1	4	PVA	NT	NT	T
N	N	N	N	N	N	N	N	N	1	LA	B/L	N	Y	N	N	A	1,2,3	4	Gf	T	1,2,3	4	PVA	MR	NT	T
N	N	N	N	Y	N	N	N	N	1	LA	B/L	N	Y	N	N	A	2	N	PVA	T	2	N	PVA	T	T	T
N	N	N	Y	N	N	N	N	N	1	LA	RT	N	Y	Y	N	A	2,3	4	PVA	NT	N	N	N	N	NT	T
N	N	Y	N	Y	N	N	N	N	1	LA	B/L	N	Y	N	N	A	1,3	4	PVA	T	1,3	4	PVA	N	T	T
N	N	N	N	Y	N	N	N	N	1	LA	B/L	N	Y	N	N	V	N	4	Co	T	1,3	N	Gf	T	T	T
N	N	N	N	N	N	N	N	N	1	LA	B/L	N	Y	Y	N	V	2,3	4	Co	T	3	4	Co	T	T	T

KEY TO MASTER CHART

1)-Column 1- S/N – Serial number
2)-Column 2 – D – Date
3)-Column 3 – N- Name
4)-Column 4 – A/S – Age /Sex, M- Male; F- Female
5)-Column 5 – H/N- Hospital number
6)-Column 6 – T – Type of fistula
7)-Column 7 - E – Etiology; T- Traumatic, S- Spontaneous
8)-Column 8 - S/P- Side at presentation, Lt- Left; Rt – right; B/L – Bilateral
9)-Column 9 – o – Onset; G- Gradual, A- Acute
10)-Column 10 – T- Trauma, Y- Yes, N- No
11) Column 11- LOC- Loss of consciousness; Y- Yes; N- No
12)-Column 12- F – Fractures; Y-Yes; N- No
13)-Column 13- S/E- Swelling of eye; Y-Yes; N-no
14)-Column 14 – D- Duration of symptoms; D- Days; M-Months, Y- Years.
15)-Column 15- HA- Headache; Y-Yes; N-No
16)-Column 16 – Vo- Vomiting; Y- Yes, N-No
17)-Column 17-P- Pain in eye; Y-Yes; N-No
18)-Column 18 –D/V- Diminution of vision, Y-Yes; N- No; Vl- Visual loss
19)-Column 19- Di – Diplopia; y-Yes; N-No
20)-Column 20 – R- Redness, Y- Yes,N- No
21)-Column 21- Ve- Vertigo; Y- Yes;N- No
22)-Column 22- Ti – Tinnitus; Y-Yes; N- No
23)-Column 23 – Wa- Watering; Y- Yes; N- No
24)-Column 24- A/F- Additional Features
25)-Column 25 –O/E- On Examination
26)-Column 26- P-Proptosis;Y-Yes; N-No
27)-Column 27-R-Redness; Y-Yes, N-No
28)-Column 28 – C-Chemosis; Y-Yes, N-No
29)-Column 29 – 6NP- 6th Nerve palsy, L-6 – left 6th, R-6 – right 6th; B/L-6- Bilateral 6th E0- No Extra- ocular movements
30)-Column 30 – MNP- Multiple nerve palsy;Y- Yes; N-No
31)-Column 31- ONP- Other nerve palsy;
32)-Column 32-B- Bruit,orbital; Y- Yes ;N-No
33)-Column 33- A/B-Additonal bruit; Y-Yes; N-No
34)-Column 34 -Di – Diplopia ;Y-Yes; N-No
35)-Column 35- VARE- Visual acuity right eye, N-Normal; C- Compromised; Nil- visual Loss.
36)-Column 36- VALE –Visual acuity left eye , N-Normal; C- Compromised; N-Nil.
37)-Column 37- VF- Visual filed, N-Normal; C- Compromised;PC- Peripheral constriction; Nil- Blind
38)-Column 38-OA- Optic atrophy ,Y-Yes;N- No
39)-Column 39- N/E- No of embolisations
40)-Column 40-LA/GA,local/General anaesthesia
41)-Column 41- S/A – Side of CCF on angiogram, Lt- left, Rt- Right, B/L- bilateral

42)-Column 42- S/R- Site of rent
43)-Column 43- SOV- Superior ophthalmic vein,Y-Yes;N- No
44)-Column 44- IPS- Inferior petrosal sinus, Y- Yes: N- No
45)-Column 45- ADV – Additional venous drainage ;IOV- Inferior ophthalmic vein, CVR- Cortical venous reflux ,DVD- Deep venous drainage ;C/L CS- Contralateral cavernous sinus
46)-Column 46- R/E- Route of embolisation
47)-Column 47- RECA- Right ECA; Y- Yes; N- No;1=IMA,2=MMA,3=Asc Ph,4=ICA dural
48)-Column 48 – RICA- Right ICA; Y- Yes; N- No
49)- Column 49- MAT R – material used on right ;B- balloon,C- coils ,B+C – Balloons + coils, Hg- Hydrogel; Gf- Gelfoam, PVA- PVA particles
50)- Column 50- E/O R – Extent of occlusion right; T- total , NT- Near total
51)-Column 51 – LECA – Left ECA; Y- Yes ;N- No;1=IMA,2=MMA,3=Asc Ph,4=ICA dural
52)-Column 52- LICA- Left ICA
53)- Column 53 Mat L- Material used on left side , B- balloon,C- coils ,B+C – Balloons + coils, Hg- Hydrogel; Gf- Gelfoam, PVA- PVA particles.
54)- Column 54 – E/O L- Extent of occlusion left ;T- total , NT- Near total
55)-Column 55- Status after first embolisation, T- Total ;NT- Near total; MR – minimal Residual ; Tr- Trapping of ICA
56)- Column 56- FU- Follow up – T- Total closure, NT- Near Total; Sp –Spontaneous closure ;MR – Minimal residual , Tr- Trapping of ICA,MA- Multiple attempts
57)- Column 57- C- Complications ; N- No complication ;D- Death ; Hp- Hemiparesis, Mi- Minimal , Ma-Major .