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INTERVENTIONAL NEURORADIOLOGY

Endovascular treatment of direct carotid cavernous fistulae: a pictorial review

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Abstract
Introduction Direct carotid-cavernous fistulae (CCFs) are type A fistulae according to Barrow's classification. Endovascular treatment of these lesions is challenging.
Methods The purpose of this review was to evaluate the endovascular treatment of direct CCFs. We also describe the technique, symptomatology and complications associated with the procedure and report on the long-term follow-up in our treated patients.
Results A total of 89 patients with a direct CCF were treated. All patients had baseline brain CT or MR imaging. Treatment comprised transarterial balloon or coil embolizations. The patients were followed up at 1 month and then every 6 months thereafter. Detachable balloons were used in 79 fistulae. In 12 fistulae the balloon could not be negotiated through the fistula and these fistulae were treated with transarterial coil occlusion. Clinical outcomes of the treated patients evaluated at 1 month were: 79 patients (88.8%) cured, 9 (10.1%) significantly improved, 1 (1.1%) remaining static.
Conclusion Endovascular treatment of direct CCFs is safe and effective and results in long-term cure.

Keywords Carotid-cavernous fistula · Balloon · Embolization · Angiography

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Introduction

Direct carotid-cavernous fistulae (CCFs) are type A fistulae according to Barrow's classification [1]. Etiologically, most direct CCFs are traumatic, but less commonly they may be spontaneous [2–7]. Signs of CCF feature a triad of pulsatile exophthalmos, orbital bruit, and conjunctival injection. Indications for aggressive treatment include visual impairment, progressive paresis of extraocular muscles, intractable orbital pain or bruit, and progressive or severe exophthalmos [8, 9]. In 1973, Parkinson reported successful direct surgical repair of direct CCFs with preservation of the parent artery [10]. However, the technical difficulty and high invasiveness of this procedure have precluded wide adoption of surgical treatment. Endovascular techniques rapidly evolved thereafter and are now the first line of treatment for most CCFs.

The purpose of this review was to evaluate the therapeutic benefit of endovascular treatment of direct CCFs. We describe the technique, symptomatology and complications associated with the procedure, and discuss the long-term follow-up in our treated patients.

Etiopathogenesis and symptomatology

A total of 89 patients with a direct CCF were treated (72 male, 17 female; age range 12–57 years, mean 37.6 years). Direct CCFs are high-flow shunts between the cavernous portion of the internal carotid artery (ICA) and the cavernous sinus, and are usually caused by traumatic laceration of the ICA or rupture of an intracavernous carotid aneurysm [11]. They occur rarely in children with connective tissue disorders such as Ehlers-Danlos syndrome or after trauma [12–17]. Spontaneous childhood

fistulae are extremely unusual, the youngest reported patient being a 5-year-old boy [18]. In our series, the youngest patient was 12 years old with a fistula of traumatic origin. Rai et al. reported the case of an 11-month-old female patient with a symptomatic direct CCF without a preceding history of trauma or coexisting collagen vascular disorder [19].

The clinical presentations of our patients are given in Table 1. Direct CCF usually presents with ocular symptoms. Halbach et al. reported that 32.3% of patients with direct CCF in their series presented with diminishing visual acuity. Minor decreases in visual acuity were reversible with the closure of the fistula. We found a visual deficit in 25.8% of patients with complete recovery in 82.6% after treatment. Proptosis has been found to occur in 75% of indirect CCFs and 77% of direct CCFs [20]. Rapidly progressive proptosis may signify spontaneous thrombosis of venous outflow pathways to the orbit. Because of the severe symptoms associated with this occurrence, such a finding is an indication for immediate treatment.

Hemorrhage is a devastating complication of a CCF. The first case was reported by de Schweinitz and Holloway in 1908 [21]. Sattler reviewed 322 cases of CCF reporting an incidence of 1.5% fatal epistaxis and 0.9% intracerebral hematoma [22]. Halbach et al. reported that hemorrhage occurred in 8.4% of patients with direct CCF. All patients who suffered an intracerebral hemorrhage demonstrated cortical venous drainage [20]. We found an intracranial bleed in 4.5% of patients with features of venous ectasia and cortical venous drainage.

Patients with direct CCF can also present with the features of raised intracranial pressure (ICP). In the series of Halbach et al., 7.7% of the patients presented with symptoms of raised ICP [20]. Flow problems in these patients are created more by the increase in venous pressure and consequent passive congestion of the brain than by arterial steal [23]. These symptoms appear to be related to chronic passive congestion due to a retrograde increase in venous pressure towards the venous drainage routes of the normal brain with concomitant diminution of cerebral fluid absorption resulting in an increase in ICP [24, 25]. We have encountered raised ICP in patients with cortical venous drainage.

Table 1 Clinical presentations of the 89 patients in this study

Types of clinical presentation	Number of patients
Headache	75
Proptosis	87
Chemosis	89
Diplopia	78
Visual deficit	23
Tinnitus	64
Seizure	5
Neurological deficits including cranial nerve deficit	39
Abnormalities of mentation	5
Intracranial bleed	4

Imaging

All patients had baseline neck vessels and transcranial Doppler studies, brain CT or MR imaging (Fig. 1), routine blood investigations and cardiological work-up before the procedure. Initial imaging often consists of a CT scan. An asymmetrically enlarged cavernous sinus or superior ophthalmic vein is suggestive of a CCF and should prompt a call to the referring physician for further clinical information. Digital subtraction angiography is essential in confirming the diagnosis and delineating the exact patterns of venous drainage. We encountered evidence of fistula in all cross-sectional studies.

Route and technique of embolization

In 1973, Parkinson reported successful direct surgical repair of direct CCFs but the high invasiveness of this procedure precluded its wide adoption [10]. Subsequently, other direct interventional treatments have been attempted such as electrothrombosis of the cavernous sinus [26, 27]. Since Serbinenko reported his experience with a detachable balloon, endovascular treatment has become the first choice for the treatment of direct CCF [28]. Fistula occlusion using a detachable balloon delivered by a transarterial route is the preferred method for treating direct CCF [2, 7, 9, 29, 30]. Materials for detachable balloons include latex and silicone. Of these, latex balloons are preferred [7, 31, 32]. Balloons can be inflated with polymers or contrast material. But the former may impede recovery from ocular palsy and the latter may deflate within a few months and may result in recurrence of the aneurysm [31–34].

Our technique of embolization

All procedures were done under local anesthesia. In few uncooperative patients additional neuroleptoanalgesia was administered. The transarterial technique was used. A 9F guiding catheter was placed in the cervical ICA. Then detachable balloons were mounted on the microcatheters. Previously we used a coaxial double microcatheter for balloon attachment with latex threads (Nycomed, France), but now we prefer to use a single microcatheter (Goldbal, BALT extrusion, France) for balloon deployment. The

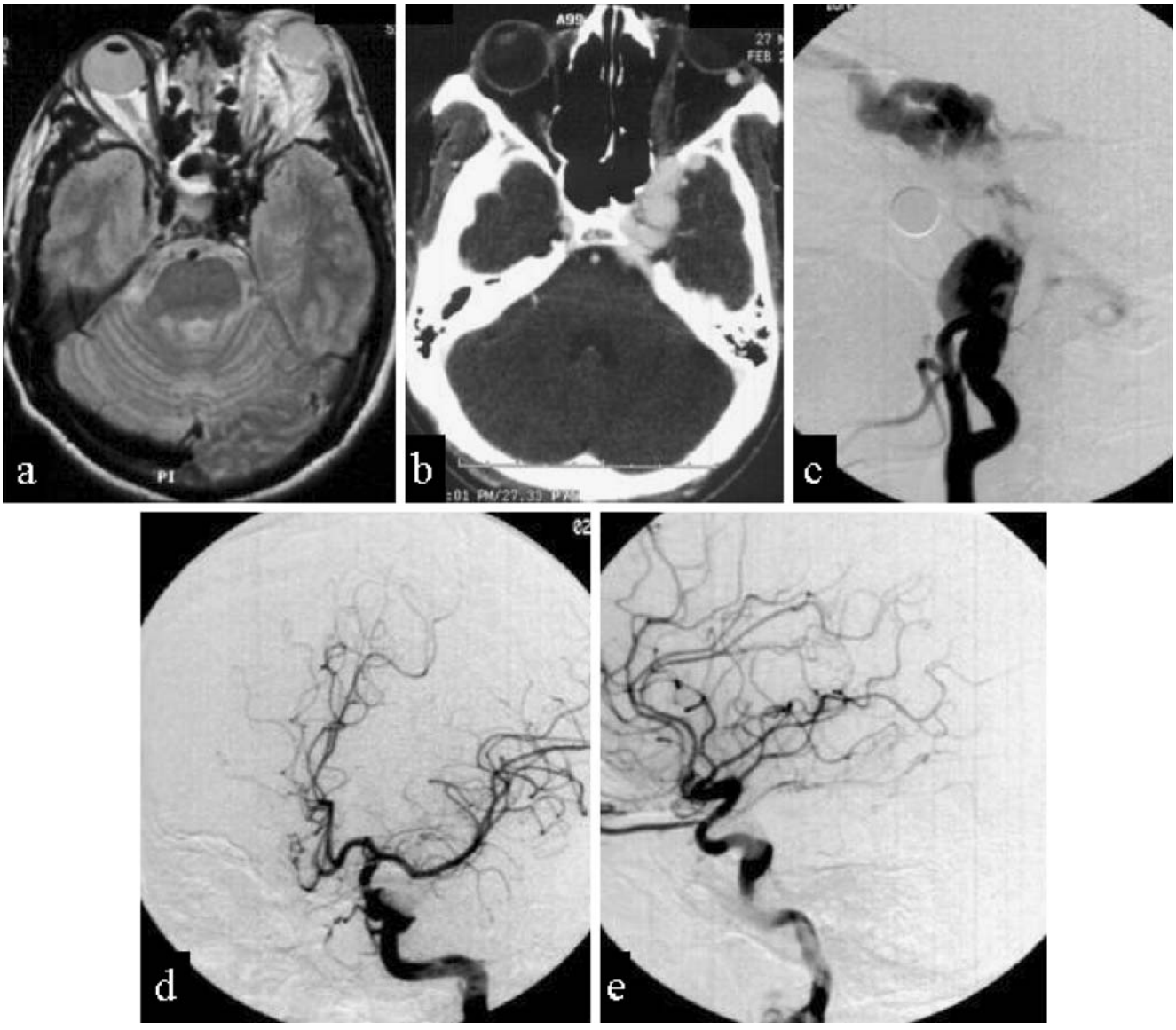

Fig. 1 **a**, **b** Axial MRI and CT scan shows enlarged left cavernous sinus with left sided proptosis. **c** Injection of the left ICA shows the CCF. Note drainage into the inferior petrosal sinus and mild cortical reflux. **d**, **e** Angiogram after embolization shows complete closure of the fistula

balloon-mounted microcatheter was negotiated slowly in to the fistula by flow guidance. In some procedures micro-wires were also used to support the assembly for proper navigation. In order to confirm the location of the balloon and to characterize the targeted fistula, contrast agent was injected through the guiding catheter. Once a satisfactory position of the balloon had been achieved, it was inflated to see the status of the fistula. Sometimes the balloon was repositioned inside the fistulous sac for proper closure of the fistulous site. The balloon was then detached. In some procedures more than one balloon was used to close the fistula properly. In a few patients, where the balloon could be negotiated in the sac or the fistula was very small and slow, transarterial coil occlusion of the fistula was also performed. Electrolytically or mechanically detachable coils were used to close the fistulae. The coil position was checked prior to detachment to ensure there was no prolapse into the ICA.

A transarterial approach is recommended for balloon embolization [9, 30]. However, transvenous embolization for traumatic direct CCF has been reported [2, 7, 9, 30, 35]. There are some pitfalls in balloon embolization. In some instances, the balloon may protrude into the lumen of the ICA when inflated sufficiently to occlude shunt flow. In some other cases in which the balloon protrudes, occlusion of the ICA may be the best alternative; turbulent flow around a protruding balloon can cause thromboembolism or distal migration of the balloon itself [7]. This happens when multiple balloons are used, and we encountered this in one patient with a huge fistula for which seven balloons were

used. Transarterial cavernous sinus packing with detachable coils is another useful method, particularly for small to medium fistulae with limited shunt flow [36–38]. Also the transarterial approach may not be successful if the volume of the venous compartment is too small to accommodate the balloon or the communication is too small to allow balloon entry or such that sharp objects may puncture the balloon during inflation [2]. In 1988, Halbach et al. reported the results of 165 direct type fistulae treated by endovascular therapy. In 14 patients (8.5%) it was performed via the transvenous route because of failure of transarterial attempts. In 13.2% of patients in our series the fistula required coil embolization, most secondary to aneurysm rupture. In some fistulae with low shunt flow, aneurysm packing with coils may suffice [38, 39]. However, the optimal volume of coils to be embolized is difficult to determine and coil migration is a risk [36–38]. The use of a neck-bridging device (TriSpan; Target Therapeutics/Boston Scientific, Fremont, Calif.) and intracranial stents can prevent coil migration into the ICA during transvenous occlusion of a high-flow, large-tear CCF [40–43].

Results of interventional treatment of CCF

We encountered a total of 91 direct CCF were in 89 patients, of which 85 were traumatic in origin (including two bilateral fistulae), and 6 were secondary to aneurysmal rupture. Of the 91 fistulae, 67 were seen in the ICA at C3, 7 at C4, 9 at the C3/C4 junction, and rest in the proximal genu. In 85 fistulae there was a medium sized rent in the ICA segment with venous drainage into the superior ophthalmic vein, inferior ophthalmic vein and inferiorly to the pterygoid venous plexus. Six fistulae had predominantly cortical venous drainage, 18 showed contralateral venous

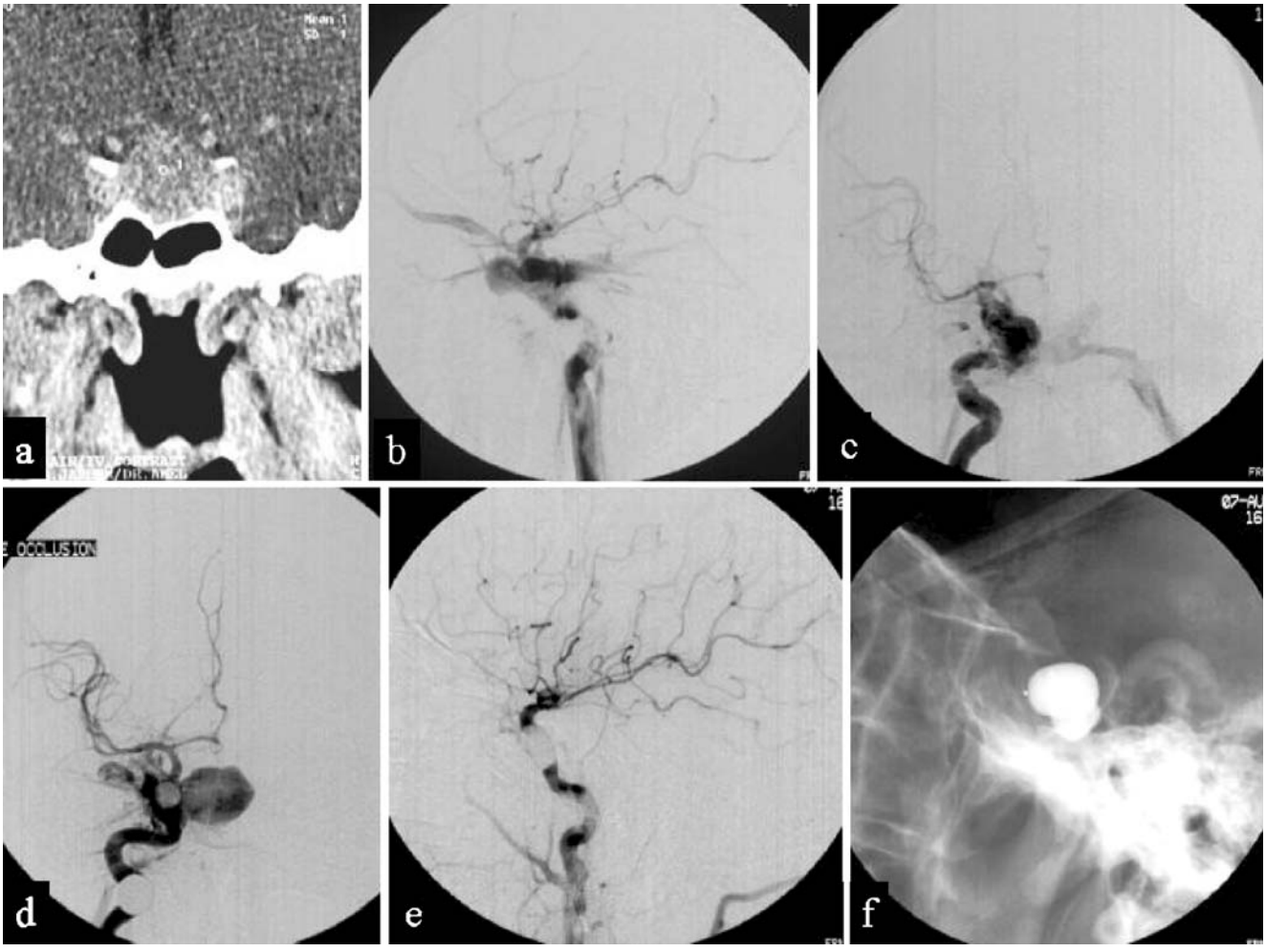

Fig. 2 **a** Postcontrast coronal CT scan shows an enlarged right cavernous sinus. **b**, **c** Lateral and AP views of the right ICA following injection shows a type 1 CCF. Note drainage to the opposite side also. **d** AP view after deployment of the first balloon shows filling of the remaining fistula. Note the large venous ectatic sac. **e** The second balloon completely closes the fistula. **f** Fluoroscopic image shows the balloons

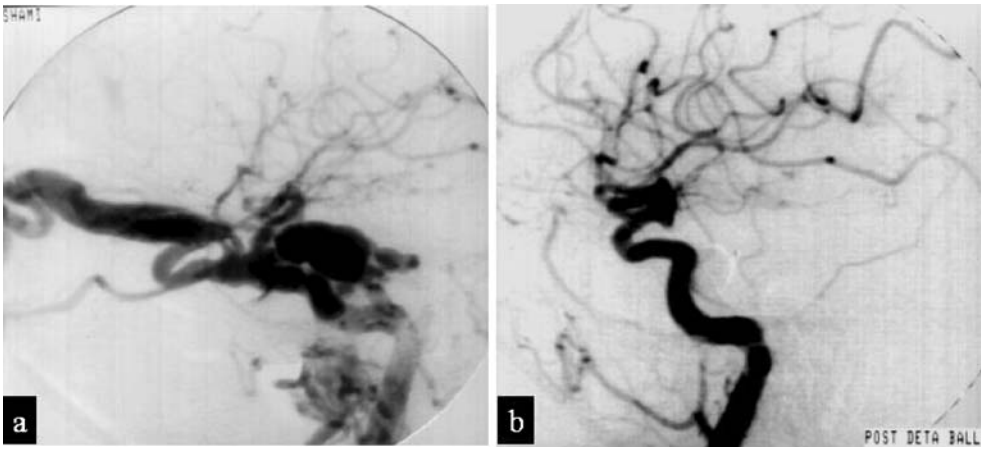

Fig. 3 **a** Type 1 CCF draining into the superior ophthalmic vein. The fistula is located at the junction of the first genu and the ascending segment. **b** A single balloon closed the fistula

drainage to the opposite cavernous sinus through the circular sinus, and 56 showed posterior drainage through the inferior petrosal sinus. The facial vein was seen in all predominant cases of anterior drainage. Venous ectasia are seen in eight fistulae and all were of traumatic origin (Fig. 2). Distal arterial tree filling was limited in 78 fistulae. Pial collateral filling from the posterior circulation was noted in 45 fistulae. The exact site of the fistula was established clearly in all cases by injection from the vertebral artery with ipsilateral carotid compression.

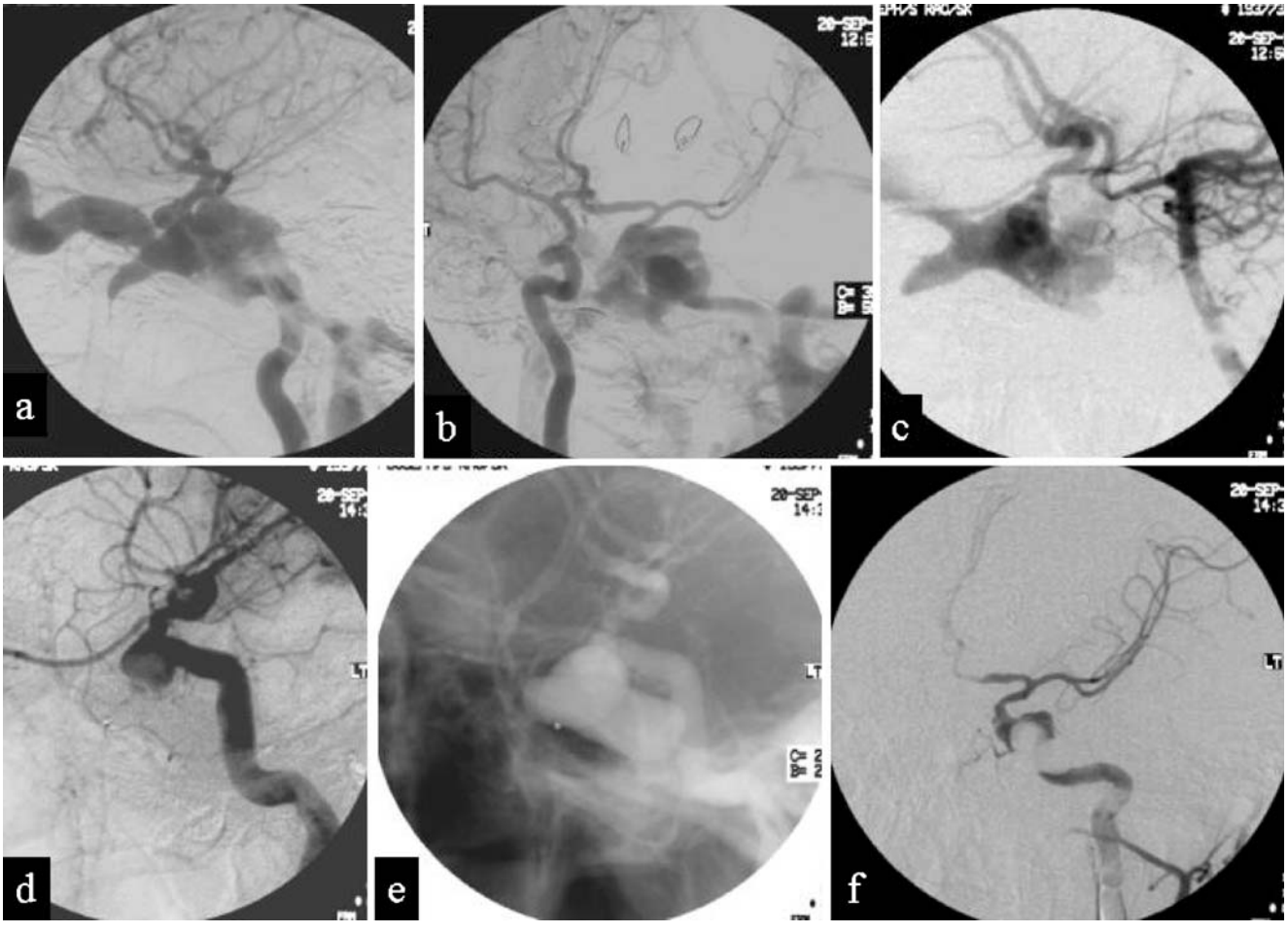

Fig. 4 Lateral view of the left ICA following injection shows a type 1 CCF draining into the superior ophthalmic vein. Note the venous pouch. **b**, **c** Oblique and lateral views of the right ICA following injection show the exact site of fistula. **d**–**f** Lateral and AP views of the left ICA following injection show complete closure of the fistula. Two large balloons were placed in the cavernous sinus

Fistulae were treated via the transarterial route. Detachable balloons were used in 79 fistulae. A single balloon was sufficient for complete treatment of 55 fistulae (Fig. 3); 6 required two balloons (Fig. 4), 8 required three balloons (Fig. 5), 8 required 4 balloons, 1 required six balloons, and 1 required seven balloons. In the last mentioned case, after detaching the seventh balloon, the ICA suddenly became occluded. We found no recurrence of traumatic CCF after treatment with balloons. We used contrast medium for balloon inflation. We found that thrombosis in the cavernous sinus prevents recurrence of the fistula even after balloon deflation.

In 12 fistulae the balloon could not be negotiated through the fistula, and in these cases transarterial coil occlusion was done. An average of seven coils were needed to close the fistula (Fig. 6).

Out of 79 fistulae treated using balloons, 72 (91.1%) showed complete angiographic occlusion, of which 7 (9.7%) showed mild filling of the fistula and 2 (2.8%) showed moderate filling of the fistula. Nine fistulae (75.0%) treated using coils showed complete occlusion. The remaining three (25.0%) showed only mild filling of the fistula. The clinical outcomes of the treated patients evaluated at 1 month were: 79 patients (88.8%) cured, 9 (10.1%) significantly improved, and 1 (1.1%) remaining static. Doppler studies were done in all patients during follow-up.

We encountered minimal complications in 3.4% of patients. One patient developed multiple cranial nerve palsy but partially recovered after steroid treatment. In one patient the ICA became closed after placing seven balloons in a huge fistula. He had good collaterals from the contralateral ICA and via the posterior communicating artery and he did not suffer any deficit. In one patient who was treated by transarterial coil embolization with mechanically detachable coils, the last coil would not detach, and on mild pulling the whole coil mass moved. We decided to leave the coil in the arterial tree cutting it at the level of the femoral sheath. We kept the patient on anticoagulation for 1 week and then on antiplatelet therapy. The patient did not suffer any deficit.

The range of follow-up was 2 months to 9 years (average 4.67 years). All patients at 1 month follow-up had a Glasgow outcome score of 5. The angiographically cured patients did not show any recurrence of symptoms. Among the remainder, nine had minor headache and diplopia on looking laterally to the extreme. No patient had any degree of proptosis. The remaining two patients had persistent mild

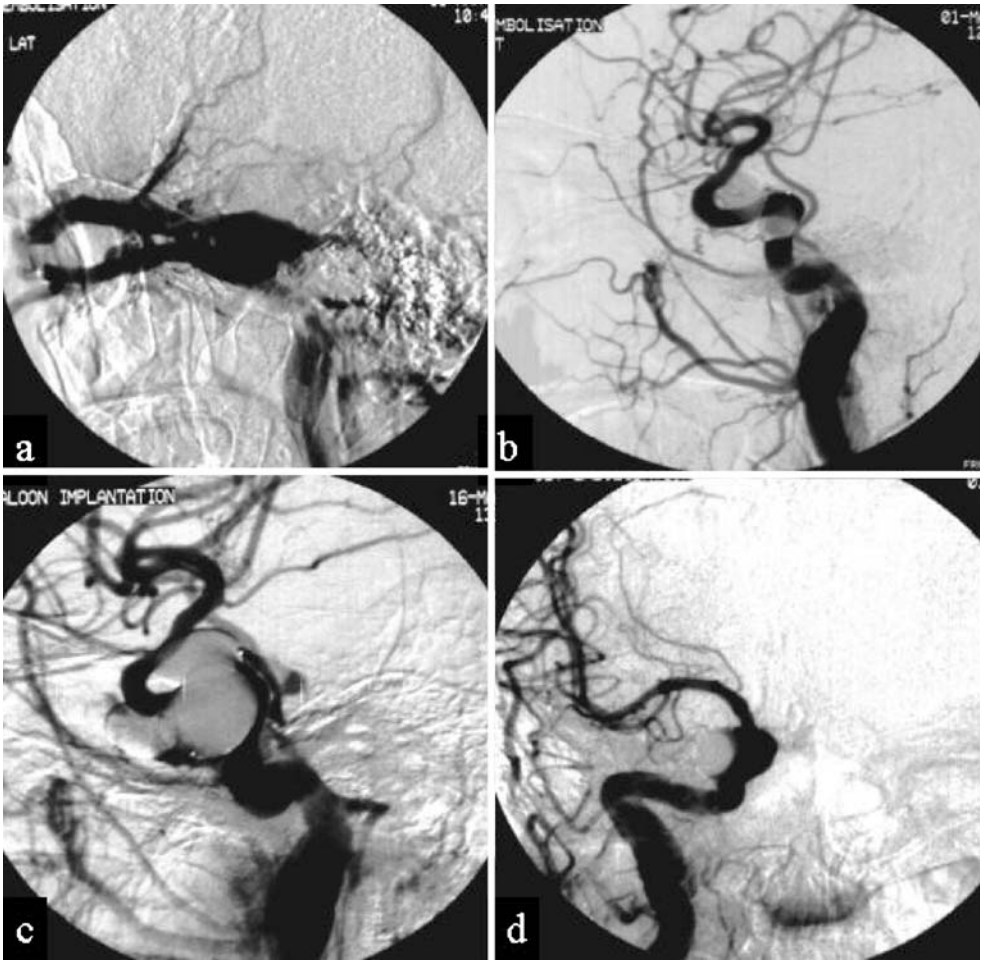

Fig. 5 **a** Lateral view of the right ICA following injection shows a type 1 CCF draining into both ophthalmic veins. Mild cortical venous reflux is noted. **b–d** Lateral and anteroposterior views show three balloons deployed into the fistula with complete closure of the fistula

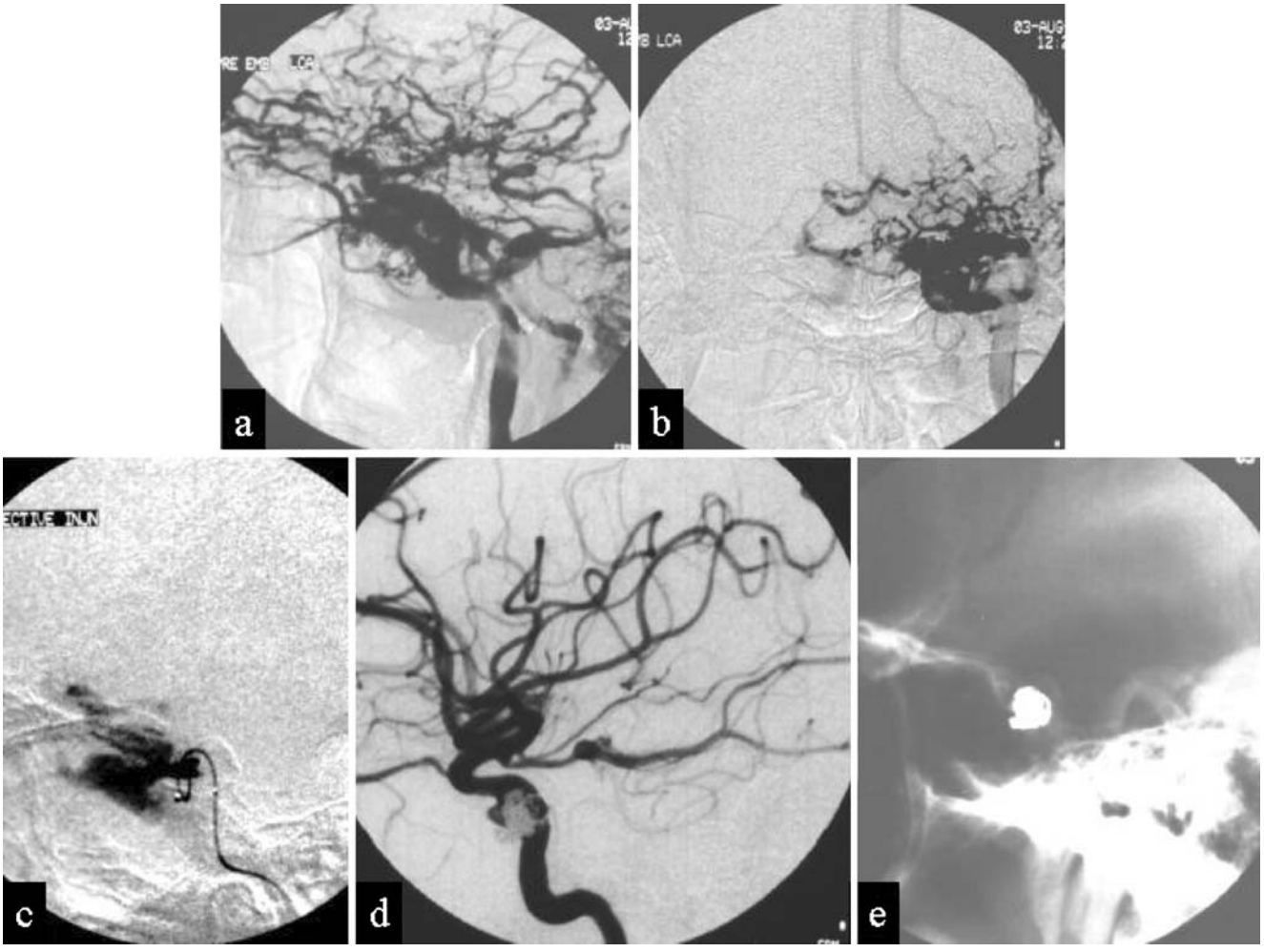

Fig. 6 **a**, **b** Lateral and AP views of the left ICA following injection shows a type 1 CCF with massive cortical venous drainage along with drainage to inferior petrosal sinus. **c** Due to the small size of the rent a balloon could not be negotiated into the fistula. A microcatheter was taken transarterially into the fistula. **d** Coils were deposited into the sac with complete closure of the fistula. **e** Fluoroscopic image shows the coil mass

proptosis and diplopia. All patients who showed abnormalities in mentation in the acute stage showed clear improvement. Out of 23 patients who suffered various grades of visual deficit, 19 recovered completely, and 4 had fixed deficits.

The reported success rate of balloon embolization of CCF is 88–98% [7, 44, 45]. Kobayashi et al. achieved 80% aneurysmal and 55% post-traumatic CCF closure by balloon [46]. However, the risks involved are deterioration of ocular palsy and ICA occlusion in approximately 10–40% of patients [7, 44, 47, 48]. We encountered this complication 1.1% of patients, which is lower than in previous series. Transarterial embolization has emerged as the treatment of choice for direct CCFs [28, 45]. Halbach et al. treated over 200 traumatic CCFs with complete occlusion of the fistula in 99% of patients and with preservation of the parent artery in 88% of patients [45]. We achieved complete occlusion in 86.3% of fistulae, near total occlusion in 11.0% and ICA preservation in 98%. Most of the fistulae that showed complete or nearly complete occlusion were treated within 2 weeks of occurrence.

Intracranial bleed with CCF is commonly seen in the presence of venous ectasia. So these cases need urgent intervention. Early treatment can lead to reversal of visual deficits in these patients and also increase the chance of complete cure. In most patients with proper technique the parent artery can be saved and the fistula can be closed. To conclude, interventional treatment of direct CCFs is a safe and effective treatment and can be done without any significant long-term complications.

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Conflict of interest statement We declare that we have no conflict of interest.

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