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Original Article

Aggressive cavernous sinus dural arteriovenous fistula: Angioarchitecture analysis and embolization by various approaches

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Abstract

Background: Most cavernous sinus dural arteriovenous fistulas (CSDAVFs) present with benign neuro-ophthalmic symptoms. CSDAVFs manifesting with aggressive neurologic symptoms are rare. The purpose of this study was to analyze the different angioarchitectures of aggressive CSDAVFs and to report our experiences of embolization.

Methods: Over the past 10 years, a total of 118 CSDAVFs were managed by embolization. From the databases containing such patient information, nine patients (7.6%) were found to have aggressive CSDAVFs presenting with neurologic deficits. There were seven women and two men, ranging in age from 51 years to 78 years (mean, 66 years). We retrospectively analyzed the angioarchitectures of aggressive CSDAVFs, further reviewing patient and angiographic as well clinical outcomes after embolization.

Results: The cause of clinically aggressive CSDAVFs was insufficient fistula drainage because of occlusion ($n = 6$) or stenosis ($n = 1$) of the inferior petrous sinus (IPS) or compartment of IPS−cavernous sinus ($n = 2$) with fistula flow reflux to the veins of brainstem ($n = 7$) leading to brainstem ischemia, while two fistula flow reflux to the cortical vein leading to cerebral infarction. Transvenous embolization via IPS to fistula was achieved in one case; six patients underwent transorbital access, while transarterial embolization was performed in two cases. Total fistula occlusion was achieved in eight CSDAVFs. All patients had total ($n = 7$) or partial ($n = 2$) resolution of their symptoms gradually within 6 months. One patient undergoing transarterial embolization had limb weakness because of inadvertent pial artery occlusion. Their overall mean clinical follow-up period was 17 months.

Conclusion: Aggressive CSDAVFs are associated with occlusion/stenosis of the IPS or compartment of IPS−cavernous sinus with leptomeningeal reflux. In this limited case series, aggressive CSDAVFs most presented with brainstem ischemia, followed by nonhemorrhagic/hemorrhagic stroke in the cerebrum. Embolization through various access routes is a feasible method to manage these aggressive CSDAVFs, with an acceptable level of periprocedural risks.

Keywords: cavernous sinus; dural arteriovenous fistula; embolization; stroke

Conflicts of interest: The authors declare that they have no conflicts of interest related to the subject matter or materials discussed in this article.

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1. Introduction

Intracranial dural arteriovenous fistulas (DAVFs) are uncommon cerebrovascular lesions appearing in 10−15% of the total intracranial vascular malformations.[1] Intracranial DAVFs have a wide spectrum of clinical presentations,[1−4] although aggressive DAVFs are generally considered as fistulas with insufficient venous drains with leptomeningeal or pial venous reflux.[3] Aggressive DAVFs have a tendency to produce hemorrhagic or nonhemorrhagic stroke or death. Minor neurologic deficit such as cranial nerve palsy was not considered as aggressive DAVFs.[5] Most cavernous sinus DAVFs (CSDAVFs) are low-flow shunts fed by dural branches of the internal and/or external carotid arteries. CSDAVFs commonly present with benign ocular symptoms of orbital venous hypertension, or focal third and/or sixth cranial nerve palsy. CSDAVFs presenting with aggressive behavior with hemorrhagic or nonhemorrhagic neurologic deficit is uncommon.[6−8]

The purpose of this study was to evaluate the angioarchitectures of aggressive CSDAVFs, and to report our experiences of access and embolization to manage aggressive CSDAVFs.

2. Methods

2.1. Patients

From May 2004 to April 2014, a total of 102 patients with 118 CSDAVFs were referred to our institute to assess the feasibility of embolization because of intolerable neuro-ophthalmic symptoms or hemorrhagic/nonhemorrhagic stroke. In the databases, there were nine cases of CSDAVFs (7.6%) presenting with clinically aggressive behavior with ischemic stroke ($n = 9$; Figs. 1 and 2) and being associated with cerebral hemorrhagic transformation ($n = 1$). These patients consisted of two men and seven women, and their ages ranged from 51 years to 78 years (mean, 66 years). Prior to conducting this study, informed consent was obtained from all nine patients; their clinical data are summarized in Table 1.

2.2. Techniques of embolization

Embolization was performed on all patients under general anesthesia using bilateral femoral approaches, with the placement of 5-F and 6-F femoral sheaths in the left femoral artery and right femoral vein, respectively. Activated clotting time was monitored and maintained at a value twice the baseline value by intravenous administration of heparin. In all patients, transvenous access to CSDAVFs via the inferior petrous sinus (IPS) and coiling of the fistula was attempted first. This procedure was performed by placing a 4-F diagnostic catheter into the feeding carotid artery as a guide for subsequent transvenous catheterization; then a 6-F guiding catheter (Envoy; Codman & Shurtleff, Rayhnam, MA, USA) was placed into the internal jugular vein, followed by retrograde catheterization of the IPS and then the fistula site of the cavernous sinus (CS) for coiling. However, this route of access was successful only in one patient (Fig. 2). Access to the other eight aggressive CSDAVFs failed after many attempts because of total occlusion ($n = 6$; Fig. 1) or stenosis of the IPS. In these six patients with brainstem ischemia and edema, we decided to undertake a more invasive technique using a transorbital access to the fistula site by direct needle puncture of the proximal ophthalmic vein (OV; $n = 4$; Fig. 1) or CS ($n = 2$) under fluoroscopic and roadmapping guidance. An 18-gauge puncture needle (Terumo Corporation, Tokyo, Japan) was utilized. To avoid injury, the eyeball was pushed to its superior aspect, and the puncture needle was carefully advanced via the inferior orbital rim. During the advancement of the puncture needle, it was adjusted and checked intermittently to target the proximal OV or anterior CS depending upon the inclination of the inferior orbital wall. Once the tip of the puncture needle reached the OV or CS, the arterialized blood flowed from the fistula to the puncture needle, which was confirmed by hand-injection angiography. The inner metallic puncture needle was removed for subsequent microcatheter navigation into the fistula. Coiling of the fistula was initiated and continued until total fistula occlusion or recoil of the microcatheter back to the OV. In one patient, a microcatheter recoiled back to the anterior CS during coiling with residual fistula, to enhance the effect of fistula occlusion; Onyx-18 (ev3, Inc., Irvine, CA, USA) was slowly infused into the fistula site.

Two patients underwent transarterial embolization because they refused to undertake the risk of transorbital approach. Transarterial embolization was performed by navigation of a microcatheter into the accessory meningeal arteries by delivery of a 25% Lipiodol/liquid adhesive mixture ($n = 1$) or particle ($n = 1$) until the embolizer refluxed to the tip of the microcatheter or the fistula flow was sluggish.

A postembolization angiogram was performed immediately after the completion of the procedure to check for fistula occlusion. Seven cases had neuroimaging follow-up; conventional digital subtraction angiography (DSA) was obtained in two cases, while seven cases underwent magnetic resonance angiography (MRA) to evaluate the neuroimaging outcome of embolized aggressive CSDAVFs (mean, 8 months). All patients have been followed up clinically for an average of 17 months (range, 8−25 months).

3. Results

The demography and treatment outcomes of nine patients with aggressive CSDAVFs are summarized in Table 1. The clinical manifestations of these patients were chemosis ($n = 7$), limb weakness ($n = 8$), proptosis ($n = 2$), diplopia ($n = 2$), aphasia ($n = 1$), bruit ($n = 1$), unstable gait ($n = 1$), and respiratory failure ($n = 1$). Prior to performing embolization, all patients underwent brain computed tomography ($n = 3$) and/or MRA ($n = 8$). Brainstem ischemia and edema were detected in eight patients (Figs. 1 and 2); the other patient presented with ischemia with hemorrhagic transformation in the left temporal lobe. Pre-embolization carotid and vertebral DSAs showed all cases of aggressive CSDAVFs were fed by dural branches of internal and external carotid arteries

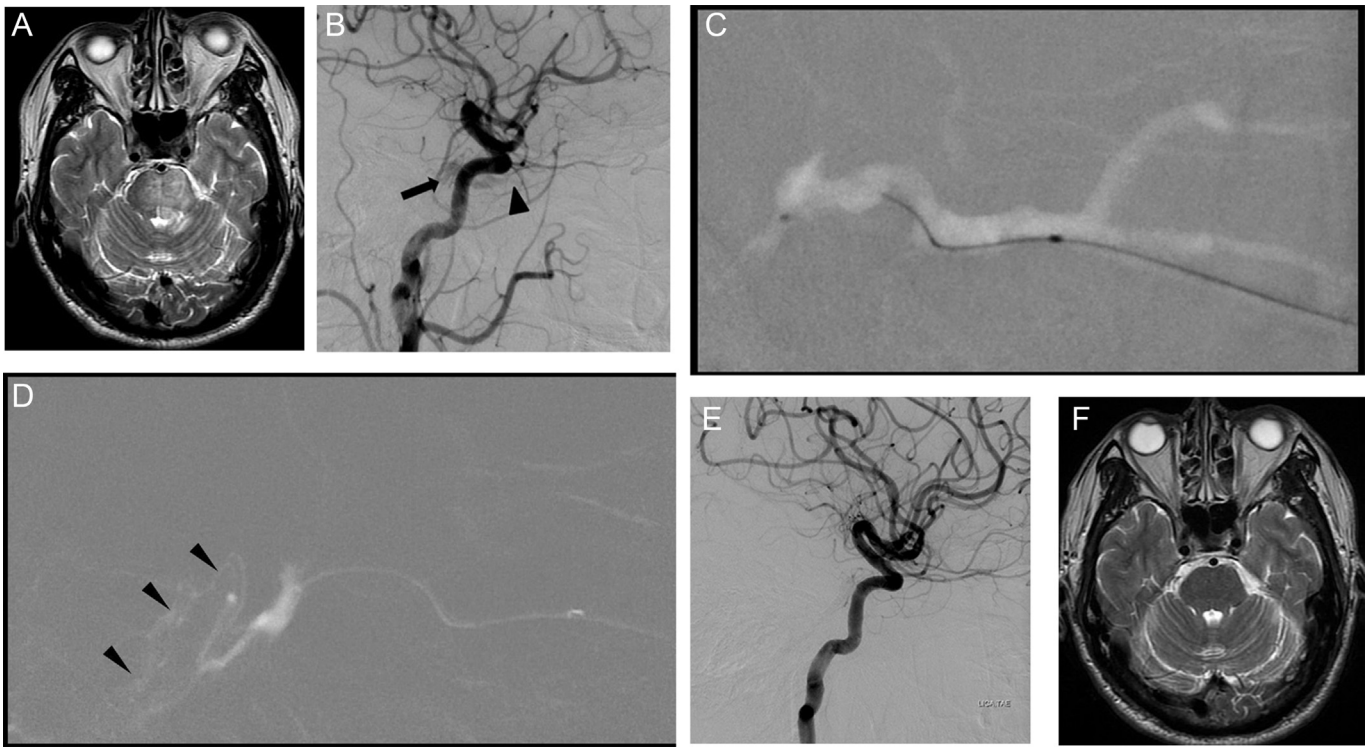

Fig. 1. A 60-year-old man presented with chemosis, proptosis, and unstable gait. (A) Brain MRI of T2WI demonstrated high-signal ischemia and edema at the brainstem. (B) Left lateral carotid angiograms demonstrated slow-flow CSDAVFs; fistulas were barely visible (arrow) and drained to SOV (arrowhead). (C, D) Patient underwent transorbital embolization by direct puncture of OV. A microcatheter was navigated into the fistula site; hand-injection angiogram demonstrated occlusion of inferior petrous sinus with fistula reflux to pial veins of brainstem (arrowheads). (E) Embolization using detachable coils and Onyx injection led to almost total fistula occlusion. (F) Follow-up MRI 5 months after embolization showed total resolution of his brainstem ischemia and edema. CSDAVF = cavernous sinus dural arteriovenous fistula; MRI = magnetic resonance imaging; OV = ophthalmic vein; SOV = superior ophthalmic vein; T2WI = T2-weighted image.

(Barrow-type D carotid-cavernous fistula). Six of them were slow flow fistulas. In three patients with brainstem ischemia and edema, the fistulas were barely visualized (Fig. 1), largely due to the constraint of fistula drainage. Total occlusion ($n = 6$) or stenosis ($n = 1$) of the IPS was confirmed in seven patients with brainstem ischemia/edema when the catheter systems failed to pass through to the IPSs. Compartment with occlusion of IPS to CS was considered in two patients with successful navigation to IPS, but IPS did not communicate with CS on hand-injection angiography. For six patients, fistula flow shunted to the superior ophthalmic vein (SOV). In two patients who presented with ischemia of the left temporal lobe, fistula flow reflux to the superficial middle cerebral vein was evident (Fig. 2). In seven patients with brainstem ischemia/edema, DSA or hand-injection angiograms demonstrated fistula reflux to the venous structures of the brainstem.

Complete fistula closures were documented on immediate postembolization angiograms in eight aggressive CSDAVF cases. One patient with transarterial particle embolization had residual fistula: however, the patient then underwent radiosurgery treatment. One patient experienced temporary impairment of the sixth cranial nerve because of coil mass effect in the CS; this resolved completely within 4 months. One patient, undergoing transarterial liquid adhesive embolization, had limb paresis; this was presumed to be due to a liquid embolic material flow into the branch of middle cerebral artery via the dangerous external–internal carotid artery. In the six patients who underwent transorbital embolization, there was no evidence of injury of the eyeball or optic nerve, or procedural-related intracranial hemorrhage.

For eight patients with angiographic occlusion of CSDAVFs, the cephalic-ocular symptoms and neurologic deficit related to fistulas such as chemosis, proptosis, limb weakness, and respiratory issues gradually improved after endovascular treatment. No evidence of recurrent or residual fistula was demonstrated in any of these eight patients during neuroimaging follow-up. In one patient undergoing transarterial partial embolization and radiosurgery, in an 8-month clinical and MRA follow-up, limb weakness improved and no new neurologic deficit was found. Additionally, brain MRA demonstrated almost total fistula occlusion.

4. Discussion

The CS is a deep-seated dural sinus. It collects a large amount of venous blood from the superficial and deep venous systems; these include the superior and inferior ophthalmic veins, the sphenoparietal sinus, and the middle cerebral vein. CS drains to the inferior and superior petrous sinuses, and ultimately flows into the internal jugular vein. It also connects inferiorly to the pterygoid plexus. In most CSDAVFs, the fistula flow is usually retrograde, and drains to the IPS or

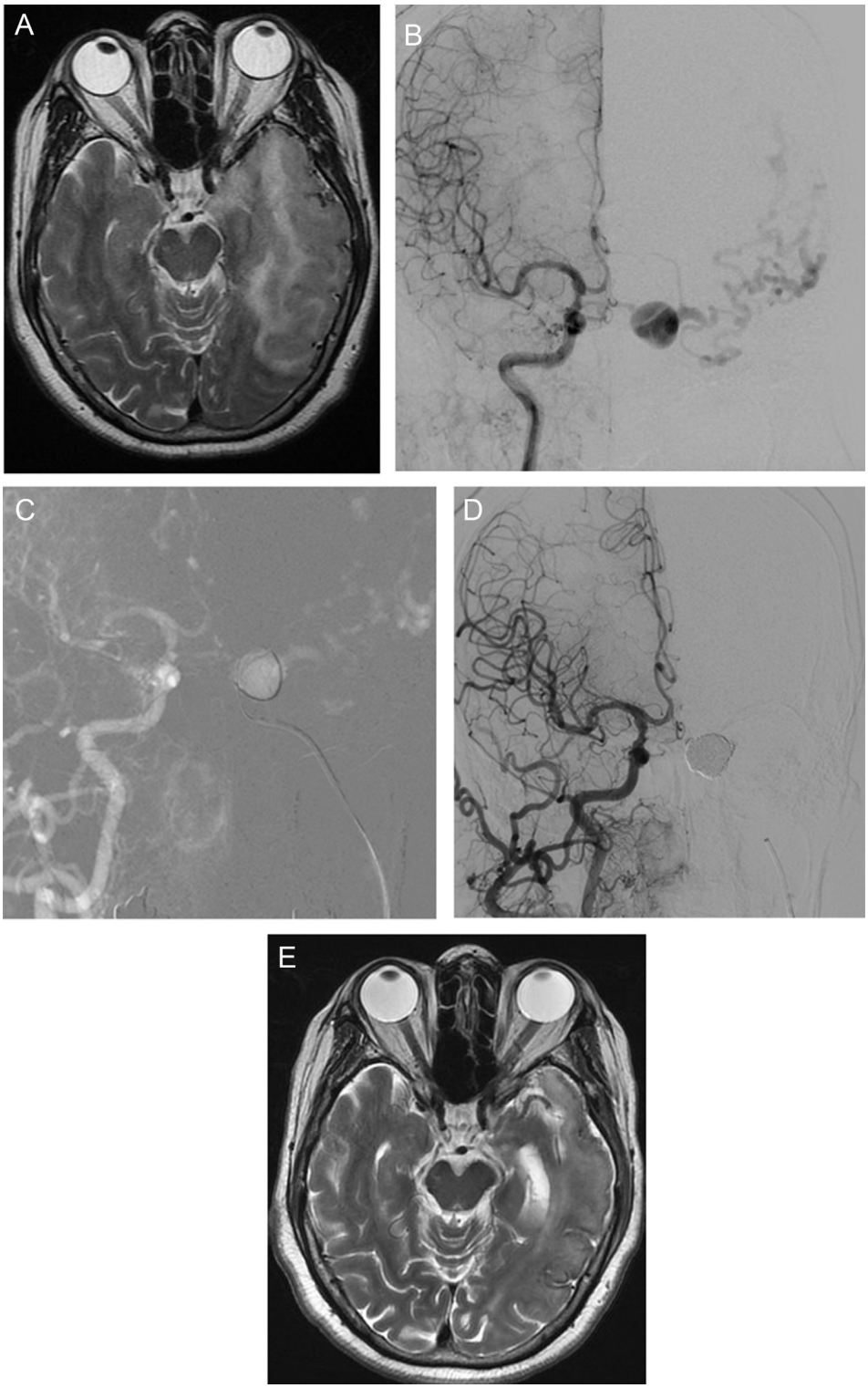

Fig. 2. A 57-year-old woman suffered from right hemiparesis and dysphasia. (A) Brain MRI of T2WI depicted ischemia at the left frontotemporal lobes. (B) Pre-embolization right carotid angiograms showed aggressive CSDAVFs majorly fed by dural branches of right carotid arteries with fistula flow reflux to the left superficial middle cerebral vein; note occlusion of the left IPS. (C, D) Transvenous IPS embolization was performed with successful navigation of the micro-catheter/guidewire into the fistula site; postembolization carotid angiogram demonstrated total fistula occlusion. (E) Follow-up brain MRI on Day 18 after embolization depicted partial regression of left frontotemporal edema. CSDAVF = cavernous sinus dural arteriovenous fistula; IPS = inferior petrous sinus; MRI = magnetic resonance imaging; T2WI = T2-weighted image.

Table 1
Demographics and outcomes of nine patients with clinically aggressive CSDAVFs managed by embolization.

Patient/ sex/age (y)	Clinical manifestations	Neuroimaging findings	Fistula drainage/reflux	IPS	Access route for embolization (access vessel)	Embolic material	Angiographic outcome	Complication	Clinical follow-up (mo)
1/F/57	Chemosis, limb weakness, aphasia	Temporal lobe ischemia	SOV, pial vein	Patent, compartment of IPS−CS	Transvenous (IPS)	Coils	Cure	Nil	11
2/F/69	Chemosis, limb weakness	Brainstem ischemia, edema	SOV, pial vein	Occlusion	Transarterial (AMA)	NBCA	Cure	Pial embolization	25
3/M/62	Chemosis, proptosis, unstable gait	Brainstem ischemia, edema	SOV, pial vein	Occlusion	Transorbital (OV)	Coils, Onyx	Cure	Nil	21
4/F/78	Chemosis, limb weakness, respiratory failure	Brainstem ischemia, edema	Pial veins	Stenosis	Transorbital (CS)	Coils	Cure	Nil	18
5/F/70	Chemosis, limb weakness	Brainstem ischemia, edema	SOV, pial vein	Occlusion	Transorbital (OV)	Coils	Cure	Transient CN6 palsy	8
6/F/73	Chemosis, diplopia, limb weakness	Brainstem ischemia, edema	SOV, pial vein	Occlusion	Transorbital (OV)	Coils	Cure	Nil	19
7/F/66	Bruit, proptosis, limb weakness	Brainstem ischemia, edema	Pial vein	Occlusion	Transorbital (CS)	Coils	Cure	Nil	14
8/F/64	Chemosis, diplopia, limb weakness	Brainstem ischemia, edema	SOV, pial vein	Occlusion	Transorbital (OV)	Coils	Cure	Nil	25
9/M/51	Limb weakness	Temporal lobe ischemia with ICH	Pial vein	Patent, compartment of IPS−CS	Transarterial (AMA)	Particles	Partial embolization	Nil	9

AMA = accessory meningeal artery; CN = cranial nerve; CS = cavernous sinus; CSDAVF = cavernous sinus dural arteriovenous fistula; ICH = intracerebral hemorrhage; IPS = inferior petrous sinus; NBCA = n-butyle-2-cyanoacrylate; OV = ophthalmic vein; SOV = superior ophthalmic vein.

superior petrous sinus leading to bruit or cranial nerve palsy, and/or antegradely refluxes to the ophthalmic vein resulting in chemosis, proptosis, and impairment of visual acuity. Occasionally, the CSDAVFs present with angiographic aggressivity by fistula flow reflux to the cerebral or cerebellar veins.[6] However, angiographically aggressive CSDAVFs presenting with hemorrhagic or nonhemorrhagic neurologic deficit were rare,[7,8] largely because most CSDAVFs are slow-flow fistulas abundant venous connections, without significant fistula outlet constraint.

In patients with CSDAVFs presented with benign and tolerable clinical symptoms without risk of hemorrhagic or nonhemorrhagic stroke, observation or manual compression of the carotid artery or radiosurgery can be recommended.[9,10] By contrast, in cases of angiographically aggressive CSDAVFs with pial reflux and/or those at risk of clinical deterioration such as gradual loss of visual acuity, cranial nerve palsy, or aggressive CSDAVFs, embolization is required. The goal of embolization is to achieve total fistula occlusion or partial embolization for the reversal of aggressive-type DAVFs to benign type, to facilitate less invasive management.

IPS is the major venous drainage of CS, and there are numerous connecting channels between IPSs, basilar venous plexus, vertebral venous plexuses, pterygoid venous plexus, and the epidural vein, and some anatomical variation.[11] Absence of a connection between the IPS and internal jugular vein occurs in 1% of the general population.[12] By far, transfemoral venous access via the IPS is the easiest and most successful route to embolize CSDAVFs with promising results.[13−15] This technique is implemented by placement of a guiding catheter to the internal jugular vein, followed by retrograde catheterization of the IPS and fistula site of the CS. This route is short, direct, and easier to access than other routes; however, trans-IPS navigation of a microcatheter into the CS may be difficult in patients with angiographic occlusive or stenotic IPS.[15] In fact, angiographic occlusion of the IPS can be found in truly occlusive IPS or patent IPS with compartment/occlusion of IPS to CS. In our series, genuinely occlusive IPSs leading to brainstem ischemia and edema were found in six patients. In these aggressive CSDAVFs, successful navigation of the microcatheter to occlusive IPS to CS is almost impossible, because of the long segmental occlusion of the IPS. On the contrary, compartment/occlusion of IPS to CS was considered in other patients, through easy passage of the microcatheter into the IPS, but it posed difficulties while trying to negotiate the microcatheter to the fistula site of the CS because of the septum or short-segmental occlusion between IPS and CS. In our series, five aggressive CSDAVFs were considered slow-flow fistulas, and in case of three aggressive CSDAVFs with brainstem ischemia/edema, the fistula was barely identified on DSA because of significant constraint of venous drainage. However, hand-injection angiograms via a catheter or puncture needle in the CS manifestly demonstrated fistula flow reflux to the brainstem pial veins.

When trans-IPS approach fails to access CSDAVFs, other potential venous approaches, such as the transcontralateral IPS−CS approach, transbasilar plexus approach, transfacial

vein−SOV approach, or transcortical vein approach, are used. Nevertheless, catheterization via these routes may present technical difficulties and usually are time consuming and risky, with possible rupture and thrombosis of venous routes. This is largely because the roadmapping guidance is not available, these fistulas are isolated without connection with these venous channels, or the cortical veins are too long and tortuous to access.

Transarterial embolization is another option for management of CSDAVFs. Although some studies have shown good outcomes with low complication rates using transarterial occlusion of CSDAVFs with few dural feeders by delivery of liquid embolic agents[16] or particles, the majority of CSDAVFs performed are frequently inadequate to achieve an angiographic cure by the transarterial route. This is because most CSFDAVFs are fed by numerous and tiny dural branches of carotid arteries that cannot be successfully accessed or embolized. Furthermore, some risks may occur by inadvertent embolization of the carotid territory with ischemic stroke via dangerous anastomosis of the external−internal carotid artery or embolization of feeders of the cranial nerves. This complication occurred in one patient of our series. Therefore, to avoid embolic material flow from the dural branches of the external-to-internal carotid artery, protective balloon navigation and inflation to cavernous ICA during injection is a feasible method.

Transorbital access to embolization of CSDAVFs is the most invasive modality of embolization. This technique was first introduction by Teng et al,[17] for treatment of direct carotid-cavernous fistulas with difficult fistula anatomy. Subsequently, some intermittent sporadic case reports had been published utilizing transorbital technique to manage CSDAVFs or direct carotid-cavernous fistulas.[17−19] This access was considered an alternative technique and selected only when other approaches failed. This technique can be performed by surgical exposure of the distal SOV, followed by needle puncture of the exposed SOV. The advantage of this access is easy puncture of the exposed SOV with less risk of injury to the intraorbital structure. The potential disadvantage is surgical-related bleeding, infection, and scarring of the exposure site. Another concern is the potential risk of dislodgement of the catheter−wire system during advancement, leading to hemorrhage because of tortuous distal parts of the SOV. Other transorbital accesses can be achieved by direct needle puncture of the ophthalmic vein or CS under fluoroscopic guidance. Although the superficial location involves palpable access to the distal SOV, direct needle puncture of the distal SOV under fluoroscopic or sonographic guidance may be associated with dislodgement of catheter systems, leading to orbital hemorrhage as referenced earlier. In our series, we preferred to undertake direct puncture of the OV or CS due to superior needle support with less risk of dislodgement of the catheter system. The potential risks of this method are injury to the eyeball and optic nerve, and orbital or subarachnoid hemorrhage. To avoid these complications, a high-quality roadmapping angio-machine, a detailed understanding of the orbital anatomy and CS, as well as meticulous manipulation of the puncture needle were crucial. Owing to the superior location of the optic canal/optic nerve in comparison to the inferior location of the superior orbital fissure/ophthalmic vein, we preferred to select the inferior orbital rim as a puncture site. Prior to initiating the procedure, the eyeball was pushed to a superior position. The needle direction was adjusted intermittently to target the superior orbital fissure. In our series, we utilized a smaller-caliber puncture needle (18 gauge), due to the prospect of reduced orbital injury and bleeding during needle penetration. Another concern of transorbital access is the hemostasis after embolization. To reduce the risk of intraorbital hemorrhage, heparin should be reversed before removal of the puncture needle. In our series, we successfully accessed the fistula site by direct puncture of the OV ($n = 4$), anterior CS ($n = 1$), or posterior CS ($n = 1$, Case 4).[19] One patient underwent direct puncture of the posterior CS, largely due to a low-flow fistula, which was barely visible on the posterior CS of the fistula site without opacification of the SOV, OV, or anterior CS. The major concern and risk of puncture of the posterior CS is overpenetration of the puncture needle leading to perforation of the nearby dura, resulting in subarachnoid hemorrhage.

In conclusion, aggressive CSDAVFs presenting with hemorrhagic or nonhemorrhagic neurologic deficit were uncommon. In this limited case series, most cases of CSDAVFs occurred in the brainstem with ischemia/edema or presented in the context of cerebral nonhemorrhagic or hemorrhagic stroke. Embolization, by way of various access routes, is a feasible and effective method to manage these aggressive CSDAVFs, with a high rate of success and an acceptable level of periprocedural risk.

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