Original Article

Endovascular Treatment for Traumatic Carotid Cavernous Fistula: Case Series

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Objective: Traumatic carotid-cavernous fistula (TCCF) is a rare neurovascular condition that occurs after blunt head trauma. This condition accounts for approximately 4% of traumatic cerebrovascular injuries. Various symptoms can be observed in TCCF, and aggressive treatment is frequently required. Herein, we reviewed the treatment of TCCF in our hospital.

Methods: We retrospectively reviewed patients with TCCF between December 2021 and May 2023. The physical findings, clinical images, and surgical details of patients were investigated.

Results: Three men and 1 woman were included. Only 1 case was diagnosed with CCF using initial 3D-CTA; the other 3 were diagnosed after admission using DSA. All patients received endovascular treatment; 2 were initially treated with transarterial embolization, and the other 2 were treated with transvenous embolization, although 1 case of transarterial embolization required additional treatment with transvenous embolization. Complete occlusion was achieved in all cases. Two of the cases were accompanied by skull base fractures, both of which were middle fossa fractures.

Conclusion: TCCF is caused by direct injury to the internal carotid artery and can be accompanied by skull fractures or vessel wall damage as a result of shear force. We should suspect TCCF, especially when a skull base fracture is detected, even if the initial 3D-CTA shows no evidence of TCCF. Treatment for TCCF is mainly endovascular; however, the specific treatment approach should be determined for each case based on various factors, including vessel anatomy.

Keywords head trauma, endovascular treatment, carotid-cavernous fistula

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Introduction

Traumatic carotid-cavernous fistula (TCCF) is a relatively rare condition that can accompany blunt head trauma; it accounts for approximately 4% of all traumatic cerebrovascular injuries.[1)] Various symptoms are often observed in TCCF, including orbital bruit, exophthalmos, and chemosis. Some cases are accompanied by intracerebral or subarachnoid hemorrhage. Most cases present with a direct shunt and require aggressive therapeutic interventions. We reviewed the treatment of TCCF in our hospital.

Materials and Methods

The study was approved by the ethics committee of NHO Osaka National Hospital (approval number: 23022) and performed in accordance with its guidelines. Informed consent was obtained using the opt-out method; eligible patients were able to decline participation via the ethics committee's website.

Of the 303 patients transported to our hospital for blunt head trauma between December 2021 and May 2023, 4 patients were diagnosed with TCCF and underwent endovascular treatment. The diagnoses were mainly made using CT, 3D-CTA, and DSA. 3D-CTA is preferred over MRA on admission because it takes a shorter time than MRA, especially if problems such as renal dysfunction or an allergic response to contrast occur. In our institution, patients with blunt head trauma usually undergo 3D-CTA on admission to our department. However, this technique is sometimes insufficient for detecting small vascular lesions. We therefore also perform DSA in many patients, especially those with intracranial hemorrhage or skull fractures; this usually occurs within 1 week of admission, even if no lesions are detected by 3D-CTA. In the present study, we retrospectively collected the clinical characteristics of these patients, including age, sex, Glasgow Coma Scale score at admission, CT findings at admission, initial management for blunt head trauma, TCCF symptoms, DSA findings, endovascular treatment details, treatment results, follow-up period, and modified Rankin Scale score at 30 days.

Results

The mean age of the 4 patients was 46.3 (range, 40–72) years, and 3 of the patients were men (**Table 1**). Plain CT of the head on admission revealed skull base fractures in 2 patients and intracranial hemorrhage—such as acute epidural hematoma, acute subdural hematoma, or traumatic subarachnoid hemorrhage—in 3 patients. Only 1 of the 4 patients was diagnosed with TCCF using 3D-CTA on admission; the other 3 patients were diagnosed with TCCF for the first time using DSA during hospitalization. One patient presented with preoperative tinnitus, 1 patient presented with an eye movement disorder, and 1 patient presented with convulsions. One patient had accompanying diffuse subarachnoid hemorrhage, and another patient had accompanying temporal lobe hemorrhage and subarachnoid hemorrhage as a result of TCCF. The initial treatment was transarterial embolization (TAE) in 2 patients and transvenous embolization (TVE) in 2 patients, with no cases of parent artery occlusion. One TAE case received additional TVE because of worsening shunt blood flow on postoperative day 2. Complete occlusion was achieved in all 4 cases. The modified Rankin Scale score at discharge was 5 in 1 patient only; this patient was transferred to a convalescent hospital. One patient experienced abducens nerve palsy as a postoperative complication; however, this gradually improved. There was no recurrence in any patient after the last treatment. The average hospital stay was 50 days. Except for heparinization during the procedure, antithrombotic therapy was not administered in any of the cases, and no cases had ischemic complications after treatment. A representative case (Case 1) is described in the following section.

Representative case

A 40-year-old man was injured in an accidental fall and transferred to our hospital. Although he was in cardiopulmonary arrest when he arrived, his heartbeat resumed on its own. A head CT revealed a left acute subdural hematoma, left acute epidural hematoma, and multiple skull fractures, including a fracture of the left middle skull base (**Fig. 1A** and **1B**). Furthermore, 3D-CTA on admission revealed venous depiction in the arterial phase around the right internal carotid artery (**Fig. 1C**), and TCCF was suspected. We performed a decompression craniotomy for the left acute subdural hematoma on the same day, and the patient was maintained under sedation after the surgery.

Six days after admission, DSA revealed a left CCF and a pseudoaneurysm in the cavernous portion of the left internal carotid artery (**Fig. 2**). The fistulous flow drained into the contralateral cavernous sinus via the intercavernous sinus, contralateral superior orbital vein, contralateral inferior petrosal sinus, and contralateral sphenoparietal sinus (**Fig. 3**). It was assumed that a rupture of the pseudoaneurysm might have caused the CCF; that is, the aneurysm may have originally been located in the cavernous portion, and its rupture may have caused the CCF. Moreover, the ipsilateral venous system was thought to have been disrupted by the fracture, which may explain why much of the blood flow from venous drainage drained to the contralateral side. Partly because reflux into the cortical vein or deep venous system was not observed and partly because the patient's vital signs remained unstable, we did not plan any TCCF treatment immediately after diagnosis. However, 17 days after admission, a follow-up head CT revealed a diffuse subarachnoid hemorrhage (**Fig. 4A**). DSA showed that the pseudoaneurysm had increased in size and that blood flow from the TCCF was

Table 1 Summary of the 4 traumatic CCF cases

			Case 1 (Representative case)	Case 2	Case 3	Case 4
Age/sex			40/M	72/M	32/M	41/F
Initial Glasgow Coma Scale score			3	15	15	11
CT findings	Plain		ASDH, AEDH, t-SAH Skull/skull base fracture	No specific findings (initial) SAH, ICH (when onset)	t-SAH Skull fracture	ASDH, t-SAH Skull/skull base fracture
	3D-CTA		CCF	Not implemented (initial) No specific findings (when onset)	No specific findings	No specific findings
Initial treatment			Decompressive craniotomy	Conservative	Conservative	Conservative
CCF pathology	Symptoms		Unknown	Convulsion	Tinnitus	Eye movement disorder
	Draining vein	Ipsilateral	SMCV	SOV, SMCV, BVR, frontal vein	SOV	SOV, PPx
		Contralateral	SOV, SpPS, IPS	–	IPS	IPS
	Cortical vein reflux		+	+	–	–
CCF treatment	Interventional radiology		TAE	TAE TVE	TVE	TVE
	Outcome		Complete occlusion	Complete occlusion	Complete occlusion	Complete occlusion
	Complication		None	None	Abducens nerve palsy	None
mRS 1 month after last treatment			5	0	2	1

AEDH, acute epidural hematoma; ASDH, acute subdural hematoma; BVR, basal vein of Rosenthal; CCF, carotid-cavernous fistula; F, female; ICH, intracerebral hemorrhage; IPS, inferior petrosal sinus; M, male; mRS, modified Rankin Scale; PPx, pterygoid plexus; SMCV, superficial middle cerebral vein; SOV, superior ophthalmic vein; SpPS, sphenoparietal sinus; t-SAH, traumatic subarachnoid hemorrhage; TAE, transarterial embolization; TVE, transvenous embolization

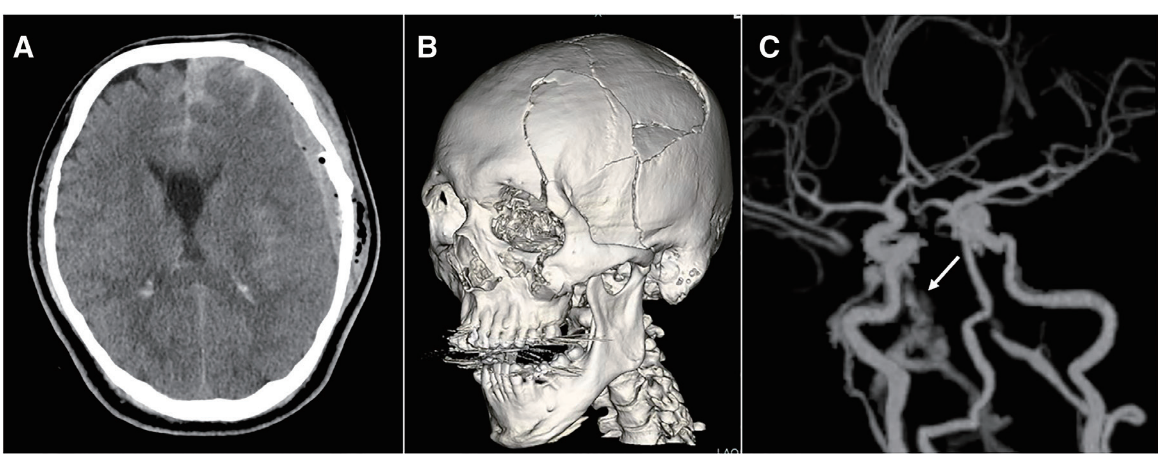

Fig. 1 Images from Case 1 on arrival at the hospital. (**A** and **B**) Initial CT when the patient was transferred. A left acute subdural hemorrhage and multiple skull fractures were detected. (**C**) Initial 3D-CTA. The venous system was depicted around the right internal carotid artery (arrow), which implied the emergence of shunt flow.

backed up into the ipsilateral superficial middle cerebral veins (**Fig. 4B–4E**). We therefore performed emergency endovascular treatment for these lesions on the same day.

We inserted an 8-Fr long sheath into the left femoral artery and placed a guiding catheter (8-Fr FUBUKI; ASAHI INTECC, Aichi, Japan) into the cervical portion of the left internal carotid artery. We then used a distal accessing catheter (Vecta71; Stryker, Kalamazoo, MI, USA) to provide additional support. A microcatheter (Excelsior SL-10 45°; Stryker) was placed in the aneurysm using a microguidewire

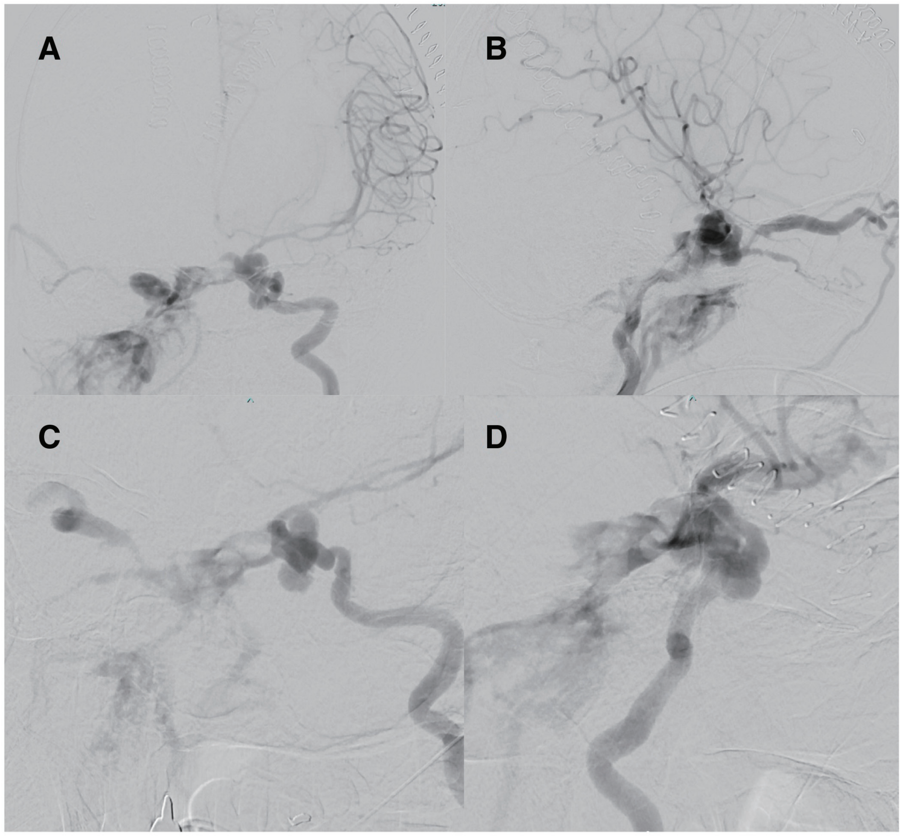

Fig. 2 DSA findings from Case 1. (**A–D**) DSA 6 days after admission. In left internal carotid angiography, a left CCF was detected. This was accompanied by aneurysmal dilation in the C3/4 section, which was considered to be a traumatic pseudoaneurysm. CCF, carotid-cavernous fistula

(Synchro SELECT soft pre-shaped; Stryker), and a balloon catheter (TransForm C 4 × 10 mm; Stryker) was placed to cover the neck of the aneurysm (**Fig. 4F**). The fistula and pseudoaneurysm were embolized with 11 detachable coils using balloon assistance (**Fig. 4G**).

Immediately after embolization, fistulous flow remained into the intercavernous sinus only (**Fig. 4H** and **4I**). Although DSA at 1 month after admission showed fistulous flow through the intercavernous sinus into the contralateral inferior petrosal sinus and sphenoparietal sinus, DSA at 2 months after admission showed that inflow into the venous system had completely disappeared (**Fig. 5**). Three months after admission, the patient was transferred to another hospital with a modified Rankin Scale score of 5.

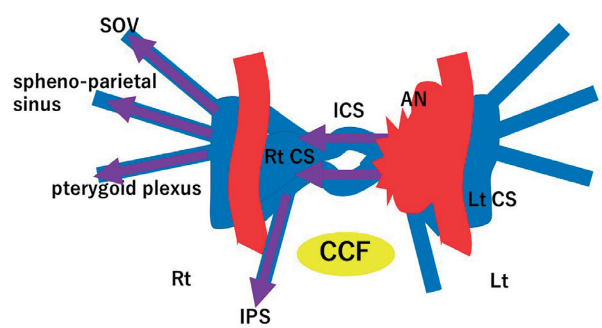

Fig. 3 Schema of the hemodynamics of the CCF in the representative case (coronal view). The left CCF refluxed into the contralateral cavernous sinus via the intercavernous sinus and drained into the contralateral SOV, sphenoparietal sinus, pterygoid plexus, and IPS. AN, aneurysm; CCF, carotid-cavernous fistula; CS, cavernous sinus; ICS, intercavernous sinus; IPS, inferior petrosal sinus; Lt, left; Rt, right; SOV, superior orbital vein

The detailed medical histories of Cases 2–4 are omitted, but a summary of their angiographic findings and a discussion of their treatment plans are described in the following text. In Case 2, the patient developed convulsions and coma on the day of admission; plain CT immediately after the attack showed intracranial hemorrhage. We therefore performed DSA on the same day and administered initial treatment straight after the DSA. The first DSA showed right CCF accompanied by reflux into the right superficial middle cerebral vein, basal vein of Rosenthal, right superior ophthalmic vein, pterygoid plexus, basilar plexus, and other cortical veins and the deep venous system. A balloon catheter (TransForm SC 7 × 7 mm; Stryker) was placed

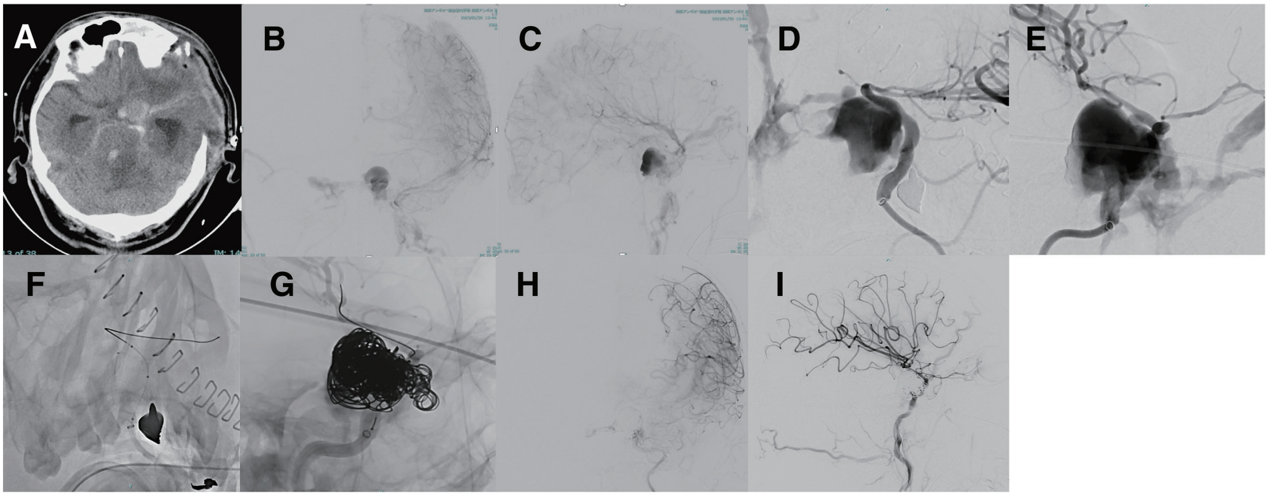

Fig. 4 Images of Case 1 during the procedure, 17 days after admission. (**A**) Plain CT 17 days after admission revealing a diffuse subarachnoid hemorrhage. (**B–I**) Images taken during the operation, 17 days after admission. (**B–E**) Left ICA angiography before embolization, showing findings consistent with a CCF and a large aneurysm. (**F**) Excelsior SL-10 45° (Stryker, Kalamazoo, MI, USA) was placed in the aneurysm, and TransForm C 4 × 10 mm (Stryker) was placed in the left ICA to cover the neck of the aneurysm. (**G**) Eleven coils were filled in the aneurysm. (**H** and **I**) Final angiography during the operation showing complete occlusion of the aneurysm, although a slight drain into the intercavernous sinus remained. CCF, carotid-cavernous fistula; ICA, internal carotid artery

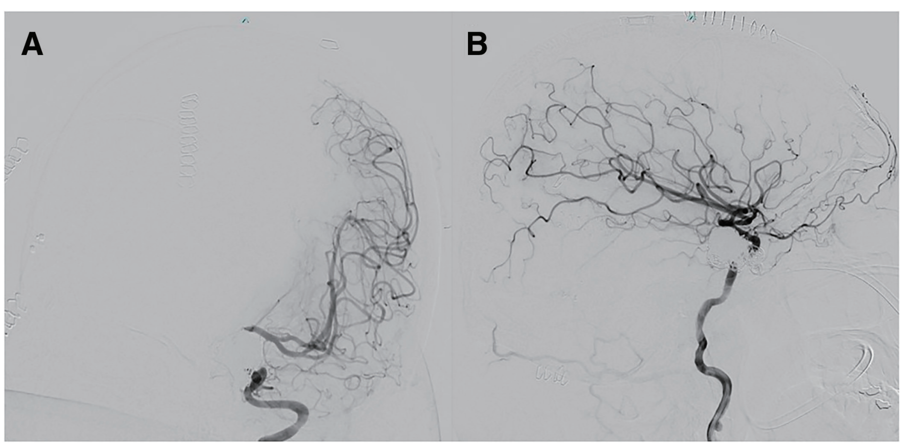

Fig. 5 DSA 2 months after the operation. Neither the CCF nor the aneurysm remained. (**A**) A–P view and (**B**) lateral view. CCF, carotid-cavernous fistula; A–P, anterior posterior

just proximal to the CCF, and left internal carotid artery angiography was conducted, which revealed a fistula in the cavernous portion of the right internal carotid artery. A microcatheter (Excelsior SL-10 45°; Stryker) was placed in the cavernous sinus through the fistula using a manually shaped microguidewire (Synchro SELECT standard straight; Stryker). The cavernous sinus was embolized with 13 detachable coils, resulting in the disappearance of reflux into the right superficial middle cerebral vein, basal vein of Rosenthal, and right superior ophthalmic vein; we thus finished the procedure. However, 2 days after embolization, reflux into the right superior ophthalmic vein recurred. We therefore planned another embolization. Although the same method was attempted, the microcatheter (Excelsior SL-10 45°; Stryker) was unstable because of the coil mass, and we changed our plan to a TVE procedure. The same microcatheter was placed proximal to the right superior ophthalmic vein through the cavernous sinus. The right superior ophthalmic vein and cavernous sinus were embolized with 50 detachable coils. Consequently, reflux into the right superior ophthalmic vein disappeared, and flow into the cavernous sinus decreased. One week after the second session, DSA revealed the complete occlusion of the CCF.

In Case 3, ipsilateral tinnitus developed 4 days after admission, and DSA was performed 2 days after these symptoms developed. The left CCF was accompanied by reflux into the left superior ophthalmic vein, inferior petrosal sinus, and contralateral cavernous sinus. TVE was performed 11 days after the DSA. One week later, TVE was planned. Immediately before the procedure, new reflux into the superior petrosal sinus emerged. A microcatheter (Excelsior SL-10 45°; Stryker) was placed in the left superior ophthalmic vein using a microguidewire (Synchro SELECT soft pre-shaped; Stryker). The left superior ophthalmic vein was then embolized with 15 detachable coils, and the left superior petrosal sinus was embolized with the same microcatheter and 11 detachable coils. Consequently, reflux into the intercavernous sinus increased, and the intercavernous and left cavernous sinuses were embolized with 12 detachable coils.

In Case 4, abducens nerve palsy developed the day after admission. We performed DSA 6 days after admission, which showed CCF, and conducted TVE 10 days after admission. Left CCF was accompanied by reflux into the left superior ophthalmic vein, superior petrosal sinus, inferior petrosal sinus, contralateral cavernous sinus, pterygoid plexus, and vertebral venous plexus. Initially, TAE was planned. Right internal carotid artery angiography was conducted using a balloon catheter (Scepter C 4 × 10 mm; Terumo, Tokyo, Japan) placed in the cavernous portion of the left internal carotid artery to identify the fistula; however, we were unable to observe it. The plan was therefore changed to TVE, and a microcatheter (Excelsior SL-10 45°; Stryker) was placed into the left superior ophthalmic vein through the left cavernous sinus. Although coils were embolized from the left superior ophthalmic vein to the left cavernous sinus, reflux into the left superior middle cerebral vein gradually increased. The reflux persisted after tightly packing the cavernous sinus with 36 detachable coils; however, it flowed into the left transverse sinus through the vein of Labbé. We therefore thought that the possibility of an immediate intracerebral hemorrhagic complication or intracranial pressure increase was low, and we terminated the procedure. One year later, an MRI showed that the CCF had disappeared.

Discussion

TCCF is a rare complication of blunt head trauma. Its occurrence can be attributed to direct damage to blood vessels caused by fractures or other causes, or to damage to the vessel wall because of shear forces.[2–4] In skull base fractures in particular, TCCF has been reported to occur in 8.3% of middle skull base fractures, 2.4% of anterior skull base fractures, and 1.7% of posterior skull base fractures.[2] Generally, it is considered that 0.17% to 0.27% of head and facial trauma cases are complicated by TCCF.[5] Thus, the probability of a TCCF complication is higher if a patient has skull base fractures, especially middle skull base fractures. It has been reported that TCCF onset may occur days to months after the injury.[6,7] Symptoms appear within 24 hours of injury in 30% of cases, within 1 week in 15% of cases, and approximately 2 months after injury in half of all cases.[6,7] It has been reported that a shorter time between symptom onset and treatment is associated with a lower likelihood of complications from TCCF.[2]

In our institution, we perform treatment as soon as possible after symptoms develop or dangerous CCF is detected using DSA. In our representative case (Case 1), multiple traumatic injuries occurred, and the patient required systemic treatment as a priority, which delayed DSA and consequently the treatment of TCCF. In Case 2, because there were no specific findings on plain head CT at the time of admission, 3D-CTA was not performed. As a result, the TCCF was not identified until the onset of intracranial hemorrhage, although this occurred on the same day. By contrast, the other 2 cases were successfully treated before hemorrhagic events occurred. We therefore believe that, using our protocol, it may be possible to detect and treat TCCF before it develops.

TCCF is almost always a direct type of CCF (Barrow type A) and rarely resolves spontaneously, meaning that it requires active treatment.[7–9] When considering the rate of direct-type shunts in TCCF, 1 report of 13 TCCF cases included 11 direct types and 2 indirect types. The 2 indirect types were accompanied by a feeder of the meningeal branch of the external carotid artery. Furthermore, all cases with direct-type shunts were accompanied by high-flow shunts, whereas those with indirect-type shunts were accompanied by low-flow shunts.[10] There is no specific definition to distinguish high-flow shunts from low-flow shunts, meaning that their differentiation is subjective. Generally, a high-flow CCF fills the cavernous sinus and efferent veins within a fraction of a second; under such conditions, the intracranial branches of the internal carotid artery partially fill or cannot be visualized.[7] A few cases of spontaneous resolution have also been reported; Iampreechakul et al.[11] described 9 cases with the spontaneous resolution of direct CCF. These authors noted that thrombosis of the cavernous sinus or venous drainage may be related to the spontaneous resolution of direct CCF. It has also been reported that possible mechanisms of spontaneous resolution may be local embolization caused by changes in pressure differences between the internal carotid artery and cavernous sinus as a result of catheter placement or contrast agent injection, and a valve mechanism in the aneurysmal wall (traumatic pseudoaneurysm) or fistula.[12,13]

TCCF has been reported to cause ocular protrusion, conjunctival hyperemia, a pulsating sound, diplopia because of ocular motility disturbance, and epistaxis. These symptoms are relatively common, whereas intracranial hemorrhage as a result of cortical reflux is rare. Specifically, the incidence of intracranial hemorrhage caused by cortical vein reflux is reportedly between 0.9% and 2.6%.[14] As in our representative case, subarachnoid hemorrhage has been reported as a complication of TCCF.[15] Cho et al. reported 1 case of subarachnoid hemorrhage as a result of CCF caused by the rupture of a pseudoaneurysm.[16] In this report, a pseudoaneurysm in the patient was treated with coil embolization, and complete occlusion was achieved. Moreover, in another report, a CCF formed a varix that ruptured ventral to the prepontine cistern, resulting in delayed subarachnoid hemorrhage.[17]

Endovascular embolization is the main treatment method for TCCF. Previous reports indicate that there is no significant difference between TAE and TVE in terms of the complete occlusion rate.[18] In a 2021 systematic review,[18] the rate of complete occlusion with TAE was 93.9%; however, many of the included cases were treated with detachable balloons, which are not currently used. The cases treated with detachable coils only achieved a complete occlusion rate of 92.6%. The TAE-treated group also included cases treated with a covered stent, stent and coils, coils and Onyx (Medtronic, Irvine, CA, USA), and polyvinyl alcohol and a gelatin sponge, all of which were reportedly effective. In the TVE group, the complete occlusion rate was 91.67%. Most of the TVE-treated cases were treated with detachable coils only. Both TAE and TVE are therefore associated with a high rate of fistula obliteration.[18]

In our institution, we consider both TAE and TVE before choosing the easiest one (after discussing its approach route, etc.). If the arterial access route is difficult because of factors such as artery tortuosity, or if it is difficult to identify the fistula or set the optimal working angle, TVE is selected. Furthermore, in cases where TAE is dangerous because of vascular conditions (e.g., vascular Ehlers–Danlos syndrome), TVE is selected.[19] Conversely, in re-treatment after TAE (e.g., in patients such as Case 2), the coil mass often prevents an approach from the artery; in such cases, we choose TVE.

Detachable balloons were traditionally used for the treatment of TCCF with high occlusion rates; however, they sometimes resulted in unexpected events such as early detachment, premature deflation, or puncture of a fractured fragment. Currently, detachable balloons are not available, and coil embolization is the main treatment. Nonetheless, problems associated with coil embolization include its high cost, occlusion of the main venous drainage, and cranial nerve palsy. We must therefore be careful with respect to these possible complications during coil embolization, whether via TAE or TVE.[20] The present study had some limitations. For example, the present study included 4 cases who were all examined and treated at 1 institution. Its results and their interpretation therefore cannot be generalized because of the small sample size and single-center approach.

Conclusion

We reviewed 4 cases of endovascular treatment for TCCF. TCCF may be associated with skull base fractures; thus, if the initial CT reveals this pathology, careful follow-up with DSA imaging may be desirable. The outcome of endovascular treatment for TCCF is reportedly quite good, and the choice between TAE and TVE should be considered carefully for each case based on vascular anatomy and ease of access. For cases in which TAE is difficult or impossible because of an unidentifiable fistula or difficult access routes, TVE may be a possible solution.

Disclosure Statement

The authors declare that they have no conflicts of interest.

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