

# Transcatheter Embolization Combined with Surgical Resection for Traumatic Middle Meningeal Artery-vein Fistula: A Case Report

Shengshan Li, Shuzhi Li\*, Guohua Liu, Yong Zhang, Bin Wang, Jiawei Chai, Dawei Ren, Dunyong Mou,

#### Xin Xu

Department of Neurosurgery, Gaomi Hospital of Traditional Chinese Medicine, Gaomi 261500, Shandong, China

\*Author to whom correspondence should be addressed.

**Copyright:** © 2025 Author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0), permitting distribution and reproduction in any medium, provided the original work is cited.

**Abstract:** Traumatic carotid-cavernous arteriovenous fistula (TCCAVF) is a rare but severe cerebrovascular disorder, often resulting from head trauma with temporal bone fractures. The pathogenesis involves vessel wall injury due to traction, frequently associated with fractures near the middle meningeal artery. This case highlights the typical clinical presentation, diagnostic approaches, and therapeutic management of TCCAVF, emphasizing the challenges in treating this condition. Transcatheter embolization proved effective in occluding the fistula, underscoring its role as a key intervention for traumatic meningeal arteriovenous fistulas.

Keywords: Traumatic carotid-cavernous arteriovenous fistula; Transcatheter embolization; Traumatic meningeal arteriovenous fistula

Online publication: April 30, 2025

#### **1. Introduction**

Traumatic carotid-cavernous arteriovenous fistula (TCCAVF) is a rare but potentially life-threatening cerebrovascular disease, typically caused by head trauma, especially cases involving temporal bone fractures. It is speculated that traction injury to the vessel wall leads to the formation of arteriovenous fistulas, and its pathogenesis is mostly associated with fractures adjacent to the middle meningeal artery <sup>[1, 2]</sup>.

This case demonstrates the typical clinical manifestations, diagnostic methods, and treatment strategies of TCCAVF, while also reflecting the complexity and treatment challenges of this disease.

#### 2. Case overview

A 50-year-old female patient was admitted to the hospital due to "head trauma with unconsciousness for 2 hours caused by a car accident." Physical examination on admission revealed coma, GCS score: 1 + 1 + 1 = 3, scalp hematoma on the right forehead, bilateral dilated and fixed pupils with a diameter of approximately 6mm, and absent pupillary light reflex. CT examination suggested acute subdural hematoma (left), subarachnoid hemorrhage, a small amount of intracranial air accumulation, skull fracture, skull base fracture (right), scalp hematoma; right middle ear mastoid hematoma; right zygomatic fracture; multiple rib fractures on the right side; right clavicle comminuted fracture. As the patient had cerebral hernia, preoperative preparations were completed after admission, and emergency left craniotomy hematoma removal, and decompressive craniectomy were performed. The operation was successful, and the patient recovered well, with clear consciousness and no functional disability. Approximately 60 days after the injury, a titanium mesh cranioplasty was performed, and the operation was smooth. About 75 days after the injury, the patient developed pulsatile tinnitus. No special conditions were found after consultation with otolaryngology. Auscultation of the temporal and parietal regions.

Figure 1 shows the cerebrovascular DSA examination of the patient. Figure 1(a) revealed a right dural arteriovenous fistula. During the operation, a spring coil combined with ONYX glue embolization was immediately applied, and the dural arteriovenous fistula was satisfactorily occluded (Figure 1(b)). The patient's tinnitus symptoms disappeared. About 120 days after the injury, the patient again developed pulsatile tinnitus. Cerebrovascular angiography showed recurrence of the dural arteriovenous fistula (Figure 1(c)). Preoperative preparations were completed, and craniotomy for dural arteriovenous fistula resection was performed under general anesthesia. During the operation, the accompanying veins of the dural arteries were dilated and the wall was thickened (Figure 1(d)), showing arterialization, and the surrounding skull was compressed into a groove-like indentation (Figure 1(e)). An arcuate incision was made around the arterialized vein to open the dura mater, block the blood flow, and the artificial dura mater was sutured to repair the dura mater. The bone flap was repositioned, and the subcutaneous tissue and skin were sutured in layers. The operation was successful. The patient did not experience tinnitus symptoms after the operation. More than 180 days after the operation, cerebrovascular angiography showed no recurrence of the dural arteriovenous fistula (Figure 1(f)).

# 3. Pathogenesis and pathophysiology

The formation of TCCAVF is a result of the combined effects of trauma, vascular injury, and imbalance in local repair mechanisms. During craniocerebral trauma, the mechanical stress of temporal bone fractures can directly tear the vessel walls of the middle meningeal artery and its accompanying veins, forming an arteriovenous shunt. Under normal circumstances, there is a vessel wall between the middle meningeal artery and vein, but in TCCAVF, this vessel wall is destroyed, leading to the formation of an abnormal direct connection between the artery and vein. The formation of TCCAVF is mainly related to trauma-induced vessel wall damage, especially direct tearing or traction injury of the middle meningeal artery and its accompanying veins.

Arterial blood bypasses the normal blood flow pathway, directly flows into the veins and reaches the venous sinuses, leading to increased overall venous pressure in the brain tissue, impeded blood flow, and potentially causing cerebral edema, intracranial hemorrhage, or neurological deficits <sup>[1, 2]</sup>. In this case, the patient's temporal bone fracture was located in the region of the middle meningeal artery, and it is speculated



**Figure 1.** Cerebrovascular DSA examination of the patient; (a) The arrow indicates the location of the dural arteriovenous fistula. Above the arrow is the dural vein draining into the sagittal sinus, and below the arrow is the thickened dural artery; (b) The arrow indicates no blood flow after embolization surgery combining a spring coil with ONYX glue; (c) The arrow indicates recurrence of the dural arteriovenous fistula, and the dot above is a spherical marker for the location of the draining vein; (d) The arrow indicates thickening of the dural vein and thickening of the blood vessel wall; (e) The arrow indicates the impression of the arterialized vein on the skull flap; (f) The arrow indicates the disappearance of the dural artery and dural arteriovenous fistula, with no blood flow.

that fracture fragments or shear forces during trauma caused damage to the vessel wall, resulting in the formation of an arteriovenous fistula. Additionally, local inflammatory responses and vascular remodeling after trauma may further promote the formation and expansion of the fistula <sup>[3]</sup>. It should be noted that the hemodynamic changes in TCCAVF are bidirectional. On one hand, arterial blood directly enters the venous system, causing local venous hypertension, which may lead to cortical venous dilation and, in severe cases, rupture and bleeding. On the other hand, the brain tissue distal to the fistula is affected by the "steal phenomenon", resulting in hypoperfusion, neurological dysfunction, or epileptic seizures. The recurrence of the

fistula in this case after initial embolization may be related to neovascularization around the fistula.

#### 4. Diagnosis and imaging evaluation

The diagnosis of TCCAVF combines clinical features and multimodal imaging examinations. Apart from typical pulsatile tinnitus, approximately 30% of cases can be affected by cortical venous hypertension, presenting with focal neurological deficits such as limb weakness and language disorders. Other symptoms of TCCAVF include headache, epileptic seizures, and visual changes. In this case, the patient presented with tinnitus as the initial symptom, but had no other neurological abnormalities, possibly due to the location of the fistula being away from functional venous sinuses.

Digital subtraction angiography (DSA) is the gold standard for diagnosing TCCAVF, as it can clearly show the location of the fistula, feeding arteries, and draining veins, and evaluate hemodynamic changes <sup>[4]</sup>. DSA was performed in this patient, confirming the location of the fistula in a distal branch of the middle meningeal artery and revealing the characteristic "early-appearing vein" sign, which helped doctors determine the nature and severity of the fistula and provided a strong basis for formulating later treatment strategies. Additionally, computed tomography angiography (CTA) and magnetic resonance angiography (MRA) can serve as screening tools, but they have lower sensitivity for detecting small fistulas <sup>[5]</sup>. CTA can rapidly screen for fractures and hematomas, but its sensitivity for low-flow fistulas is inadequate. The initial CTA examination in this case did not reveal abnormalities, possibly due to the incomplete formation of the fistula. Magnetic resonance venography (MRV) can clearly show venous sinus dilation and cortical venous tortuosity, but its ability to display the feeding arteries of the fistula is limited. In this case, the patient had a temporal bone fracture located in the region of the middle meningeal artery, and the formation of TCCAVF was related to direct injury of the vessel wall.

Based on the history of trauma and the presence of a clear blowing-like bruit on auscultation over the temporoparietal region, the diagnosis of TCCAVF was suspected and confirmed by DSA, which revealed a dural arteriovenous fistula located in the right middle meningeal artery region. In recent years, clinical applications of high-resolution three-dimensional rotational DSA technology have enabled stereoscopic localization of fistulas, combined with hemodynamic parameters such as pressure gradient measurements, to further optimize the placement of embolization materials. Additionally, intraoperative indocyanine green fluorescence angiography (ICG) allows real-time evaluation of fistula occlusion effects, thereby reducing the risk of postoperative recurrence.

# 5. Treatment strategy and efficacy analysis

Traumatic carotid-cavernous arteriovenous fistulas have been shown to progressively grow in repeated cerebrovascular angiography, and they are one of the causes of delayed intracranial hemorrhage <sup>[3]</sup>. Furthermore, pulsatile intracranial noise significantly affects patients' rest. Therefore, most patients require active treatment. The treatment goal of TCCAVF is to completely occlude the fistula and restore normal hemodynamics. Currently, the main treatment methods include interventional embolization and surgical resection, or a combination of both <sup>[6]</sup>. Interventional embolization is non-invasive, involving the insertion of guidewires and catheters into the fistula to apply embolization materials that block abnormal blood vessels and occlude the fistula.

In this case, the initial treatment employed a combination of coils and Onyx glue for embolization, which achieved significant short-term results. However, factors such as rapid blood flow through the fistula, incomplete embolization, and neovascularization may increase the risk of subsequent recurrence. Studies have shown that simple embolization treatment has a higher recurrence rate, especially for high-flow fistulas <sup>[7]</sup>. Therefore, considering alternative treatment methods for recurrent or complex cases is necessary.

Surgical resection of the fistula and involved dura mater is a more thorough treatment approach. In this case, successful surgical resection of the arterialized vein was performed, and no recurrence was observed during long-term follow-up, confirming the reliability of surgical treatment <sup>[8]</sup>. Apart from interventional embolization and surgical resection, other treatment strategies can be considered, such as radiotherapy for patients who cannot tolerate surgery or interventional treatment, and drug therapy (e.g., platelet inhibitors and anticoagulants) to reduce the risk of intravascular embolization. However, the effectiveness and safety of these treatment strategies require further research and clinical validation.

#### 6. Treatment selection and individualized approach

The treatment of TCCAVF should be tailored to the specific circumstances of each patient: interventional therapy is suitable for patients with deep-seated fistulas or high surgical risk, but recurrence should be vigilantly monitored. Surgical treatment is more appropriate for cases with superficial fistulas, failed interventional therapy, or concomitant intracranial hematomas <sup>[9]</sup>. The treatment process in this case suggests that surgical treatment may be a better option for high-flow, superficial fistulas <sup>[10]</sup>. When deciding on a treatment plan, the patient's overall condition, severity of the disease, and treatment risks should be fully considered. Simultaneously, open communication with the patient is essential, providing detailed information about the benefits and risks of treatment and involving them in the final decision-making process. Surgical treatment offers the advantage of directly addressing the fistula, ensuring complete removal and thereby reducing the risk of recurrence. Before surgery, a detailed surgical plan should be developed, covering aspects such as surgical timing, steps, and potential complications.

Comprehensive preoperative evaluation and preparation are crucial, including neuroimaging assessments like magnetic resonance imaging (MRI) and angiography to determine the specific details of the fistula (location, size, feeding arteries, etc.) and assess for any complications (e.g., intracranial hematoma). The patient's general health status, including the cardiovascular system, liver function, and coagulation profile, should also be thoroughly evaluated. During the surgical procedure, close monitoring of blood pressure and cerebral hemodynamics is essential to prevent intraoperative bleeding or other complications. Following surgery, the patient's condition, including neurological status and wound healing, should be closely monitored to avoid complications. The patient may require a period of rehabilitation, including physical therapy and rehabilitation training, to facilitate functional recovery. Tailoring an individualized treatment plan for each patient can maximize their needs and improve overall treatment efficacy.

# 7. Long-term follow-up and prognosis

Patients with TCCAVF require long-term follow-up, with regular monitoring for recurrence through DSA or CTA. These imaging examinations can clearly demonstrate the presence of abnormal vessels and blood flow,

enabling doctors to accurately evaluate treatment effectiveness and recurrence risk. Establishing a regular follow-up schedule is crucial to ensure patient safety. Besides imaging, patients' symptoms are also important indicators for assessing treatment efficacy. The disappearance of pulsatile tinnitus, a common symptom in TCCAVF patients, suggests improvement in hemodynamic abnormalities. However, it should be noted that the absence of pulsatile tinnitus does not necessarily indicate complete resolution of the lesion but rather indicates a certain degree of treatment success.

Therefore, long-term follow-up should also assess other neurological functions to better manage the patient's condition <sup>[11]</sup>. During follow-up, attention should also be paid to the patient's quality of life and mental health. Treating TCCAVF is a long-term process, and patients may experience adverse emotions such as anxiety, fear, and depression. Hence, it is essential to provide individualized psychological support and health education based on the patient's specific situation, aiming to improve their mental state and enhance treatment compliance. Additionally, helping patients develop healthy lifestyles, including controlling blood pressure and blood sugar levels, is important. In this case, no recurrence was observed during the 180-day postoperative follow-up.

#### 8. Conclusion

Reviewing the treatment process of this case, for the treatment of traumatic middle meningeal arteriovenous fistulas, accurate localization through DSA angiography, selection of a small incision in the localized area, and amputation of the draining vein under local or general anesthesia constitute an effective and minimally invasive treatment approach. This treatment method offers advantages such as minimal trauma and low cost, making it suitable for most patients. However, for some complex cases, a more comprehensive treatment plan may be required, such as a combination of interventional therapy and surgical treatment, to ensure effective control of the disease. In clinical practice, doctors need to select the most appropriate treatment plan based on the specific conditions and lesion characteristics of the patient to achieve the best treatment effect. Simultaneously, treatment experience and long-term follow-up observations of similar cases have important guiding significance, helping to improve treatment plans and increase the success rate of treatment.

#### **Disclosure statement**

The authors declare no conflict of interest.

# References

- Lim DH, Kim TS, Joo SP, et al., 2007, Intracerebral Hematoma Caused by Ruptured Traumatic Pseudoaneurysm of the Middle Meningeal Artery: A Case Report. J Korean Neurosurg Soc, 42: 416–418.
- [2] Kawaguchi T, Kawano T, Kaneko Y, et al., 2002, Traumatic Lesions of the Bilateral Middle Meningeal Arteries Case Report. Neurol Med Chir (Tokyo), 42: 221–223.
- [3] Sakata H, Nishimura S, Mino M, et al., 2009, Serial Angiography of Dynamic Changes of Traumatic Middle Meningeal Arteriovenous Fistula: Case Report. Neurol Med Chir (Tokyo), 49: 462–464.
- [4] Chen CJ, Lee CC, Ding D, 2018, Endovascular Treatment of Intracranial Dural Arteriovenous Fistulas: A Systematic Review and Meta-Analysis. World Neurosurg, 110: 529–541.

- [5] Gandhi D, Chen J, Pearl M, et al., 2012, Intracranial Dural Arteriovenous Fistulas: Classification, Imaging Findings, and Treatment. AJNR Am J Neuroradiol, 33(6): 1007–1013.
- [6] Gross BA, Du R, 2012, The Natural History of Cerebral Dural Arteriovenous Fistulae. Neurosurgery, 71(3): 594–602.
- [7] Lv X, Jiang C, Li Y, et al., 2009, Complications Related to Percutaneous Transarterial Embolization of Intracranial Dural Arteriovenous Fistulas in 40 Patients. AJNR Am J Neuroradiol, 30(3): 462–468.
- [8] Zipfel GJ, Shah MN, Refai D, et al., 2009, Cranial Dural Arteriovenous Fistulas: Modification of Angiographic Classification Scales Based on New Natural History Data. Neurosurg Focus, 26(5): E14.
- [9] van Dijk JM, terBrugge KG, Willinsky RA, et al., 2002, Clinical Course of Cranial Dural Arteriovenous Fistulas With Long-Term Persistent Cortical Venous Reflux. Stroke, 33(5): 1233–1236.
- [10] Lawton MT, Chun J, Wilson CB, et al., 1999, Ethmoidal Dural Arteriovenous Fistulae: An Assessment of Surgical and Endovascular Management. Neurosurgery, 45(4): 805–811.
- [11] Borden JA, Wu JK, Shucart WA, 1995, A Proposed Classification for Spinal and Cranial Dural Arteriovenous Fistulous Malformations and Implications for Treatment. J Neurosurg, 82(2): 166–179.

#### Publisher's note

Bio-Byword Scientific Publishing remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.