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Case Report

Cross-Circulation Thrombectomy for Acute Middle Cerebral Artery Occlusion from Dislodged Thrombus of a Giant Internal Carotid Aneurysm: A Case Report

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Objective: Giant aneurysms of the cavernous segment of the internal carotid artery presenting as acute ischemic stroke (AIS) are rare and often misdiagnosed. Limited treatment experience further complicates management.

Case Presentation: A 70-year-old female presented with acute right middle cerebral artery (MCA) occlusion due to a dislodged thrombus from a giant internal carotid aneurysm. Emergency multimodal brain imaging techniques, including CT, CTA, and DSA, were used to clarify the diagnosis. Given the giant aneurysm's interference, cross-circulation thrombectomy via the anterior communicating artery was performed to revascularize the occluded artery. The patient achieved a relatively good outcome due to timely reperfusion.

Conclusion: This case highlights the importance of advanced imaging and innovative surgical techniques in managing complex cerebrovascular conditions, offering insights for future treatment of giant intracranial aneurysms with cerebral embolization.

Keywords giant intracranial aneurysms, dislodged thrombus, middle cerebral artery occlusion, acute ischemic stroke, mechanical thrombectomy

Introduction

Giant intracranial aneurysms are defined as those with a maximum diameter greater than 25 mm, representing a rare subset that accounts for approximately 5% of all cerebral aneurysms. These aneurysms usually develop in the 50s to 70s, with a slight female predominance. Giant intracranial aneurysms carry a significantly greater risk of rupture and a worse prognosis than other aneurysms. The annual risk of rupture for small aneurysms (<10 mm) is 0.7%, whereas the annual risk of rupture for giant intracranial aneurysms is 33.4%.[1)] Untreated giant intracranial aneurysms have a 2-year mortality rate of 68% and a 5-year mortality rate of 80%.[2)]

The presentation of intracranial aneurysms with acute ischemic stroke (AIS) is uncommon. Common clinical manifestations of giant intracranial aneurysms include subarachnoid hemorrhage (SAH), ischemic stroke, and mass effects. A part of the unruptured giant aneurysms is asymptomatic and is detected by accident on neuroimaging. The treatment of giant intracranial aneurysms is quite challenging, and there is no uniform protocol. We report a rare case of AIS resulting from a dislodged thrombus from a giant internal carotid aneurysm, which was subsequently managed with endovascular treatment to achieve recanalization of the embolized artery.

Case Presentation

A right-handed female in her 70s presented to the emergency room with a witnessed unconsciousness and left

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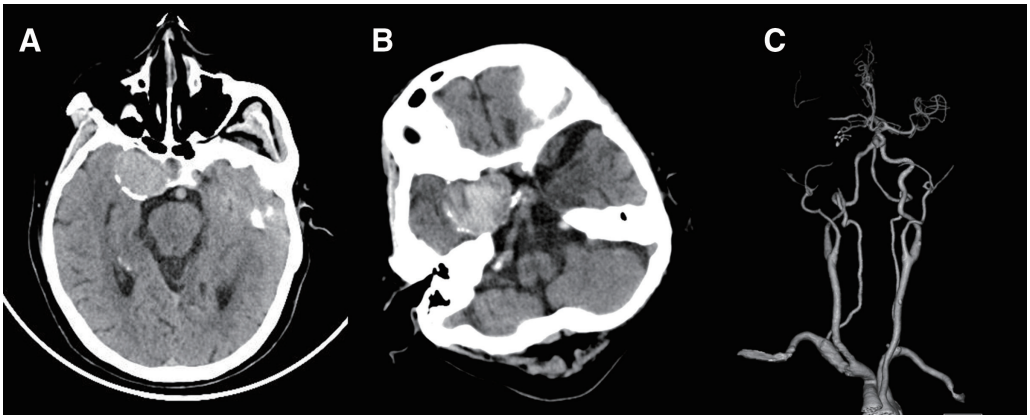

Fig. 1 (**A**) A head CT scan 2 weeks prior to admission showed a giant unruptured intracranial aneurysm. (**B**) A head CT scan at admission showed an intraparenchymal mass (35 × 30 × 44 mm) with calcifications in the parasellar region. (**C**) CT angiography at admission reveals occlusion of the right MCA and invisibility of the cavernous segment of the right ICA. ICA: internal carotid artery; MCA: middle cerebral artery

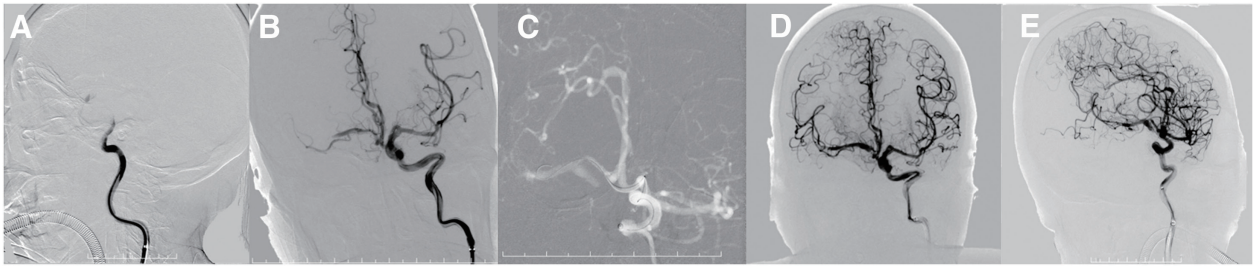

Fig. 2 (**A**) Preoperative DSA showed a giant intracranial aneurysm at the cavernous segment of the right internal carotid artery. The right internal carotid artery at that level was nearly occluded due to the presence of a giant aneurysm. (**B**) DSA before endovascular treatment showed occlusion of the right MCA. (**C**) The microcatheter passed through the left internal carotid artery and reached the occluded right MCA via the anterior communicating artery to carry out MT. (**D**) Postoperative DSA in anteroposterior view showed successful recanalization of M1 and one M2 branch. (**E**) Postoperative DSA in oblique view. MCA: middle cerebral artery; MT: mechanical thrombectomy

limb hemiparesis. She has a medical history of hypertension, coronary artery disease, ischemic stroke, SAH, and seizures after SAH. Two weeks prior to admission, she had a fever due to acute cholecystitis. Computed tomography (CT) of the head revealed a giant unruptured intracranial aneurysm (**Fig. 1A**), which was not given intervention because of her weak condition at that time. At this admission, CT demonstrated a large parasellar mass with calcifications, suggesting a giant intracranial aneurysm (**Fig. 1B**). CT angiography revealed right middle cerebral artery (MCA) occlusion (**Fig. 1C**). This could explain her acute ischemic symptoms and was the indication for mechanical thrombectomy (MT).

Endovascular treatment

Under general anesthesia, 6-Fr Neuron MAX (Penumbra, Alameda, CA, USA) long sheath was inserted into the right internal carotid artery (ICA) via a right femoral artery approach. A 6-Fr intermediate catheter (Ton-Bridge Medical, Zhuhai, China) was inserted into the C3 segment of the right ICA. Digital subtraction angiography (DSA) confirmed a giant intracranial aneurysm located at the cavernous segment of the right ICA with thrombosis in the aneurysmal sac (**Fig. 2A**). Attempts to advance a microcatheter (Rebar microcatheter 18; Medtronic, Minneapolis, MN, USA) and a 0.014-inch guidewire (Synchro2 guidewire; Stryker, Fremont, CA, USA) across the occlusion and into the distal right ICA were unsuccessful. Subsequently, the 6-Fr Neuron MAX long sheath and 6-Fr intermediate catheter were shifted into the left ICA, where DSA confirmed occlusion of the right MCA (**Fig. 2B**). A Rebar microcatheter 18 was advanced on a 0.014-inch guidewire through the anterior communicating artery (ACommA), successfully crossing the occlusion site and reaching the upper branch of the M1 segment (**Fig. 2C**). A detachable intracranial stent (Solitaire FR stent 4 ∗ 20 mm; Medtronic) was deployed, obtaining partial recanalization of the target vessel with a Thrombolysis in Cerebral Infarction (TICI) Scale score of 2b (**Fig. 2D** and **2E**).

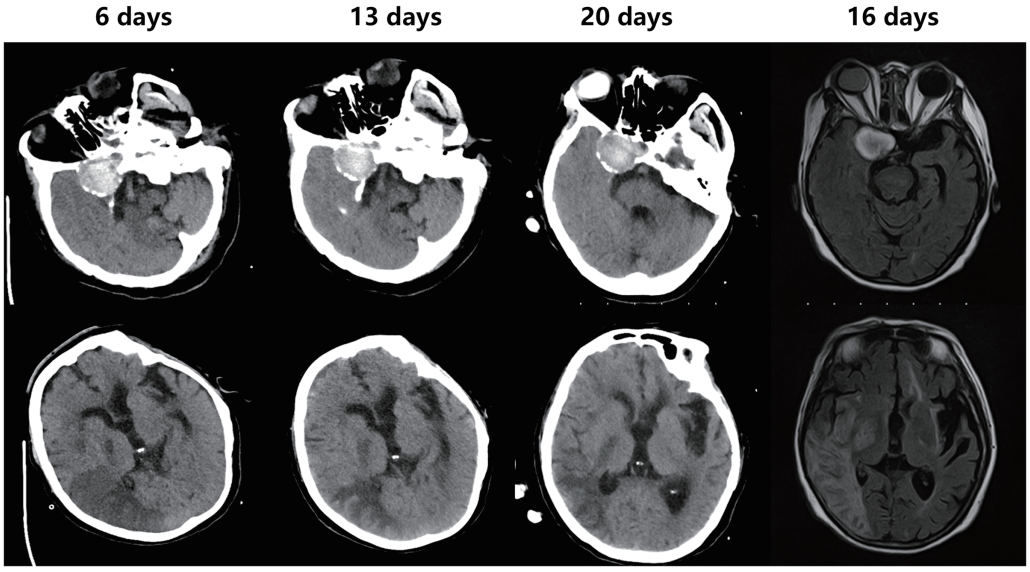

Fig. 3 Follow-up head CT and MRI at different times showed cerebral infarction in the right temporal lobe.

Dual antiplatelet therapy (aspirin and ticagrelor) was initiated following the implantation of the Solitaire FR stent. MRI at 2 weeks showed cerebral infarction in the right temporal lobe (**Fig. 3**). The patient achieved a relatively favorable outcome and did not experience any recurrent stroke during the 3-month follow-up period after the procedure. Despite the continued embolic potential of the patent ICA, the possibility of parent artery occlusion of the right ICA following MCA reperfusion was considered. However, given the patient's advanced age, frail condition, the family's wishes, and the satisfactory outcome already achieved, a palliative approach was chosen.

Discussion

We describe a rare case of a patient presenting with a giant intracranial aneurysm in the cavernous sinus segment of the ICA, manifesting as an AIS. The dislodgment of the thrombus within the aneurysm led to the occlusion of the M1 segment of the distal ipsilateral MCA. Emergency MT was taken and part of the vessel was opened by proceeding from the contralateral ICA, which resulted in a relatively good clinical outcome.

Spontaneous thrombosis is quite frequent in giant intracranial aneurysms, with an incidence of about 50%.[3] Aneurysm geometry and hemodynamics within the aneurysm cavity are factors that contribute to thrombosis. The high incidence of thrombosis within giant intracranial aneurysms is associated with a critical ratio between aneurysmal volume and aneurysmal neck size; when this ratio exceeds the critical threshold, intra-aneurysmal thrombosis is likely to occur.[4] The Virchow triad, consisting of stasis, hypercoagulability, and endothelial injury, is closely associated with intravascular thrombosis. Experimental models have also indicated that blood flow within giant aneurysms is characterized by blood stasis, increased viscosity, and slow flow, which may contribute to aneurysm thrombosis.[5] In this case, the hypercoagulable state accompanying cholecystitis probably enhanced intra-aneurysmal vascular stasis. Inflammation of the vessel wall is a possible contributor to thrombosis within the aneurysm. Turbulent flow within the aneurysmal sac causes repeated endothelial injury, inducing mural thrombus formation. This subsequently leads to wall scarring, fibroblast invasion, collagen formation, and deposition of fibrous material.[6] Wall calcification appearing as peripheral ring enhancement on imaging is thought to be associated with long-standing aneurysmal thrombosis.[7] Therefore, multiple factors may interact to contribute to the thrombosis of the giant aneurysm in our patient. Partially thrombosed aneurysms can be a source of emboli, potentially causing transient ischemic attacks (TIA) or cerebral infarction.[8,9]

Intracranial aneurysms presenting with AIS are uncommon. Approximately 4% of giant intracranial aneurysms present with cerebral ischemic manifestations, including TIA or stroke. These symptoms are secondary to distal vascular embolization due to thrombus dislodgement from the sac. Embolization is most common in giant aneurysms of

the internal carotid and middle cerebral arteries. Busse and Grote[9] reported a case of a partially thrombosed carotid bifurcation aneurysm manifesting as recurrent ischemic attacks, which was ceased after clipping the aneurysm.[9] In the case of Zerouali Boukhal, et al., a giant aneurysm of the subclinoid portion of the ICA was presented with a rare combination of complications, including subarachnoid hemorrhage, a right subdural temporal hematoma, and total Sylvian ischemic stroke.[10] Sometimes, it was difficult to diagnose an intracranial aneurysm hidden in the distal site of an occluded artery by existing imaging.[11–13]

The treatment of giant intracranial aneurysms combined with AIS is challenging. Treatment protocol needs to be tailored based on individual patient factors. Treatment experience regarding giant intracranial aneurysms combined with acute cerebrovascular events is even more scarce. It is critical for an overall outcome to perform thrombectomy using novel access techniques to obtain reperfusion of an occluded cerebral vessel. A cross-circulation technique is to gain circuitous access to a cerebral vessel through a patent anterior or posterior communicating artery of the Willis circle. Previous reports have proved successful cross-circulation techniques for the treatment of large-vessel occlusion (LVO) in AIS patients under extremely rare situations.[14–16] Unlike our cases, these prior reports involve chronic tandem occlusion of the more proximal approach vessel. Our case is the first to employ a trans-ACommA approach to treat an MCA occlusion by dislodgment thrombus from the giant intracranial aneurysms using stent retriever thrombectomy. The cross-circulation thrombectomy technique offers advantages for cases with anatomical constraints or unfavorable direct access. This approach allows for the recanalization of occluded arteries that are otherwise unreachable through standard anterograde MT methods. However, this technique also carries considerable risks, including vessel rupture, dissection, and the propagation of emboli into uninvolved vascular territories.[17,18] Therefore, cross-circulation thrombectomy should be reserved for carefully selected cases where anterograde approaches are not feasible. The Solitaire FR stent retriever is widely recognized for its use in MT for AIS due to LVOs. Moreover, the detachable Solitaire FR can also be used as a permanent stent because of the reduction in procedural time as well as cost. It is particularly suitable for complex procedures such as cross-circulation thrombectomy. This case may provide some implications for future treatment of giant intracranial aneurysms combined with cerebral embolization.

Conclusion

AIS combined with an intraparenchymal mass should prompt consideration of the possibility of an aneurysm. Comprehensive radiologic assessment is crucial for accurate diagnosis. Endovascular treatment represents a viable and effective therapeutic option, particularly with the application of innovative surgical techniques for managing complex cerebrovascular conditions.

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Disclosure Statement

The authors declare that there are no conflicts of interest related to this work.

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