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Combined High-Flow and Low-Flow Extracranial-Intracranial Bypass for a Giant Cavernous Segment Internal Carotid Artery Aneurysm: A Case Report and Literature Review

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Abstract

Aneurysms of the cavernous segment of the internal carotid artery (ICA) are relatively uncommon, accounting for 2%-9% of all intracranial aneurysms [1]. Most patients with these aneurysms are asymptomatic [2]. However, some may present with neurological deficits due to mass effect, such as oculomotor nerve palsy, or develop carotid-cavernous fistulas upon rupture. Rarely, rupture can lead to subarachnoid hemorrhage or bleeding into the sphenoid sinus [2]. Clinically, aneurysms are classified by maximum diameter: small (<5 mm), medium (5 mm ≤ diameter <15 mm), large (15 mm ≤ diameter <25 mm), and giant (≥25 mm). The risk of rupture for cavernous ICA aneurysms correlates with size; for diameters <13 mm, the 5-year rupture risk is 0%; for 13-24 mm, it is 3%; and for ≥25 mm, it rises to 6.4% [3]. Treatment strategies for symptomatic cavernous ICA aneurysms remain debated and include direct surgical clipping, endovascular coiling, flow diverter placement, or indirect approaches like parent artery occlusion following revascularization [4]. Advances in microsurgical techniques and endovascular flow diversion have significantly improved treatment success rates.

We report a case of a ruptured giant cavernous ICA aneurysm treated with combined high-flow and low-flow extracranial-intracranial (EC-IC) bypass followed by parent artery ligation.

Keywords: Giant cavernous internal carotid artery aneurysm; High-flow bypass; Low-flow bypass; Aneurysm isolation; Graft occlusion; Arterialization

Introduction:

Aneurysms arising from the cavernous segment of the internal carotid artery (ICA) represent a distinct entity, accounting for approximately 2-9% of all intracranial aneurysms [1]. Their natural history is often considered more benign than their intradural counterparts, with a substantial number of patients remaining asymptomatic [2]. However, these lesions can manifest clinically through mass effect, leading to cranial neuropathies such as oculomotor nerve palsy, or rarely, rupture resulting in life-threatening conditions like carotid-cavernous fistula or epistaxis [2]. The risk of rupture is strongly correlated with size, with giant aneurysms (≥25 mm) carrying a significantly higher 5-year rupture risk compared to their smaller counterparts [3].

The management paradigm for symptomatic or enlarging cavernous ICA aneurysms is multifaceted and continues to evolve.

Treatment options span from direct microsurgical clipping and endovascular techniques—including coiling, stent-assisted coiling, and more recently, flow diversion—to indirect strategies involving parent artery occlusion (PAO) with or without prior cerebral revascularization [4, 18]. While endovascular flow diverters have revolutionized the management of complex proximal ICA aneurysms, they are not without limitations, including the necessity for prolonged dual antiplatelet therapy—associated with inherent risks of hemorrhage and thromboembolism [19] and considerable economic cost, which may preclude their use in certain patient populations [20].

A fundamental principle governing indirect treatment via PAO is the adequacy of the cerebral collateral circulation. The balloon occlusion test (BOT) remains the gold standard for assessing tolerance to ICA sacrifice [8]. However, in clinical scenarios involving acutely ill or uncooperative patients, performing a definitive BOT may be unfeasible. In such contexts, bypass surgery followed by aneurysm trapping emerges as a critical life-saving procedure. The strategic choice between a low-flow bypass (e.g., using the superficial temporal artery) and a high-flow bypass (e.g., using a radial artery or saphenous vein graft) is predicated on the anticipated cerebral blood flow demand after ICA occlusion [9].

In this article, we present a instructive case of a ruptured giant cavernous ICA aneurysm in a young woman. The patient's clinical condition rendered preoperative BOT and perfusion studies impossible, and financial constraints precluded the use of a flow diverter. Faced with these challenges, we successfully executed a combined surgical strategy: a protective low-flow extracranial-intracranial (EC-IC) bypass followed by a high-flow bypass using a radial artery graft, and subsequent aneurysm isolation. The postoperative course was marked by an unexpected yet instructive event—the occlusion of the high-flow radial artery graft—which, critically, was well-tolerated by the patient due to the compensatory function of the pre-established low-flow bypass and robust native collaterals. This case not only exemplifies a tailored surgical solution for a complex pathology under significant constraints but also provides profound insights into the dynamics of cerebral collateral circulation and the indispensable role of a protective bypass. We discuss the technical nuances, the potential mechanisms behind graft occlusion, and review the relevant literature.

Case Presentation:

A 39-year-old woman presented to our emergency department with a 27-hour history of sudden severe headache, nausea, and vomiting. A cranial CT scan performed at a local hospital revealed a "round, hyperdense shadow in the left parasellar region." She was admitted after a cerebral CTA in our ER. On admission, the patient was somnolent with left ptosis and anisocoria (left pupil 4.0 mm, right pupil 3.0 mm). Both pupils showed sluggish direct light reflexes. Ocular movements were full in the right eye but restricted in the left. Limb muscle strength was normal, with noted neck stiffness.Subsequent digital subtraction angiography (DSA) confirmed a giant left ICA aneurysm, measuring approximately 28.3mm × 27mm × 22mm with a neck width of 28.1mm. Compression of the left carotid artery during right ICA angiography showed no filling via the anterior communicating artery. Conventional left vertebral angiography demonstrated patent posterior communicating arteries. Left vertebral angiography with left carotid compression confirmed robust filling via the posterior communicating artery.

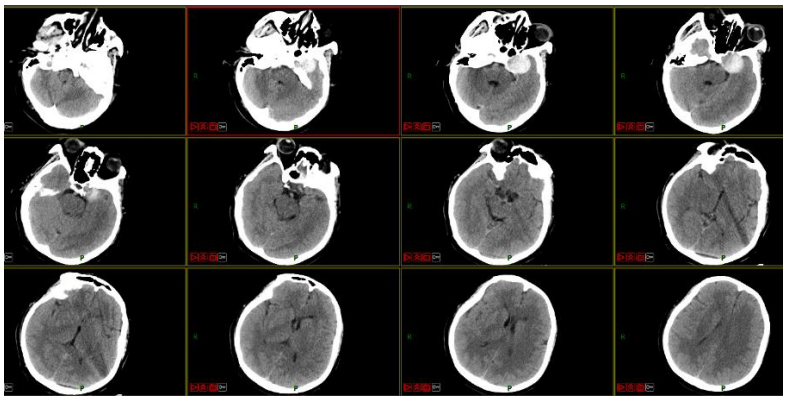


Figure 1: Emergency cranial CT on admission showing a round hyperdense lesion in the left parasellarregion.

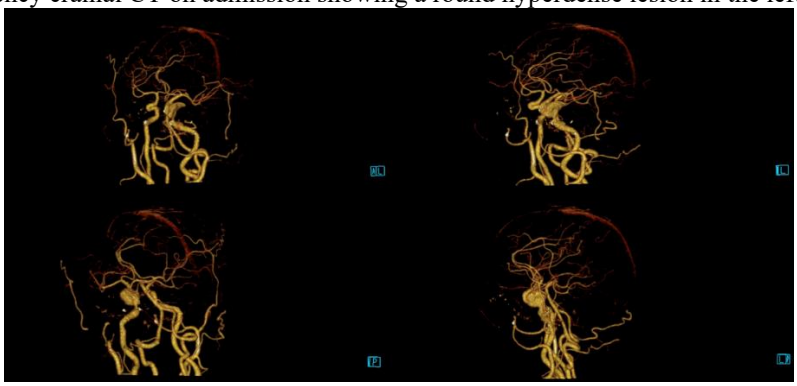


Figure 2: Admission cerebral CTA revealing an irregular outpouching from the left cavernous ICA, with a maximum cross-section of approximately 1.7×1.2 cm.

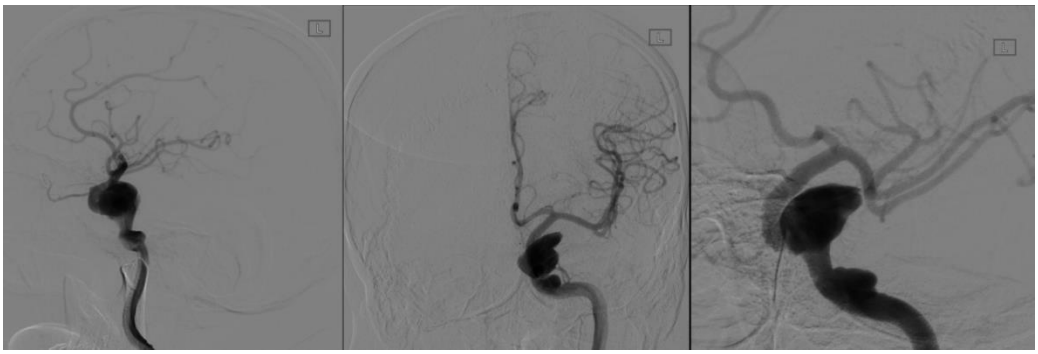
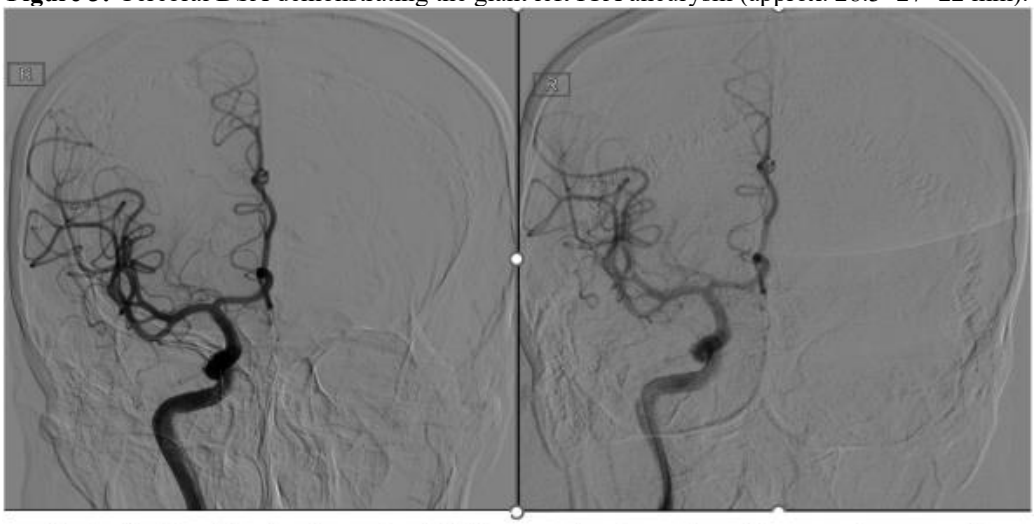


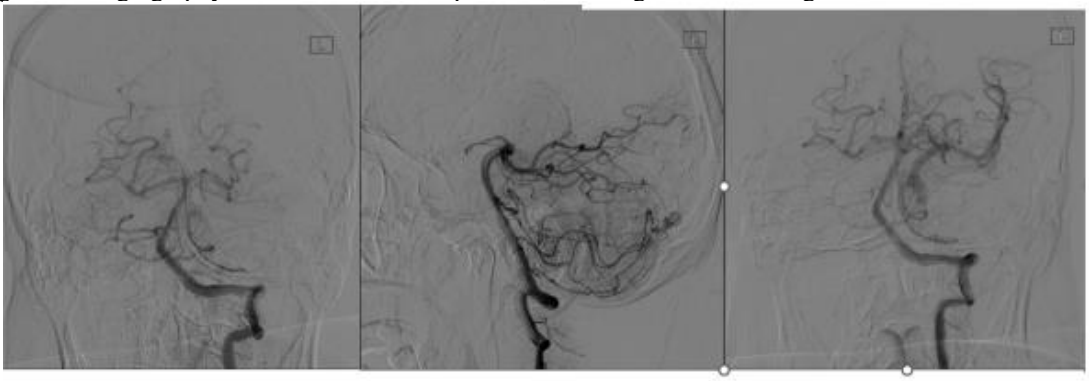
Figure 3: Cerebral DSA demonstrating the giant left ICA aneurysm (approx. 28.3×27×22 mm).



Digital Subtraction Angiography (DSA)

Angiography with manual compression of the left cervical carotid artery

Figure 4: Right ICA angiography with left carotid compression, showing no cross-filling via the anterior communicating artery.



Vertebral artery angiography demonstrated a patent posterior communicating artery (PComA).

Vertebral artery angiography performed with manual compression of the left cervical carotid artery demonstrated robust filling via the posterior communicating artery.

Figure 5: Robust left posterior communicating artery filling demonstrated.

Surgical Procedure:

Positioning, Exposure, and Incision Design

The patient was positioned supine with the left shoulder elevated. The head was rotated approximately 45° to the right, slightly extended, and fixed in a Mayfield headholder. The course of the left superficial temporal artery (STA) and its parietal branch were marked on the scalp. A curvilinear (question-mark) incision was planned along the STA. A longitudinal incision was marked along the anterior border of the left sternocleidomastoid muscle for neck

exposure. Allen's test confirmed adequate ulnar collateral circulation, and the right radial artery was harvested by a hand surgery team for use as the high-flow bypass graft.

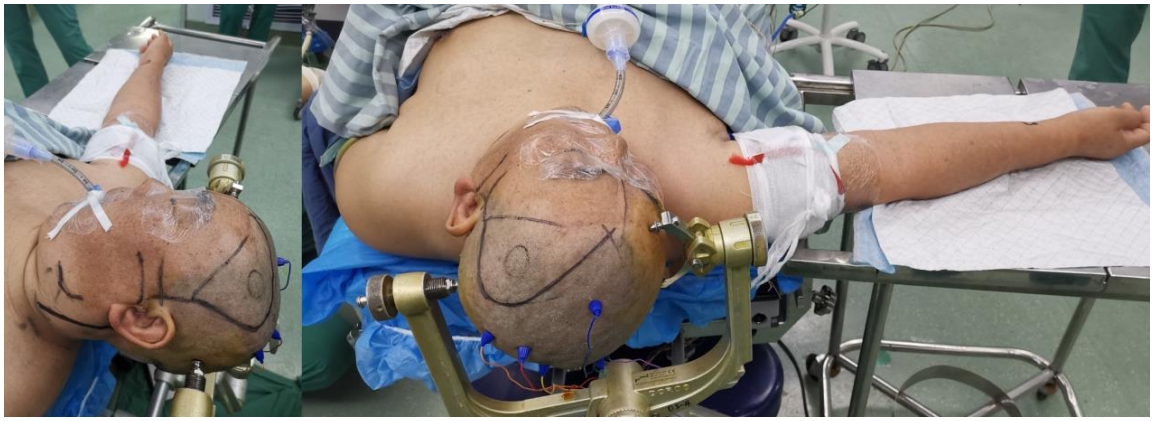


Figure 6: Patient positioning and planned incisions.

STA to M4 Segment of Middle Cerebral Artery (MCA) Anastomosis (Low-Flow Bypass)

To prevent ischemic events during the subsequent high-flow bypass procedure, a protective low-flow bypass was first established. The frontal branch of the left STA was anastomosed to a cortical artery in the temporal lobe, and the parietal branch was anastomosed to a cortical artery in the parietal lobe.

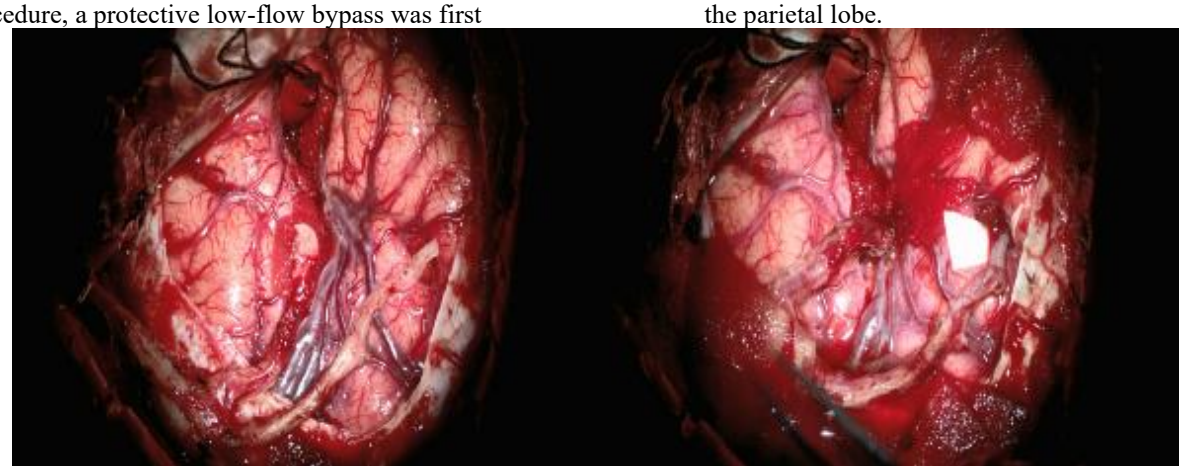


Figure 7: Intraoperative view of the protective low-flow STA-M4 bypass.

Radial Artery Graft Tunneling

The left neck was incised to expose the common carotid artery (CCA), internal carotid artery (ICA), and external carotid artery (ECA). A tunnel was created beneath the temporalis muscle, passing below the zygomatic arch, to connect the cranial and cervical fields. The harvested radial artery graft was passed through this tunnel, and its adventitia was trimmed.

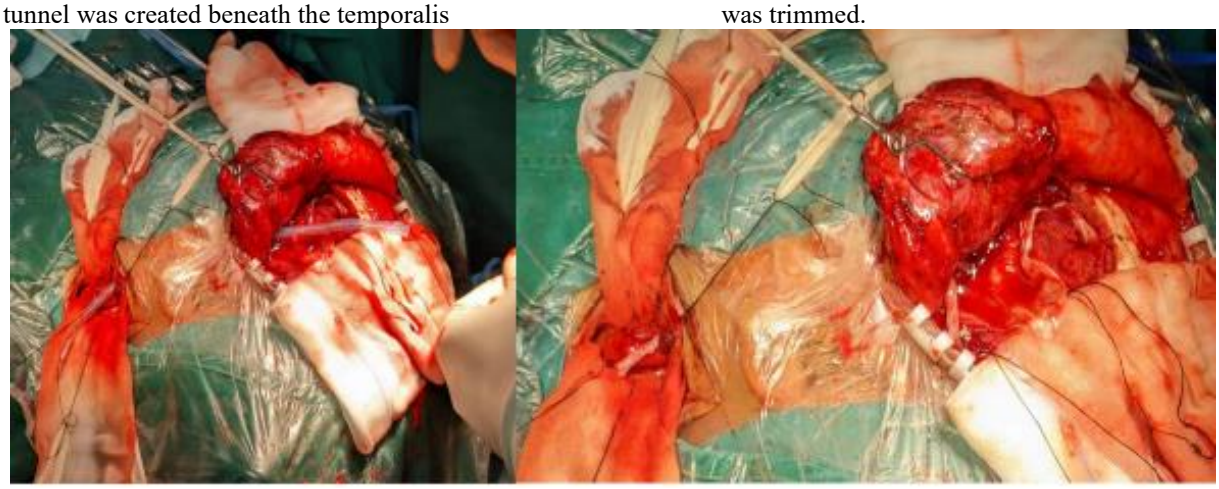


Figure 8: Radial artery graft being passed through the subcutaneous tunnel.

Radial Artery to ICA and M2 Segment Anastomosis (High-Flow Bypass)

Temporary aneurysm clips were applied to the cervical ICA. An arteriotomy was made in the anterior wall using a 3.5 mm punch, and the lumen was irrigated with heparinized saline. The proximal end of the radial artery graft was anastomosed end-to-side to the ICA. The distal M2 superior trunk of the MCA was temporarily clipped, incised longitudinally, and irrigated. The distal end of the radial artery graft was then anastomosed end-to-side to the M2 segment.

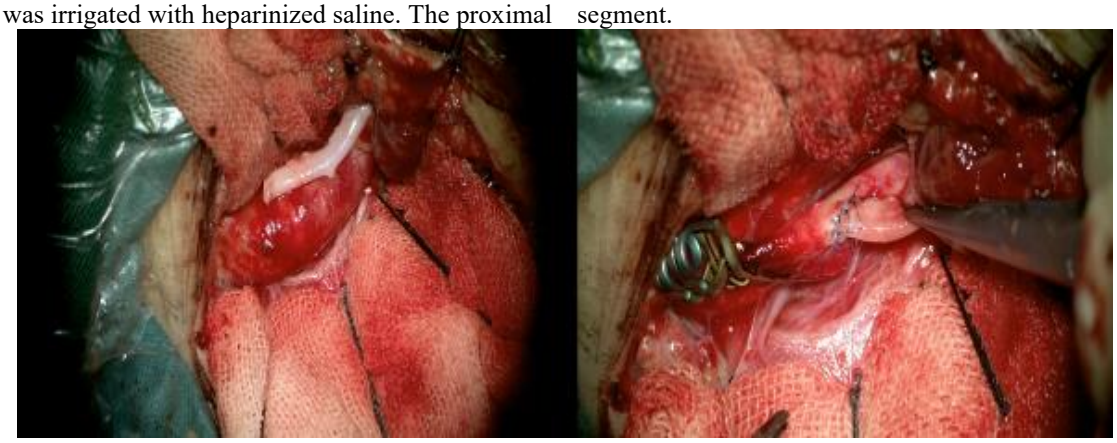


Figure 9: Proximal anastomosis of the radial artery graft to the cervical ICA.

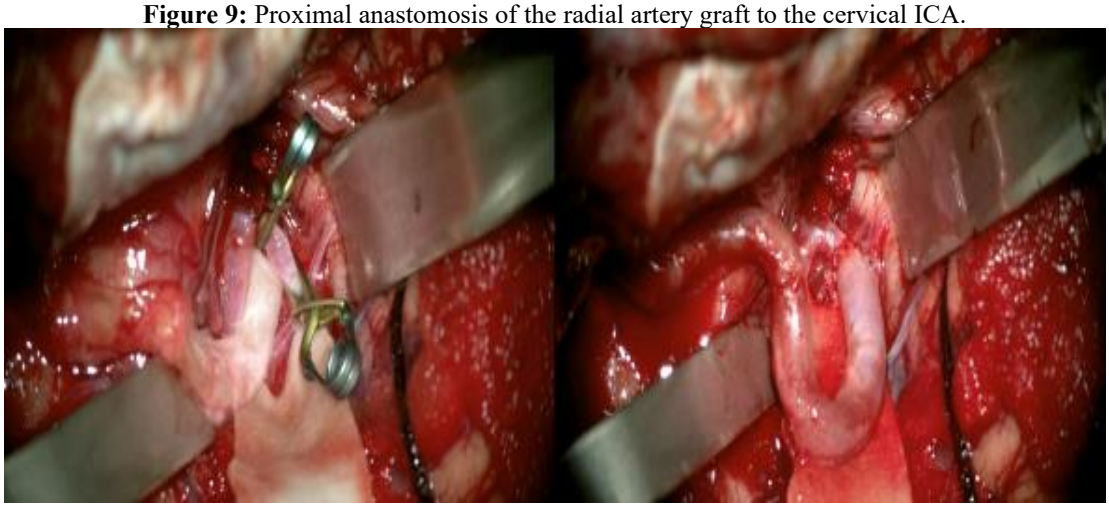
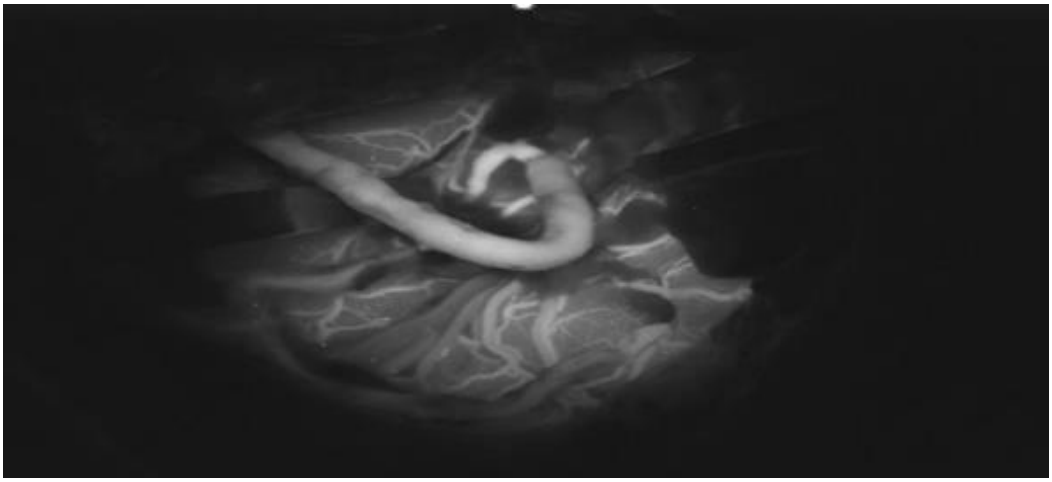


Figure 10: Distal anastomosis of the radial artery graft to the M2 superior trunk.

Intraoperative Blood Flow Monitoring

Intraoperative transcranial Doppler ultrasonography confirmed patent blood flow in the radial artery graft, the MCA M2 segment (proximal and distal to the anastomosis), and the STA frontal and parietal branches. Indocyanine green videoangiography demonstrated rapid filling of the M2 segment via the radial artery graft and prompt cortical artery filling via the STA bypass.



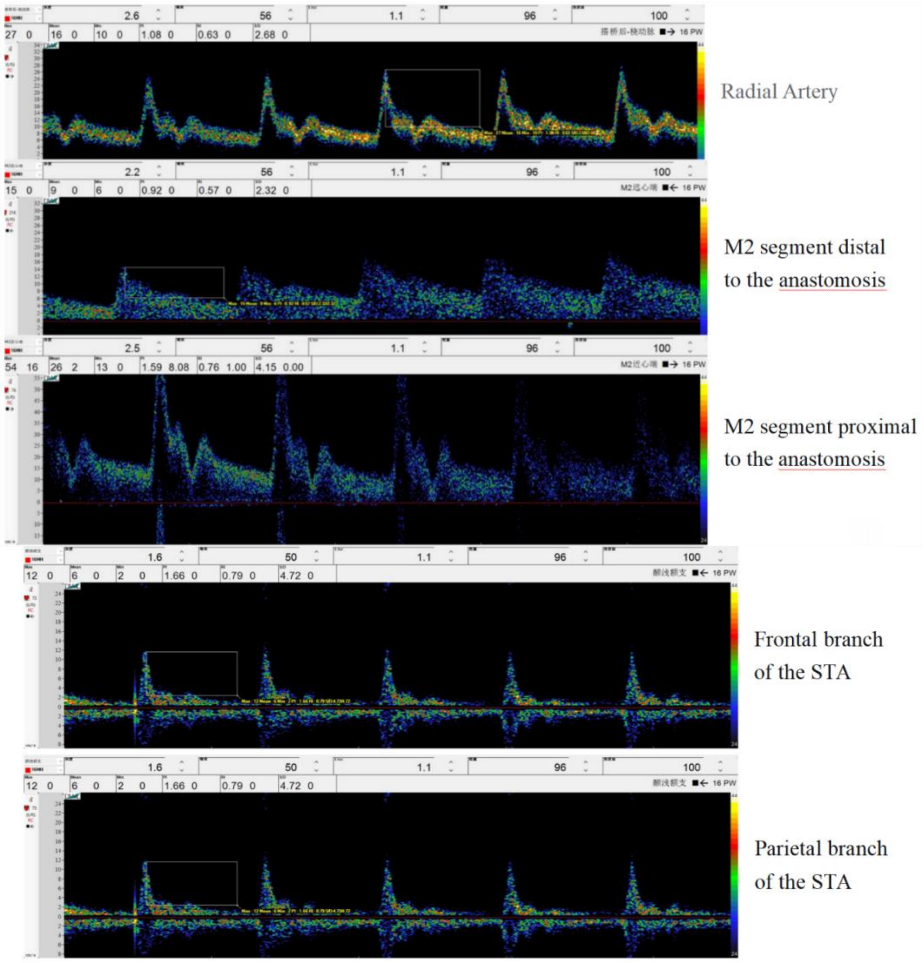


Figure 11: Intraoperative flow monitoring using transcranial Doppler and indocyanine green videoangiography.

The ICA was ligated distal to the anastomosis with the radial artery, effectively isolating the giant cavernous aneurysm. To prevent agitation-induced anastomotic rupture upon emergence from anesthesia, the patient was kept sedated and transferred intubated to the neurosurgical ICU. Postoperative day 1 CT showed no new hemorrhage or infarction. Slight enlargement of the aneurysm was noted, likely due to intra-aneurysmal thrombosis post-isolation. Supportive care continued.On postoperative day 7, a head and neck CTA showed the STA bypass was patent, but the radial artery graft was not visualized. Follow-up transcranial Doppler revealed a decrease in the STA pulsatility index (PI) from 2.28 preoperatively to 0.70 postoperatively, indicating 'arterialization' of the STA and adequate cortical perfusion. By postoperative day 10, the patient was alert, oriented, with normal limb movement, but persistent left oculomotor nerve palsy. The patient was discharged on postoperative day 18 in improved condition.

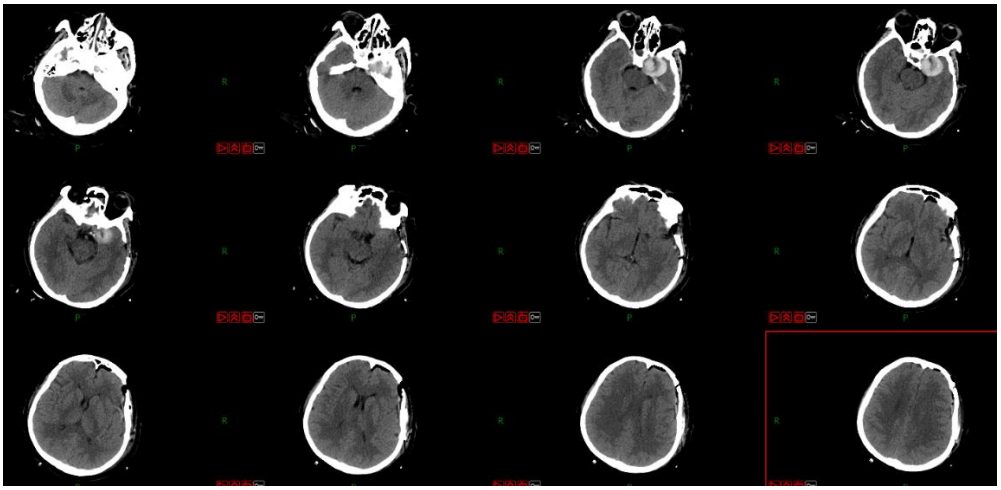


Figure 12: Postoperative day 1 CT scan showing no new hypodensities or hyperdensities.

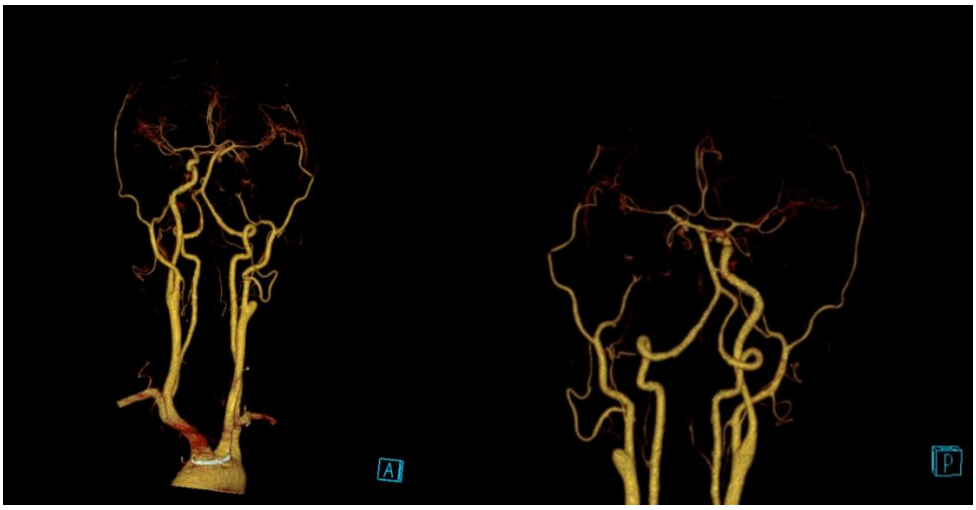


Figure 13: Postoperative day 7 CTA.

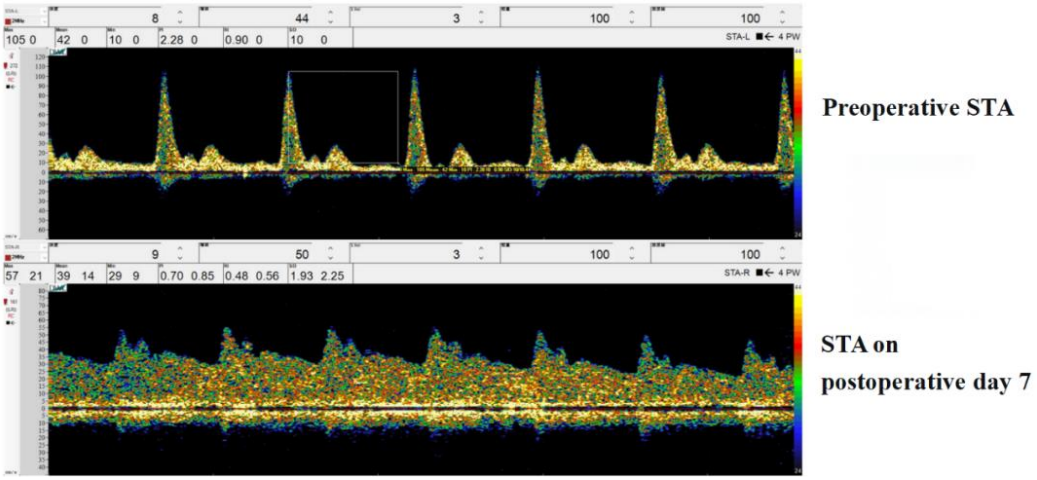


Figure 14: Postoperative imaging demonstrating 'arterialization' of the STA and adequate cortical supply.

Discussion:

Cavernous ICA aneurysms often follow a relatively benign natural history. Stiebel-Kalish et al., in a follow-up of 111 untreated patients, reported adverse events in 10%, including stroke (2%), subarachnoid hemorrhage (1%), direct carotid-cavernous fistula (1%), and compressive optic neuropathy (6%) [5]. Asymptomatic cavernous ICA aneuryms can often be managed conservatively with serial imaging, unless evidence of growth emerges.

Treatment options are diverse, falling into direct and indirect strategies [6]. Endovascular coiling or stent-assisted coiling are effective treatments [7]. More recently, flow diversion has been employed for giant intracranial aneurysms, particularly complex, wide-necked lesions [6]. A significant drawback of flow diverters is the requirement for prolonged dual antiplatelet therapy, associated with risks of hemorrhagic or thromboembolic complications [8]. The substantial cost of these devices is also a consideration. When endovascular treatment is not feasible, direct surgical clipping is an alternative, though it demands advanced microsurgical skill and anatomical expertise.

If neither direct clipping nor endovascular therapy is straightforward, indirect treatment via EC-IC bypass followed by parent artery occlusion and aneurysm isolation offers a viable solution, with acceptable morbidity and mortality rates [9]. This strategy bypasses the need for direct aneurysm manipulation. By reconstructing flow distal to the lesion and subsequently occluding the parent artery, it reduces flow into the aneurysm sac, thereby lowering rupture risk. Although the aneurysm sac persists, the altered hemodynamics significantly reduce the probability of rupture.

For this patient, our team discussed two options: flow diverter placement or bypass with aneurysm isolation. Due to financial constraints, the family opted for the surgical bypass procedure.

The necessity of cerebral revascularization following therapeutic ICA occlusion lacks definitive guideline consensus. The balloon occlusion test (BOT) is commonly used to assess cerebrovascular reserve and guide this decision. ICA occlusion without prior reserve assessment carries a 32%-60% risk of ischemic complications [10]. Even with a passed BOT, the stroke risk is 22% without revascularization, which can be reduced to 14% with additional bypass [11]. The choice between low-flow or high-flow bypass depends on the anticipated degree of flow reduction after ICA occlusion [12].

This patient's somnolence precluded formal BOT or perfusion studies. Collateral assessment was limited to rough angiographic evaluation of the circle of Willis during manual compression. Therefore, direct parent artery occlusion was deemed unsafe. Our preoperative plan involved a preliminary low-flow STA-M4 protective bypass, followed by high-flow radial artery bypass

(ICA-M2) and subsequent ICA ligation distal to the proximal anastomosis, ensuring continuous cerebral perfusion.

Common conduits for high-flow bypass are the radial artery and saphenous vein. Both have been used successfully for various intracranial pathologies, including giant aneurysms, despite inherent structural and biological differences [13]. Saphenous veins provide high flow rates but may cause flow turbulence when anastomosed to smaller (<2 mm) MCA branches [14]. The radial artery is considered to have better long-term patency but is prone to spasm, which can affect outcomes [15]. Early studies reported spasm rates up to 50% [16], though modern pharmacological and mechanical dilation techniques have reduced this to 4%-10% [17]. In this case, we administered 1500 ml of intravenous fluids preoperatively to optimize volume status and cerebral perfusion, maintaining intraoperative mean arterial pressure around 110-120 mmHg. Grafts and recipient vessels were irrigated with heparinized saline, and topical papavarine was applied to prevent spasm. Systemic nimodipine was administered for three days postoperatively. Despite these measures, the radial artery graft was occluded on the 7-day CTA. Potential reasons beyond spasm or thrombus include graft kinking or compression within the tunnel, competitive flow from the robust posterior communicating artery supply making the high-flow graft redundant, or flow competition/counteraction between the low-flow and high-flow bypasses, exacerbated by steal phenomenon from the giant aneurysm.

Despite radial artery graft occlusion, the protective STA bypass and robust posterior circulation collateralization via the posterior communicating artery ensured adequate anterior circulation perfusion, preventing major ischemic sequelae. The aneurysm was excluded from circulation on follow-up CTA, significantly reducing the risks of rupture and thromboembolism from the thrombosing sac, thus achieving the primary surgical goal.

Conclusion:

When direct endovascular treatment or simple surgical clipping is not feasible, combined high-flow and low-flow EC-IC bypass followed by parent artery ligation and aneurysm isolation presents a safe and effective treatment strategy for giant intracranial aneurysms.

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