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Visual improvement following the treatment of internal carotid complex aneurysms through internal carotid ligation combined with STA-MCA bypass

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ABSTRACT

Objective: The combination of internal carotid artery (ICA) ligation with superficial temporal artery (STA) to middle cerebral artery (MCA) bypass has proven effective in the treatment of internal complex carotid aneurysms (ICCA). Indirect exclusion through thrombosis is anticipated to alleviate the mass effect on adjacent neurovascular structures. However, visual impairment may occur secondary to thrombosis of the ICA and ophthalmic artery. The purpose of this study is to emphasise the paradoxical stabilisation and improvement of vision observed in certain cases.

Methods: A retrospective study was conducted from 2016 to 2020, analysing data from a cohort of patients with ICCAs, among whom nine patients underwent ICA ligation in combination with STA-MCA anastomosis. Preoperative and postoperative visual findings were analysed and compared.

Results: All patients underwent STA-MCA bypass in addition to complete ICA ligation. Postoperative clinical outcomes remained unchanged in 8 out of 9 patients. Complete aneurysm exclusion through thrombosis was achieved at 12 months. The STA-MCA anastomosis remained patent in 6 out of 9 patients, with no clinical deterioration observed, except in one case. Notably, irrespective of graft patency, this patient experienced a minor parietal stroke, from which full recovery was achieved after six months. Furthermore, an improvement in visual acuity was observed in two patients at three months.

Conclusion: The ICCA treatment, which involves ICA ligation combined with STA-MCA bypass, alleviates the mass effect on adjacent neurovascular structures. Despite the occurrence of ICA thrombosis, an enhancement in visual acuity has been documented, which can be attributed to the patency of the ophthalmic artery via the meningeal anastomotic circuits.

Keywords

internal carotid complex aneurysms, ophthalmic artery, carotid ligation, superficial temporal artery to middle cerebral artery anastomosis



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INTRODUCTION

Visual impairment caused by Internal Carotid Complex Aneurysms (ICCAs) stems from various factors. The ipsilateral optic nerve, oculomotor, trochlear, and abducens nerves are affected along their course by either the mass effect or the Water Hammer phenomenon of large aneurysms. Additionally, the substantial volume of blood within giant aneurysms generates a whirling and swirling stream, leading to a steal effect (1). Consequently, hypoperfusion of the ophthalmic artery results in dysfunction of the optic nerve. The only efficacious treatment involves an indirect exclusion through thrombosis, achieved via a combination of internal carotid artery (ICA) ligation and superficial temporal artery-middle cerebral artery (STA-MCA) bypass. Theoretically, management and exclusion of ICCAs alleviate the compression on the involved nerves.

Gradual thrombus retraction induces the involution of giant aneurysms through progressive fibrosis and reduction of mass effect (2). Carotid ligation facilitates thrombosis of all segments of the internal carotid artery, from cervical ligation to the bifurcation. Moreover, the ophthalmic artery may also undergo complete thrombosis.

In this report, we present cases of visual improvement observed in 2 patients out of 9 who were operated on for ICCAs at our institution between 2016 and 2019. The management involved carotid ligation combined with STA-MCA bypass, with comparative imaging assessments of the aneurysm, internal carotid artery, and ophthalmic artery pre- and post-operatively. The visual outcomes are documented and analyzed within this publication.

MATERIALS AND METHODS

Patients

We conducted a retrospective analysis of clinical and imaging data from September 2016 to December 2019 for patients treated at our institution. The study included only those patients with intracranial cavernous carotid aneurysms (ICCAs) presenting with symptoms such as cranial nerve palsy or persistent headaches unrelieved by conventional analgesics. Asymptomatic patients or those with episodic headaches were not immediate candidates for this intervention; instead, they were referred for endovascular assessment and treatment abroad. Furthermore, patients in a coma with ruptured ICCAs

in the intensive care unit are not managed using the proposed approach procedure.

Preoperative Evaluation

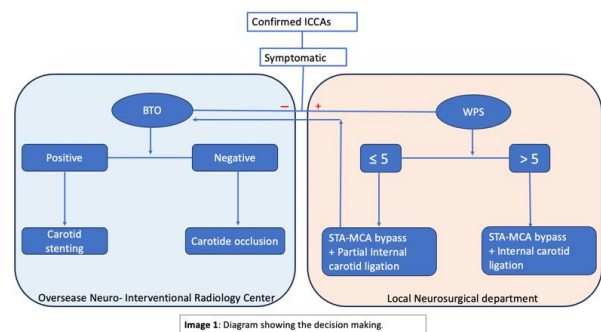
A clinical examination, encompassing neurologic and ophthalmologic assessments, is conducted during the initial visit. The modified Rankin scale is also evaluated. Epidemiologic data and imaging findings—including side, size, and location on the internal carotid artery—are documented in **Table 1**.

Table 1. The Baseline Data of Patients with Complex Internal Carotid Aneurysms

Patient Number	Age (years)	Sex	Side	Size (mm)	Symptoms	Location
1	36	Male	Right	37.81	Headaches, seizure, hallucination, visual field deficit	Paraclinoi
2	20	Female	Right	28.56	Headaches, homolateral visual acuity decrease	Paraclinoi
3	19	Female	Right	45.83	Headaches, Bilateral visual loss	Paraclinoi
4	68	Female	Left	18.63	Headaches, blurred vision, decrease in visual acuity	Intracavern
5	30	Male	Left	25.70	Headaches, homolateral visual loss, 3rd nerve palsy	Paraclinoi
6	65	Female	Right	44.20	Cavernous sinus syndrome	Transition
7	45	Female	Right	22.10	Headaches, 3 rd nerve palsy,	Intracavern
8	54	Female	Left	16.10	Headaches, left eye blurred vision.	Paraclinoi
9	48	Male	Left	23.52	Headaches, homolateral visual loss	Transition

Decision Making

In the absence of balloon test occlusion within the country, we evaluated the patients based on their Willis Polygon Score (WPS) as described by L. Houari et al. (3). The decision-making organizational chart is outlined in Image 1, where WPS 7 indicates the number of missing segments in the Willis polygons.



Operative Procedure

The patient is prepared and positioned in accordance with standard procedures, with access established to the cervical region on the side of the aneurysm. The carotid bifurcation is meticulously dissected, and the ligation at the origin of the internal carotid artery (ICA) is performed using either a secure double knot spaced one centimeter apart or a single knot occluding approximately three-quarters of the carotid lumen (a knot designed to permit passage of the hook's tip), employing a non-resorbable suture.

A superficial temporal artery is dissected, harvested, and clipped distally to a length of 10 cm. The extremity is prepared for the anastomosis, a temporo-parietal bone flap is removed, the dura mater is opened, and an M3 artery is selected after the dissection of the outer part of the sylvian fissure. A temporal clip is applied, and a termino-lateral anastomosis is performed with silk sutures.

In the absence of indocyanine green, the anastomosis is assessed using surgical techniques that involve gently occluding the proximal and distal segments of the M3 artery while observing arterial pulsation.

The entire procedure is conducted under normotensive conditions, with a slight elevation observed during the temporary clipping phase of the anastomosis.

The dural closure initially begins at the frontal aspect and then progresses to the temporal region, using a pedicle layer of temporal muscle to ensure a secure entry of the STA without undue compression.

In addition to the preoperative measures, saline-diluted heparin and calcium inhibitors are administered at the anastomosis site. In the intensive care unit, the patient receives an intravenous salicylic acid following a three-hour computed tomography scan.

RESULTS

A total of nine patients underwent surgical intervention for ICCAs during the study period. All patients received internal carotid artery (ICA) ligation combined with superficial temporal artery (STA)-middle cerebral artery (MCA) anastomosis. The cases are sequentially numbered from 1 to 9, each exhibiting distinct characteristics detailed in Table 1. Epidemiological data analysis indicated a predominance of female patients, with an average age of 43 years. Preoperative visual deficits were observed in seven out of nine patients, third nerve palsy in three patients, and one patient presented with seizures.

The aneurysm sizes ranged from 16.10 mm to 45.83 mm, with an average size of 29.16 mm. Five of the nine aneurysms were classified as giant and were primarily located in the paraclinoid segment of the internal carotid artery.

In addition to the STA-MCA anastomosis, all patients underwent ICA ligation at its cervical origin.

1. The surgical outcomes:

The evaluation of surgical outcomes employing Angio CT and Angio MR across multiple sequences is documented in Table 2, providing a comprehensive assessment of aneurysm morphometry, including thrombosed regions. Both the aneurysm and the internal carotid artery (ICA) were examined at 6 and 12 months using steady-state magnetic resonance imaging.

Patient Number	Anastomosis Patency (Angio CT)			Aneurysm Thrombosis (Angio MR)	
	3 hours after surgery	1 month	3 months	6 months	12 months
1	Yes	Yes	Yes	80%	100%
2	Yes	Yes	Yes	100%	100%
3	No	No	No	80%	100%
4	Yes	Yes	Yes	100%	100%
5	No	No	No	100%	100%
6	Yes	Yes	Yes	100%	100%
7	Yes	Yes	Yes	100%	100%
8	Yes	Yes	Yes	100%	100%
9	Yes	No	No	100%	100%

The aneurysm exclusion via thrombosis is achieved in all cases, with 7 out of 9 patients attaining 100% exclusion and the remaining 2 patients achieving 80% at six months; by twelve months, the exclusion rate reaches 100%. Postoperative assessment of the STA-MCA anastomosis was conducted at three hours, one month, and three months following surgery. The anastomosis remained patent in 7 out of 9 patients and was non-patent in 2 after three hours. Notably, in one patient, the anastomosis transitioned from being patent to thrombosed at one-month post-surgery.

2. Clinical outcomes:

The assessment of clinical outcomes in comparison to the preoperative status is documented in Table 3. The modified Rankin scale indicates stabilization across all patients. Ophthalmologic evaluations remained stable, with no alterations observed in the visual field; furthermore, an improvement in visual acuity was noted in two cases at the three-month follow-up.

Patient Number	Modified Rankin scale			Ophthalmic evaluation				
	Admission	Discharge	3 Months	Visual field		Acuity at the side of the aneurysm		
				Preop	3 months	preop	3 months	12 months
1	1	1	1	HH	HH	18/20	18/20	18/20
2	1	1	1	N	N	6/20	10/20	10/20
3	3	3	3	BB	BB	0/20	0/20	0/20
4	1	1	1	N	N	10/20	16/20	16/20
5	1	1	1	MB	MB	0/20	0/20	0/20
6	1	1	1	MB	MB	0/20	0/20	0/20
7	1	1	1	N	N	6/20	6/20	6/20
8	1	1	1	N	N	16/20	16/20	16/20
9	1	1	1	N	N	4/20	4/20	4/20

N: normal, **HH:** homonymous Hemianopsia, **BB:** Binocular Blindness, **MB:** Monocular Blindness.

A patient is reported to have a lateral homonymous hemianopsia with preserved visual acuity. This clinical condition is attributed to ipsilateral optic tract compression caused by the aneurysm (see Images 2, 3, 4). Heme MRI sequences reveal hemosiderin deposition and parenchymal destruction of the optic tract, indicated by an arrow in Image 3. The patient's visual impairment persists unchanged following surgical intervention.

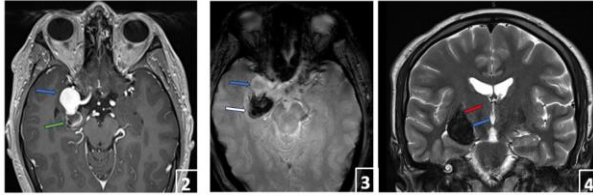


Image 2: Axial T1-weighted enhanced brain MRI showing a prebifurcation right internal carotid aneurysm (blue arrow) and a posterior pseudo-aneurysmal sac partially thrombosed, compressing the optic tract (green arrow).

Image 3: axial T2* weighted gradient echo MRI, shows the aneurysm (blue arrow) and the hemosiderin deposition in the parenchyma corresponding to the optic tract (white arrow), which is a sign of previous aneurysm bleeding.

Image 4: Coronal T2-weighted MRI showing an aneurysm in the ambient cistern measuring 25 mm on this axis (blue arrow). The superior part of the aneurysm compresses the optic tract (red arrow).

Binocular blindness was observed in a single patient, attributable to the mass effect exerted by the large aneurysms on the sellar and suprasellar regions (see blue arrows in images 5, 6, and 7).

The optic chiasm is displaced anteriorly and appears laminated vertically as a thin layer by the anterior segment of the aneurysm (indicated by the yellow arrow in image 5). Notably, this patient's visual impairment persisted postoperatively.

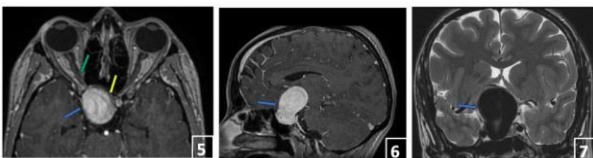


Image 4: Axial T1-weighted enhanced brain MRI showing a giant right internal carotid aneurysm (blue arrow) measuring 45,83 mm at its greatest axis, the optic nerves are normal, the chiasm is stretched anteriorly (yellow arrow) by the aneurysm.

Image 5: Sagittal T1-weighted enhanced brain MRI showing a giant right internal carotid aneurysm (blue arrow) enlarging and eroding the anterior wall and the floor of the sella turcica.

Image 6: Coronal T2-weighted MRI showing a giant internal carotid aneurysm (blue arrow). The superior part of the aneurysm raises the floor of the third ventricle.

3. Illustrative case

A 45-year-old woman with a history of embolization of a paraclinoid internal carotid aneurysm five years prior presents with persistent headaches and a left eye visual acuity of 10/20. Cerebral angiography revealed revascularization of a left paraclinoid aneurysm, characterized by coiling material at the base of an enlarged fusiform aneurysm (purple arrow, Image A), measuring 16 x 11 mm. The patient underwent a homolateral Hunterian internal carotid artery ligation and superficial temporal artery middle cerebral artery (STA-MCA) bypass. Postoperative assessment indicated stability, with a reduction in headache severity and improvement in visual acuity from 10/20 to 16/20 at three months. Follow-up imaging, including Angio MRI at three months, confirmed aneurysm thrombosis and patency of the anastomosis (red arrow, Image B). The internal carotid artery appears completely thrombosed, while the ophthalmic artery remains patent (blue arrow, Image C), as indicated by the yellow arrow.

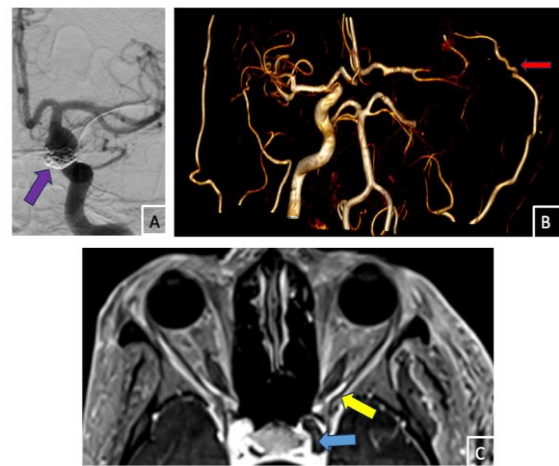


Image A: Cerebral angiography illustrates the revascularization of a left paraclinoid aneurysm, with coiling material positioned at the base (indicated by the purple arrow) of an enlarged fusiform aneurysm.

Image B: The anteroinferior three-dimensional reconstruction view derived from the Angio MR reveals the exclusion of the aneurysm and the internal carotid artery from the circulatory system. Additionally, the patency of the anastomosis and the dilation of the recipient artery are evident (indicated by the red arrow) at the three-month follow-up.

Image C: Axial enhanced T1 MRI, illustrating complete carotid thrombosis (blue arrow) with the ophthalmic artery remaining patent (yellow arrow).

DISCUSSION

The morphology and hemodynamic alterations associated with intracranial carotid cavernous aneurysms (ICCA) exert varying effects on the adjacent neurovascular structures, resulting in visual impairment. The reported recovery rates for visual disturbances caused by aneurysm compression range from 50% to 65% (4,5), whereas the recovery rate for external ophthalmoplegia is approximately 70% (6). Limited literature addresses these differential recovery rates. Matano *et al.* (7) elucidated that the disparity is attributable to differences in embryological origin and development between the optic nerve and other cranial nerves, leading to distinct responses to compression.

These discrepancies can be elucidated by considering two types of visual impairment. The first pertains to intra- and extraocular motility deficits, predominantly caused by the mass effect exerted on the oculomotor, trochlear, and abducens nerves, which are primarily situated within the cavernous sinus. The primary manifestation is ophthalmoplegia.

The second type involves impairment of the optic nerve itself. It is posited that, in addition to the mass effect, hemodynamic alterations—such as hypoperfusion resulting from a steal phenomenon observed during angiographic dye injection and the water hammer effect transmitting shock waves to the optic nerve during each systole—contribute to the pathology.

From a theoretical standpoint, the surgical exclusion of ICCAs diminishes their influence on neural structures. Practically, indirect exclusion through thrombosis, achieved via internal carotid artery (ICA) ligation combined with superficial temporal artery-middle cerebral artery (STA-MCA) bypass, is regarded as the most effective intervention. Carotid ligation promotes thrombus formation, thereby reducing the risk of further neural compromise and restoring vascular stability, particularly along the internal carotid artery, from the cervical ligature to the bifurcation, including the origin of the ophthalmic artery. Strother *et al.* (2) observed that gradual thrombus retraction causes aneurysm involution through progressive fibrosis and reduction of the mass effect. Matano *et al.* (7)

reported that visual acuity correlates with a poorer prognosis following treatment of giant paraclinoid aneurysms than with ophthalmoplegia.

In our cohort of nine patients undergoing surgical treatment for ICCAs via ICA ligation and STA-MCA bypass, we observed stabilization of visual status. Notably, an improvement in visual acuity was documented in two patients despite complete ICA thrombosis and a paradoxically patent ophthalmic artery (see image C of the illustrative case).

This outcome can be attributed to the chronic hypoperfusion induced by the steal effect, which facilitated the effectiveness of the anastomotic circuits. Specifically, there are three known anastomoses connecting the ophthalmic artery (OA) and the middle meningeal artery (MMA) (7,8): the recurrent meningeal branch, the anterior falx artery, and the anastomotic branch with MMA, all of which sustain flow in the ophthalmic artery in the event of ICA thrombosis. In such cases, these anastomoses enlarge and become the primary sources of blood supply to the orbital region and optic nerve.

A limitation of our study is the absence of angiographic investigation into the anatomical variations of the ophthalmic artery, its branches, and the associated anastomotic circuits. Furthermore, due to the stabilization of visual fields at three months post-treatment, no repeat angiographic assessments were performed at the twelve-month mark.

CONCLUSIONS

ICA ligation with STA MCA bypass is an effective treatment of ICCAs; the exclusion of the aneurysms by thrombosis relieves the extra and intraocular motor nerves as well as the optic nerve. Despite ICA thrombosis, ophthalmic artery flow, according to anatomic variation, is secured by anastomotic circuits.

ABBREVIATIONS

ICCAs: Internal Carotid Complex Aneurysms, **ICA**: Internal Carotid Artery, **STA**: Superficial Temporal Artery, **MCA**: Middle Cerebral Artery, **ICU**: Intensive Care Unit, **WPS**: Willis Polygon Score, **BTO**: Balloon test Occlusion, **MRI**: Magnetic Resonance Imaging, **CT**: Computerized Tomography, **3D**: Tridimensional, **IV**: Intravenous, **mRS**: modified Rankin Scale, **A1**: Precommunicant segment of the anterior cerebral artery, **M3**: Branches of the middle cerebral artery located in the sylvian fissure. **N**: normal, **HH**: homonymous Hemianopsia, **BB**:

Binocular Blindness, **MB:** Monocular Blindness, **Preop:** preoperative status, **OA:** Ophthalmic artery, **MMA:** Middle meningeal artery.

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