CASE REPORT

Spontaneous thrombosis of a giant cavernous-carotid aneurysm with simultaneous ipsilateral complete parent artery occlusion: a rare phenomenon and review of the literature

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ABSTRACT

Cavernous-carotid artery (CCA) aneurysms represent about 3-5% of all intracranial aneurysms. Spontaneous thrombosis of a CCA aneurysm with simultaneous occlusion of its parent vessel is an extremely rare phenomenon with few reported cases in the literature offering different management strategies. A 54-year-old Asian female presented with a one day-history of painless left eye conjunctival injection, proptosis, and features of cavernous sinus syndrome (cranial nerve III, IV, V1, V2, and VI palsies). Imaging revealed a giant thrombosed CCA aneurysm measuring 3.6cmx3.4cm with complete thrombosis of the left cervical internal carotid artery (ICA) and adequate collaterals from the anterior and posterior communicating artery and branches of the left external carotid artery. Management was conservative with antiplatelet therapy and close clinical-radiological follow-ups. The outcome was satisfactory. Data in the literature on this condition is limited due to its exceedingly rare occurrence. The majority of patients do well via a conservative approach and surgery is rarely indicated. For clinically stable patients, especially those with adequate collateral circulation and tolerance to Balloon Test Occlusion, we advocate for a conservative approach and initiation of anti-platelet therapy to treat these patients. Emphasis is needed on close serial clinical-radiological surveillance in these cases to monitor the propagation of the thrombus as well as the development of new and/or enlarging pre-existing aneurysms in the contralateral ICA circulation.

KEYWORDS: Cavernous-carotid artery; cavernous sinus syndrome; giant aneurysm; internal carotid artery thrombosis.

INTRODUCTION

The incidence of cavernous-carotid artery (CCA) aneurysms is 3-5% of all intracranial aneurysms, while that of internal carotid artery (ICA) aneurysms involving the cavernous segment is 14% [1,2]. They are rare pathological entities, carrying low bleeding risk [2]. The prevalence of spontaneous intraluminal thrombosis in these giant intracranial aneurysms is up to 20% [2,3]. However, simultaneous thrombosis of its parent vessel is uncommon and intriguing [3]. Various etiologies of CCA have been reported, including infectious, traumatic, and idiopathic causes. The clinical manifestations of CCA aneurysms include features of cavernous sinus syndrome. such as ophthalmoplegia, ptosis, proptosis, retro-orbital pain and facial numbness [1,2].

Depending on the clinical and radiological manifestations of a spontaneously thrombosed giant CCA aneurysm along with its parent artery, there are various management options available. For most patients, a conservative approach involving regular follow-ups is appropriate [1,3]. Here we highlight an interesting case of a 54-year-old Asian patient who presented with features of cranial nerve II, III, IV, V (V1 and V2) and VI palsy and was diagnosed with a giant thrombosed CCA aneurysm with simultaneous complete occlusion of the left internal carotid artery.

CASE PRESENTATION

A 54-year-old right-handed, Asian female, with unremarkable medical or surgical history, presented to the ophthalmology

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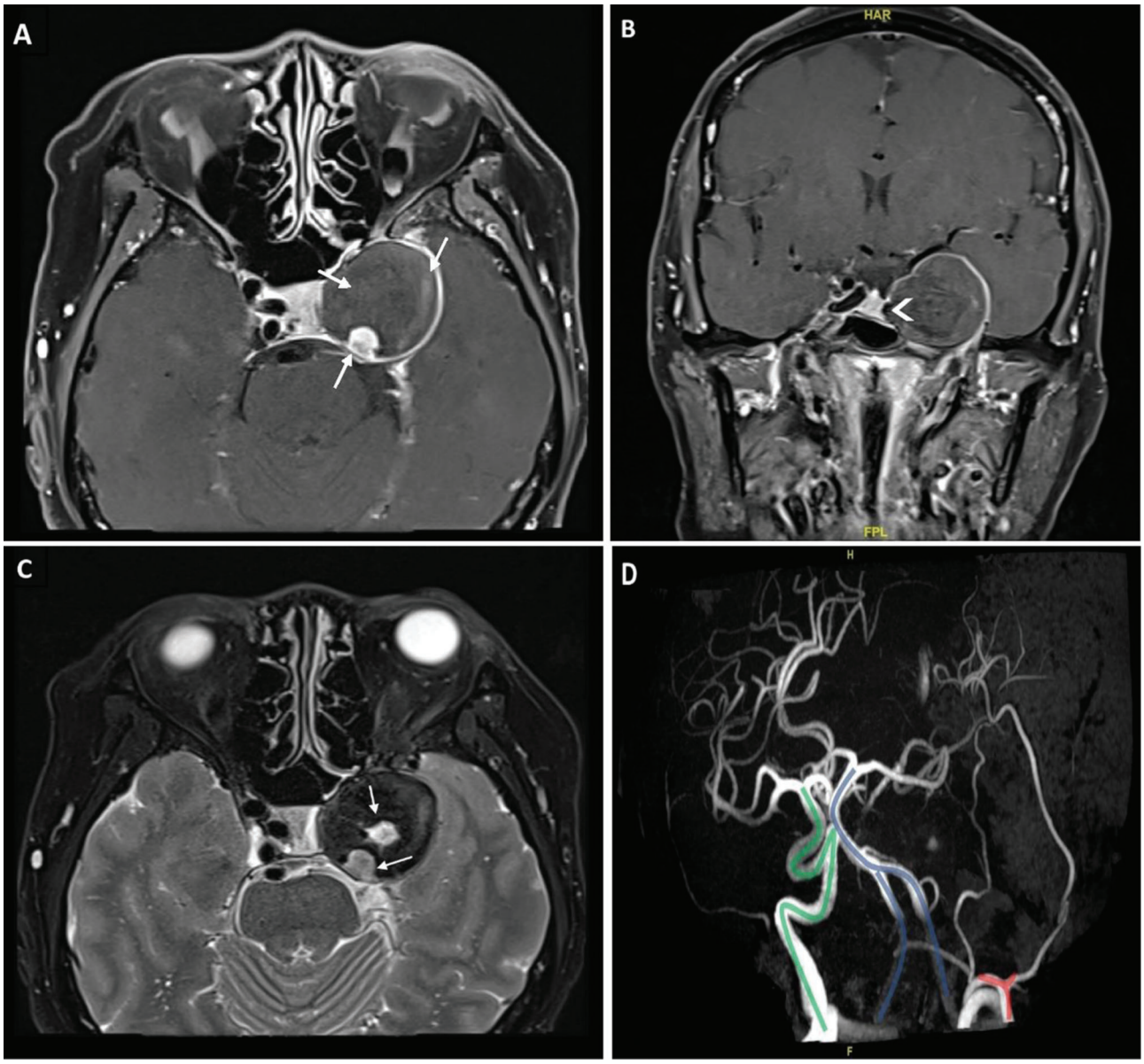

Fig. 1. MRI Orbit T1 axial (A) and coronal views (B) with contrast, and T2 axial (C) showing a giant cystic lesion measuring 3.6cmx3.4cm along the course of the cavernous segment of the left ICA (arrowhead), likely representing an aneurysm. The differential signal within the aneurysm likely represents a thrombus of different ages (arrows). (D) MR Angiography showing the right ICA (green highlight), vertebral and basilar arteries (blue highlight) and left external carotid artery (red highlight) with no definite flow within the aneurysm and absent left ICA secondary to thrombosis. No evidence of subarachnoid hemorrhage or other abnormalities on the MRI.

outpatient clinic with a one-day history of toothache, painless drooping of the left eyelid, conjunctival injection, and double-vision. There was no history of trauma. Urgent Magnetic Resonance Imaging (MRI) Orbit with contrast and MR angiography/venography was requested by the ophthalmologist and revealed a giant lesion measuring 3.6cmx3.4cm along the course of the cavernous segment of the left ICA, likely representing an aneurysm (Figure 1). Following the MRI findings, the neurosurgery team were consulted and the patient was later transferred and admitted to the neuro-intensive care unit at our institute. On examination, the patient was conscious, alert, and oriented with a Glasgow Coma Score (GCS) of 15/15, and vital signs were only significant for blood pressure of 160/100. Ocular findings showed left-sided complete ptosis and ophthalmoplegia consistent with cranial nerve (CN) 3, 4, and 6 palsy, horizontal diplopia, and reduced sensation in the ophthalmic and maxillary distribution of the trigeminal nerve on the left side. Left eye proptosis was also noticeable. Right Pupil was 2mm, equal, round and reactive to light. However, the left pupil was 5mm, round, and non-reactive to light both directly and consensually. A formal visual field assessment revealed defects as shown in Figure 2. The remainder of the neurological exam was within normal limits and there was no carotid or orbital bruit.

Cerebral digital subtraction angiography (DSA) was performed the following day and showed complete thrombosis at the cervical segment of the left ICA with the normal

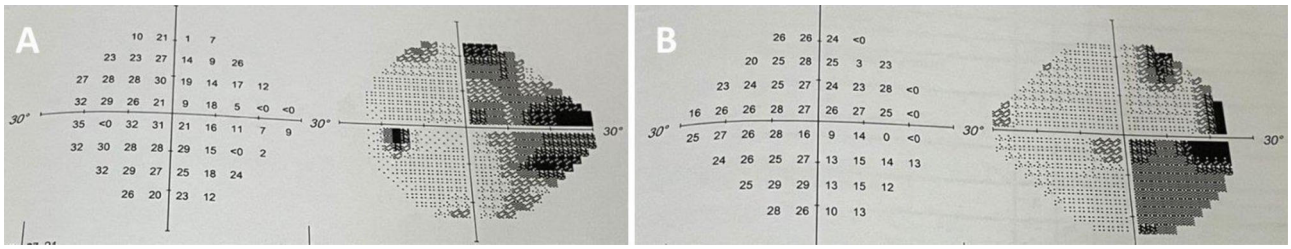

Fig. 2. Humphrey's Visual Field test showing left (A) and right eye (B) defects involving the left nasal field and right temporal field (right incongruous homonymous hemianopia).

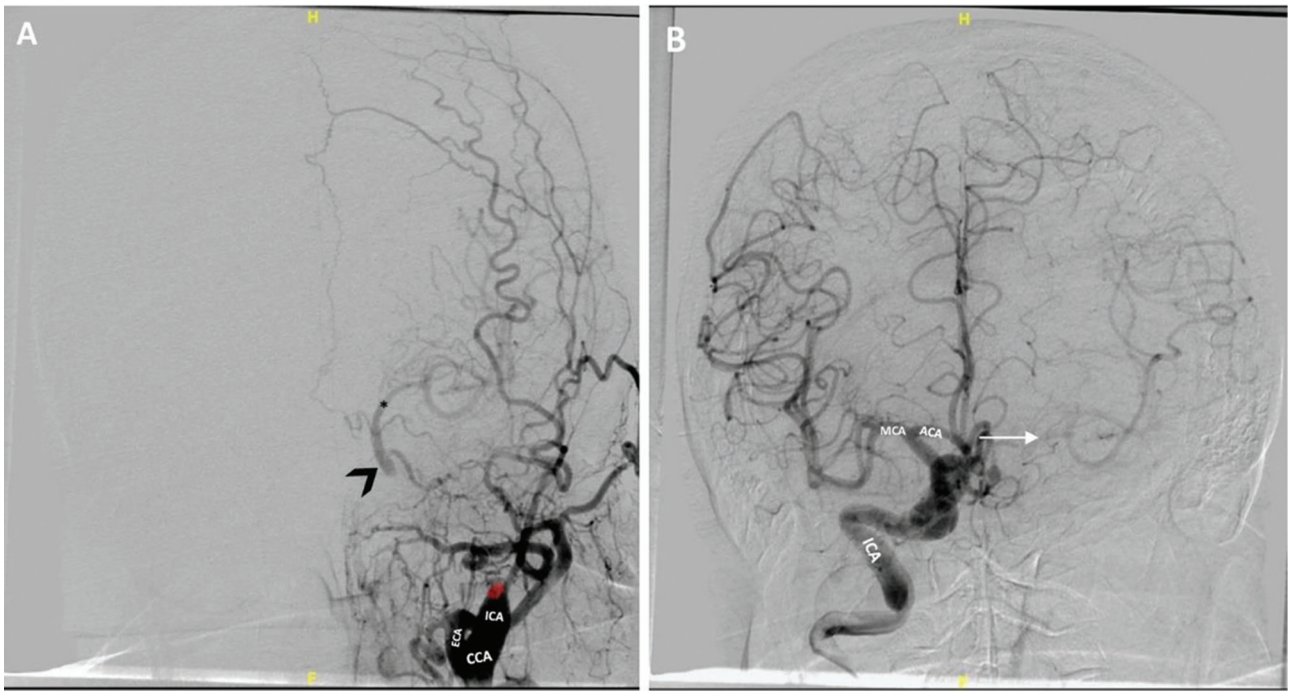

Fig. 3. Cerebral DSA showing occlusion of the left ICA from the cervical segment (C1) (red highlight) (A). The left common (CCA) and external carotid artery (ECA) are labelled (A). Branches from the ECA appears to contribute to the collaterals and reconstruction of the left terminal ICA segment (arrowhead) and its branches such as the left middle cerebral artery (MCA) (asterisk). The right internal carotid artery shows normal flow (B). The right MCA and anterior cerebral artery (ACA) are labelled (B). There is cross-flow (arrow) from the right to the left side via the anterior communicating artery (AcomA) and posterior communicating artery (PcomA) (B). No aneurysmal dilatations were identified.

flow in the right ICA and the presence of adequate collateral circulation via the anterior communicating (AcomA) and posterior communicating artery (PcomA), as well as branches from the left external carotid artery (ECA) (Figure 3). Balloon Test Occlusion (BTO) was not performed since the left ICA was already completely occluded. A final diagnosis of spontaneous thrombosed giant cavernous-carotid artery aneurysm with simultaneous complete thrombosis of its parent vessel was made. Follow-up CT Angiography with 3D-reconstruction of the vessels was done the next day and clearly demonstrated the left ICA occlusion along with the collateral blood flow (Figure 4). CT Perfusion was significant for left cerebral hypoperfusion but no signs of infarction (Figure 5).

Since the patient was clinically stable with preserved neurological status, good compensatory collateral circulation, and no radiological evidence of infarction, we decided against surgery and proceeded with a conservative approach consisting of aspirin therapy and close serial clinical-radiological monitoring in the neurosurgical outpatient clinic. At 2-month follow-up, CT Brain showed a stationary course of the aneurysm size (Figure 6), and clinically there was a slight improvement in ophthalmoplegia, ptosis, and facial sensation.

DISCUSSION

Cavernous Carotid Artery (CCA) aneurysms represents a challenge for interventional neuro-radiologist and vascular neurosurgeons. They are rare and compose 3-5% of all intracranial aneurysms with a variable presentation and course ranging from asymptomatic to gradual deterioration to spontaneous improvement [4,5]. Giant Intracranial aneurysms (ICGA) have been arbitrarily defined as being greater than 25mm [6]. The spontaneous thrombosis of ICGA is not uncommon; with a reported incidence of 60% for partial thrombosis and 13-20% for complete thrombosis, respectively [7].

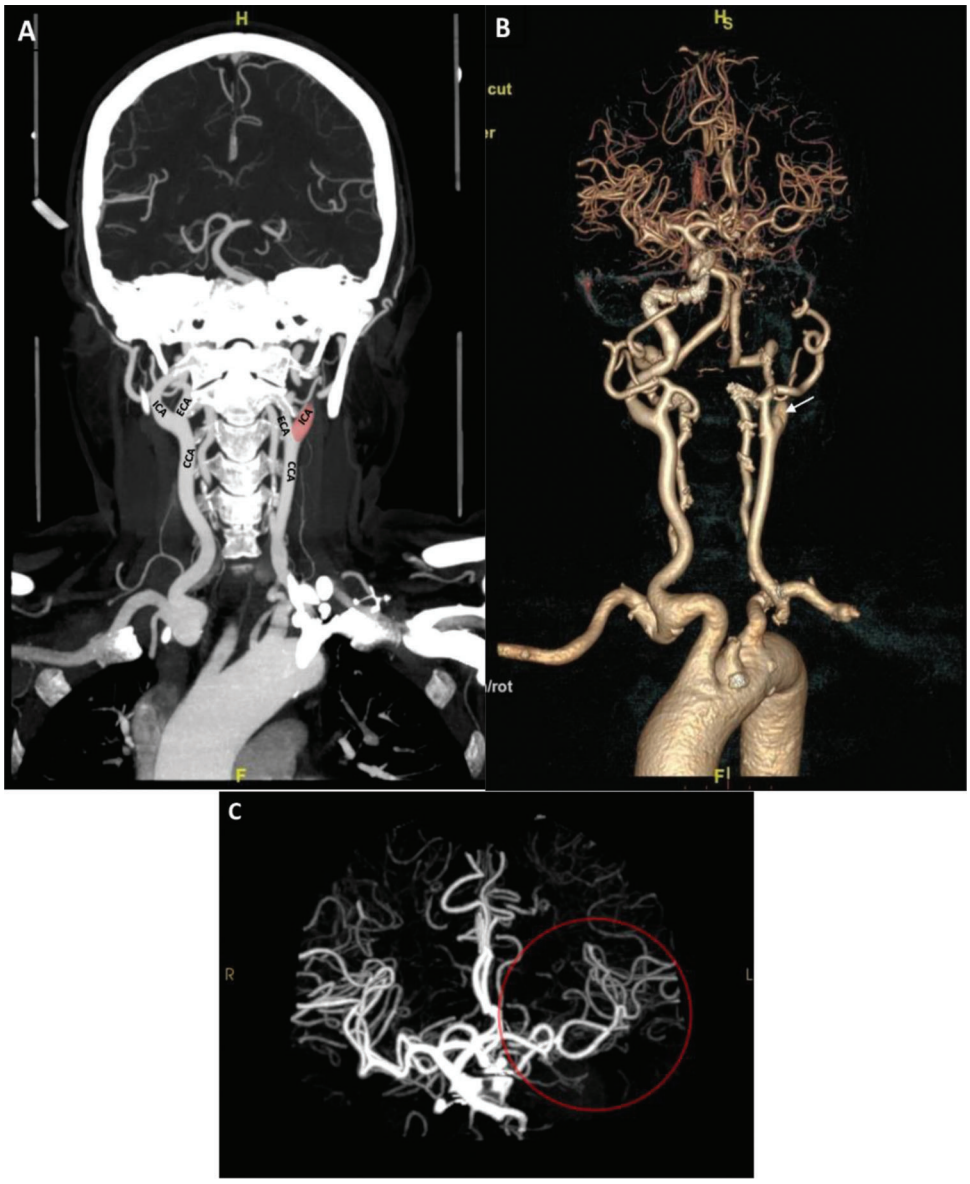

Fig. 4. (A) CT Angiography showing occlusion of the left ICA (red highlight) from the cervical (C1) to the clinoidal (C5) segment. The common carotid (CCA) and ECA are labelled. (B and C) 3D-reconstruction of the vessels clearly illustrating the left ICA occlusion (arrow) and adequate cerebral collateral circulation from cross-flow (red circle) via the AcomA, PcomA and branches of the ECA.

However, simultaneous thrombosis of the parent vessel harboring the aneurysm is an extremely rare occurrence, mostly consisting of case reports in the literature. Such reported cases involve the internal carotid artery (ICA), middle cerebral artery (MCA), and posterior cerebral artery (PCA) [8-22]. To the best of our knowledge, there have been 19 reported cases of spontaneous ICA thrombosis in association with a large or giant cavernous-carotid aneurysm (Table 1). Here, we present an additional uncommon case of a partially thrombosed giant CCA aneurysm with simultaneous spontaneous ICA thrombosis that was managed conservatively due to inability to access the aneurysm endovascularly.

The various clinical manifestation of CCA aneurysm is best explained by the close anatomical proximity of the cavernous-carotid artery to cranial nerves (CN) 3, 4, the ophthalmic (V1) and maxillary (V2) branch of the trigeminal nerve, and CN6. Thus, the majority of patients present with features of mass effect such as complete or incomplete ophthalmoplegia, headache, retro-orbital pain, ptosis, and facial paresthesia or pain (Table 1). Anterior extension of the giant aneurysm through the supraorbital fissure can occur and manifest with proptosis and progressive visual loss due to optic nerve compression [16,20]. Ischemic manifestations such as hemiplegia and dysphasia can also occur due to lateral extension and compression of the MCA or perisylvian cortex by the giant aneurysm, intolerance to ICA occlusion, inadequate collateral circulation and/or distal thrombo-embolism [13,21].

The phenomenon underlying spontaneous thrombosis of ICGA has been thought to be due to endothelial damage with subsequent thrombosis secondary to the hemodynamic stress

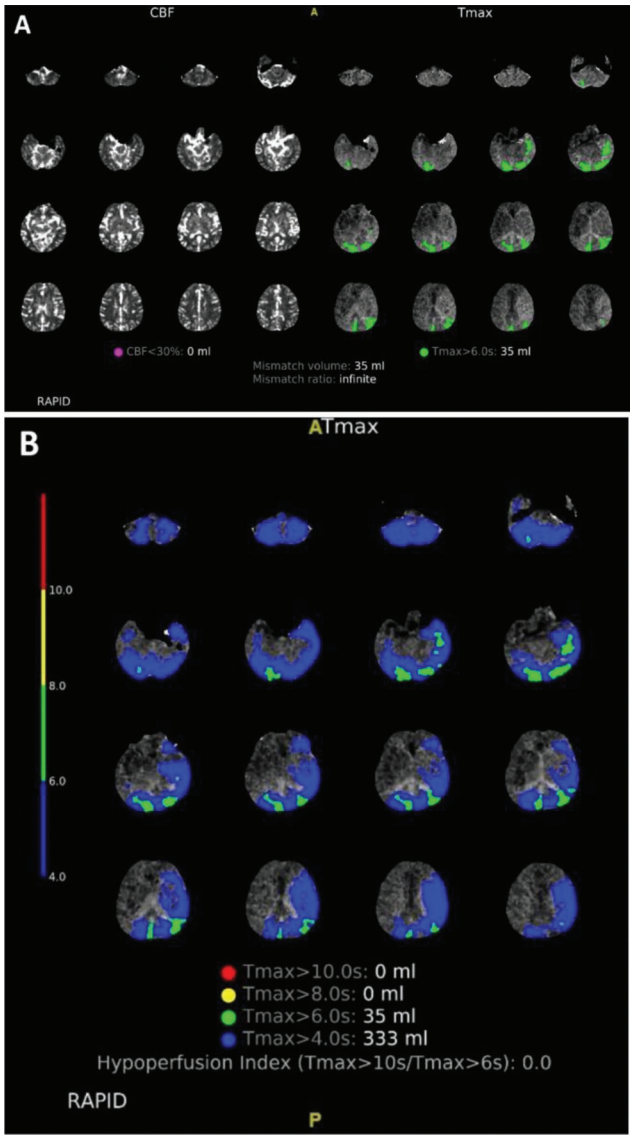

Fig. 5. CT Perfusion imaging processed by RAPID Artificial Intelligence software showing no ischemic core infarcts (A: Cerebral blood flow less than 30% volume is 0mls) and perfusion deficit with a time-to-maximum (Tmax)-more-than-6-seconds volume of 35mls, indicating hypoperfusion in the left parieto-temporal region (green). A low Hypoperfusion Index correlates with presence of good collateral circulation (B).

on the aneurysmal wall. Several predisposing factors such as increased aneurysm dome to neck ratio and chronic aneurysms are reported to increase the risk of intra-aneurysmal thrombosis [4,15,16,20]. However, the mechanism for the spontaneous thrombosis of the aneurysm along with its parent artery is not completely understood. It can occur on follow-up or at presentation as occurred in our and majority of the previously reported cases [14]. The predominant occurrence of this phenomenon in large or giant aneurysms of the CCA supports previously reported pathophysiological hypotheses of local stretching, compression, and/or distortion of the ICA by the giant aneurysm. Furthermore, the presence of rigid dural folds, anterior and posterior clinoid processes, and sellar walls in this location, in addition to the natural anatomical angulation of the CCA promotes distortion of the

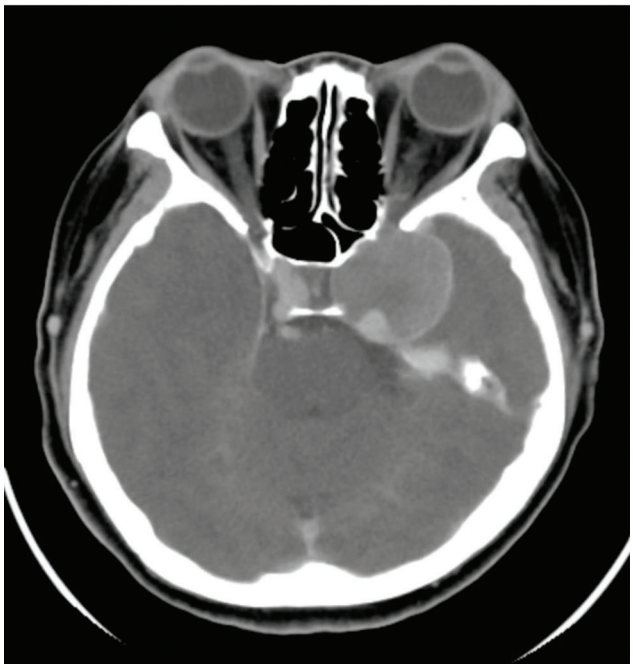

Fig. 6. Follow-up CT Brain with contrast at 2 months showing stationary size of the partially thrombosed aneurysm with no signs of infarction.

ICA, thereby resulting in stasis of blood flow and subsequent thrombus formation [12,14,17,22,23]. Moreover, the start of the parent vessel thrombosis just proximal to the location of the aneurysm rather than at the carotid bifurcation favors retrograde thrombosis from the aneurysm into the parent vessel; another potential underlying mechanism that may have occurred in our case.

It is important to take into account the course of thrombosed intracranial aneurysms in association with simultaneous ICA occlusion to construct an effective management plan. Aneurysmal thrombosis may not be the end of the story; with further aneurysmal growth, persistent mass effect, distal thrombo-embolism, and aneurysmal re-canalization being all potential occurrences even with fully thrombosed aneurysms, as long as the parent vessel is patent [9,10,22,24,25]. However, the co-existent thrombosis and hence non-patency of the parent vessel raises the question if recanalization of the thrombosed aneurysm can occur. Even though the chances are extremely narrow, it is possible and has been reported in a case of a thrombosed PCA aneurysm with parent vessel occlusion followed by recanalization of the aneurysmal neck and PCA several years later [10]. To date, of all the reported cases of thrombosed CCA aneurysms with ICA occlusion, no recanalization has been reported [11-22]. Therefore, it appears that the simultaneous parent vessel thrombosis, especially of the ICA, may act as a safeguard for recanalization and rupture prevention of a thrombosed aneurysm.

Nevertheless, the occurrence of simultaneous parent vessel thrombosis may not always be to our advantage, and it depends on the rapidity of the thrombosis as well as the presence of sufficient collateral blood flow [15]. Acute vessel occlusion by a thrombosed aneurysm via direct or indirect mechanisms mentioned previously, even in the presence of adequate cross-flow can produce neurological deficits and/

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Table 1. Brief summary of previously reported cases of spontaneous thrombosis of cavernous-carotid artery aneurysms with simultaneous internal carotid artery thrombosis

		Clinical presentation							Management		
		Cranial Nerves Involved									
Author, year of case report	Age (years), sex	II	III	IV	V_1	V_2	VI	Other Features	Neurosurgical/ Endovascular	Conservative	Outcomes
Mikabe et al., 1980 [19]	62, F	✓	✓	✓			✓	HA	Left carotid artery ligation		Resolution of II, III, IV. Persistent VI.
Whittle et al., 1982 [15]	27, M		✓		✓	✓	✓	HA, Diplopia, Proptosis, red eye	Aneurysmotomy followed by thrombectomy		Improvement in symptoms. Persistent diplopia and opthalmoplegia
Gautier et al., 1986 [20]	65, F		✓	✓			✓	Right hemiplegia, aphasia		Unknown	Improvement in hemiparesis and aphasia. Persistence of opthalmoplegia
Sato et al., 1990 [13]	49, M		✓		✓		✓	HA, Diplopia		✓	Improvement in symptoms, III and V1.
	21, M	✓						Left hemiparesis, dysarthria	STA-MCA by-pass BTO intolerance		Improvement in dysarthria and left hemiparesis.
Kurokawa et al., 2001 [14]	60, F						✓	HA, Diplopia	High flow vein EC-IC bypass BTO intolerance		Improvement in diplopia and VI
	50, F		✓		✓		✓	Diplopia, hyponatremia		✓ (BTO intolerance)	Improvement in III and V1. Persistent VI
Tsutsumi et al., 2002 [12]	75, M	✓	✓	✓	✓	✓	✓			✓	Improvement
Ray et al., 2002 [17]	66, F		✓			✓		HA, features of hypopituitarism		✓	Resolution of headache and V2. Partial recovery of III.
Perrini et al., 2005 [16]	47, M				✓	✓		Right ROP, diplopia		✓	Resolution of headache and diplopia. Persistent V1, V2
Vasconcellos et al., 2009 [21]	47, F		✓	✓	✓	✓	✓	HA, ROP		✓	Resolution of headache and pain. Persistent III, IV, VI, V1, V2
	44, F		✓	✓	✓	✓	✓	HA, Left ROP		✓	Resolution of headache, pain and III. Persistent VI.
	65, F		✓	✓	✓		✓	HA, Right ROP, diplopia		✓	Resolution of headache and pain. Persistent III, IV, VI, V1
	84, F		✓	✓	✓	✓	✓	HA, Left ROP		✓	Resolution of headache and pain. Improvement in III, IV, VI, V1, V2
	19, M		✓					HA, Left ROP		✓	Resolution of III
Sastri et al., 2013 [18]	65, F		✓		✓	✓				✓	Improvement in ophthalmopelgia. Persistent V1, V2
	55, F	✓			✓		✓	Seizures		✓	Improvement in opthalmoplegia, V1. Persistent II
Das et al., 2018 [7]	45, M							HA, altered LOC, vomiting		✓	Full recovery
Yamagami et al., 2021 [11]	68, F		✓		✓	✓		Diplopia	Endovascular thrombus aspiration followed by flow-diverter insertion		Improvement in III, V1, V2
Present case	54, F		✓	✓	✓	✓	✓	Conjunctival injection, toothache, proptosis, diplopia		✓	Improvement in ophthalmoplegia, ptosis, and facial sensation

ICA – Internal carotid artery, HA – Headache, II – optic nerve, III – oculomotor nerve, IV – trochlear nerve, VI – abducens nerve, V1 – ophthalmic branch of trigeminal nerve, V2 – maxillary branch of trigeminal nerve, EC-IC – Extracranial-to-Intracranial, BTO – Balloon Test Occlusion, ROP – Retro-orbital Pain, LOC – Loss of consciousness.

or ischemic changes on radiological imaging, as occurred in 3 of the 19 previously reported cases [13,14,21]. The remaining 17 patients, including our case, did not show any clinical ischemic manifestations despite complete ICA thrombosis. The phenomenon of new aneurysmal formation or growth of pre-existing ones on the contralateral ICA and its branches in such cases has been reported [14,21]. The likely underlying pathogenesis is due to the formation of collateral arterial networks secondary to progressive ICA occlusion [26]. These contralateral vasculature and collaterals are small and susceptible to the increased hemodynamic stress within them, thereby leading to aneurysm formation [26]. For that reason, it is important to iterate the need for long-term serial clinical and radiological surveillance in this cohort.

The management of spontaneously thrombosed giant CCA aneurysm and its parent vessel varies; with scarce data in the literature concerning this topic. A conservative approach involving regular follow-ups can be offered to most of these patients, especially asymptomatic and clinically stable ones, in the hopes that they will tolerate ICA occlusion over time. Of the 20 cases, 14 patients, including ours, were successfully managed using a conservative strategy, 4 with surgical intervention, and 1 with endovascular treatment (Table 1). Endovascular interventions for the aneurysm and/or parent vessel can also be offered and are associated with satisfactory results and less morbidity than surgical methods [11]. We initially opted for endovascular treatment. However, the distal left ICA beyond the cervical segment, and hence the aneurysm itself, could not be accessed due to its complete thrombosis, and hence procedure was aborted. Despite the hypoperfusion of some areas in the left cerebral hemisphere as evident on CT perfusion in our case, she remained neurologically intact given the excellent cross-flow from the contralateral side via a patent AcomA, PcomA, and branches from the left external carotid artery (ECA) seen on cerebral angiography, which further reinforced our decision to follow a conservative plan. Close surveillance is necessary to assess for any propagation of the thrombus, ipsilateral distal thromboembolism and formation of new or growth of existing aneurysms on the contralateral vasculature. The consensus to initiate antiplatelet as part of the conservative approach in these cases is not clear. We started our patient on aspirin mainly to prevent distal embolization, and this idea was also used in 4 of the previously reported cases, which showed clinical improvement [7,17,19]. Furthermore, improvement of these cases conservatively with antiplatelet therapy despite the persistent compressive effects of the thrombosed aneurysm may indicate that the ischemic effects play a role in the development of the patient's syndromic features [14].

Surgical interventions such as aneurysmotomy and thrombectomy as well as bypass procedures have been performed in a couple of previously reported cases. It is indicated in severely symptomatic cases with inadequate collateral flow, intolerance to BTO as well as clinical features and radiological evidence of severe ischemia, which was not observed in our case [13,14,16,20]. Outcomes in these cases were satisfactory.

This report aims to provide additional and supportive data to the literature on this scarce topic. We believe that there is a role for conservative management in these cases, and its outcomes in our and previous cases have evidently resulted in clinical improvement.

CONCLUSION

Thrombosis of a giant CCA aneurysm associated with simultaneous or subsequent spontaneous complete occlusion of the ICA is an exceedingly rare and intriguing encounter with several potential mechanisms explaining this uncommon phenomenon. Data in the literature on this condition is limited, consisting mostly of case reports and it appears that it develops gradually over time. The majority of patients do well via a conservative approach and surgery is rarely indicated. In our opinion, for clinically stable or asymptomatic patients, especially those with adequate collateral circulation and tolerance to BTO, we advocate for a conservative strategy and initiation of anti-platelet therapy to treat these patients. We also emphasize the need for close serial clinical-radiological surveillance to monitor the propagation of the thrombus as well as the development of new and/or enlarging pre-existing aneurysms in the contralateral ICA circulation.

Conflict of interest

There are no conflicts of interest to declare by all the authors.

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Ethical approval

Not applicable.

Consent

Written informed consent was obtained from the patient to publish this case report and accompanying images. On request, a copy of the written consent is available for review by the Editor-in-Chief of this journal.

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