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Surgical Neurology International

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SNI: Neurovascular

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Spontaneous thrombosis and calcification of giant cavernous carotid artery aneurysm: A rare case and management insights

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Case Report

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Received: 26 September 2023 Accepted: 19 February 2024 Published: 22 March 2024

DOI 10.25259/SNI_805_2023

Quick Response Code:



ABSTRACT

Background: Giant cavernous carotid artery aneurysms (>25 mm) are rare (3-5%), with some prone to spontaneous thrombosis (10-20% complete). We present a unique case of one of the largest aneurysms spontaneously thrombosing and calcifying.

Case Description: A 57-year-old with persistent right-sided headaches had a substantial hyperdense mass in the right middle cranial fossa, eroding petrous bone. Magnetic resonance imaging and digital subtraction angiography revealed a giant cavernous segment fusiform aneurysm of the right internal carotid artery (ICA) with spontaneous thrombosis and distal ICA occlusion. Collateral circulation maintains the cerebral blood supply. Despite anatomical challenges, conservative management was chosen due to the patient's stability.

Conclusion: This case highlights the complex interplay between thrombosed giant aneurysms and affected vessels, with unique features such as cross-flow, calcification, and bone erosion. We advocate conservative management for stable cases, supported by literature, emphasizing vigilant follow-up. This expands the spectrum of aneurysm presentations and encourages further research into their dynamics.

Keywords: Aneurysm calcification, Follow-up monitoring, Giant cavernous carotid artery aneurysms, Spontaneous thrombosis

INTRODUCTION

Giant cavernous carotid artery aneurysms are defined as those exceeding a size of 25 mm. According to existing literature, the incidence of cavernous carotid aneurysms is estimated to be in the range of 3-5%.^[8] These aneurysms occasionally develop partial intraluminal thrombosis, resulting in distal thromboembolism.^[4,14] Notably, large aneurysms (>15 mm) and giant aneurysms (>25 mm) are more susceptible to experiencing spontaneous intra-aneurysmal thrombosis. It is important to note that most instances of aneurysmal thrombosis are partial, with only 10-20% of patients experiencing complete thrombosis.[11]

Although spontaneous thrombosis of the parent artery is a rare occurrence, it poses a potentially catastrophic complication for these patients. In this current article, we present a unique case involving one of the largest aneurysms known to undergo spontaneous thrombosis and

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subsequent calcification. We provide a detailed clinical analysis of this case and conduct a comprehensive literature review of similar cases reported over the years. Furthermore, we explore potential management options in light of the limited existing literature on this subject.

CASE DETAIL

A 57-year-old male presented to our department, complaining of persistent right-sided headaches lasting two years. Physical examination revealed that the right temporalis muscles were less prominent, which may be an incidental finding in this patient. A prior non-contrast computed tomography scan showed a hyperdense mass in the right middle cranial fossa, eroding the right petrous bone [Figure 1a].

A contrast-enhanced magnetic resonance imaging displayed a hypointense lesion on T2 and fluid-attenuated inversion recovery images with subtle T1 hyperintensity. The mass measured 5×4.5 cm, exerting mass effect on adjacent structures and showing susceptibility-weighted imaging (SWI) blooming, suggesting blood products or calcifications. Post-contrast imaging hinted at a large calcified aneurysm or calcified meningioma [Figure 1b]. Subsequently, a digital subtraction angiography (DSA) unveiled a $3.46 \times 2.72 \times 2.47$ cm fusiform aneurysm in the right internal carotid artery (ICA) cavernous segment. The proximal ICA measured 2.37 mm, while the distal ICA was dilated (8.53 mm) [Figures 1c and d]. DSA also revealed a hypoplastic right A1 anterior cerebral artery (ACA), with bilateral A2 ACAs primarily supplied by the left A1 ACA.

In May 2023, the patient returned for further evaluation and cerebral DSA. This showed complete spontaneous thrombosis of the cavernous ICA aneurysm, occluding the distal ICA. Collateral circulation from extracranial sources, contralateral circulation, and posterior circulation ensured sufficient supply to the right cerebral hemisphere [Figures 2a-d]. A flow diverter was deferred to avoid potential complications, and the patient was discharged with follow-up recommendations and a low-dose aspirin regimen.

Despite the challenging decision not to proceed with invasive procedures such as superficial temporal artery and middle cerebral artery anastomosis on the right side, the patient remained asymptomatic at the 12-month follow-up, with stable thrombosed and calcified aneurysm [Figures 3a and b] and well-maintained blood flow in the right middle cerebral artery (MCA) and right ACA, supplemented by collateral circulation through the right posterior communicating (PCom) artery and left ACA through the anterior communicating artery.

DISCUSSION

This case report delves into a remarkable instance involving a giant cavernous segment fusiform aneurysm of the right

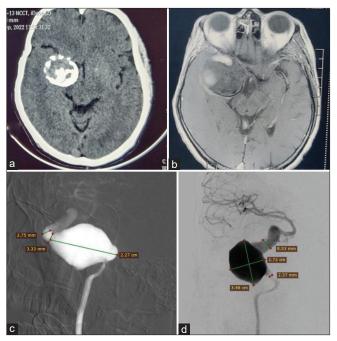


Figure 1: (a) Axial non-contrast computed tomography head demonstrates a round, heterogeneously hyperdense mass lesion in the right middle cranial fossa with calcifications in both the center and its periphery. (b) Axial gadolinium-contrast magnetic resonance imaging brain reveals a well-defined, round lesion that is iso- or even hypo-intense and is partially enhanced by intravenous gadolinium, and it is extra-axial mass along the right medial temporal convexity, closely adjacent to the right sphenoid wing. (c) Cerebral digital subtraction angiography (DSA) - Lateral view illustrates a fusiform giant aneurysm in the cavernous segment of the right internal carotid artery (ICA); the aneurysm seems to originate in the cavernous segment and likely terminates at the transition of cavernous to ophthalmic segment with measurements shown in the image. In addition, it displays the dilated and tortuous supra clinoid portion of the right ICA. (d) Cerebral DSA - Anteroposterior view shows the same fusiform giant aneurysm in the cavernous segment of the right ICA with measurements. It also highlights the dilated and tortuous supra clinoid portion of the right ICA, which is compared to the lumen size of the proximal ICA. Furthermore, it points out the hypoplastic right A1 anterior cerebral artery and normal right middle cerebral artery supply.

ICA.^[8] This aneurysm underwent spontaneous complete luminal thrombosis with obliteration of the parent vessel distal to the aneurysm.^[11,14] Importantly, the patient retained blood supply to all vascular territories of the brain, indicative of a chronic and slow occlusion process that transpired between the initial and subsequent DSAs conducted at our center.

It is important to note that giant aneurysms commonly experience partial thrombosis, with a frequency of 60%. However, complete spontaneous thrombosis is a rare occurrence and only happens in 20% of cases.^[6,8,11] The simultaneous occurrence of both giant aneurysm obliteration and vessel occlusion is exceedingly rare and reported only in



Figure 2: (a) Intraoperative right common carotid artery injection - anteroposterior view displays the proximal right internal carotid artery (ICA) and peripheral enhancement of vasa vasorum within the spontaneously thrombosed aneurysm (blue solid arrow). It also demonstrates the obliteration of the ICA distal to the aneurysm (white solid arrow) and collateral circulation provided by a branch of the right external carotid artery (red arrow) to the distal right ICA and subsequently to the right middle cerebral artery (MCA). (b) Intraoperative left ICA injection - anteroposterior view shows the filling of bilateral A2 ACA from the left ACA. Cross-compression demonstrates no cross-flow to the right A1 ACA. (c) Right vertebral artery injection, AP view, demonstrates collateral flow into the right MCA from the right posterior communicating artery. (d) Fluoroscopic image of the skull-lateral view reveals a relatively well-defined mass at the base of the skull with a clear superior margin, situated superior to the sphenoid wing. Peripheral radiopaque margins may indicate calcification. There is no evidence of sella widening, and the sphenoid sinus appears normal. ACA: Anterior cerebral artery

a handful of cases.^[1-15] As per current literature, only 20 such occurrences have been reported to date, with our case being the 21st, distinguished by its unique features.

This case posed a considerable technical challenge due to a substantial disparity between the proximal and distal segments of the right ICA. The deployment of a flow diverter could have compromised distal flow from the right PCom artery and resulted in improper placement of the device. Distal artery bypass surgery, such as the superficial temporal artery to MCA (STA-MCA), could have augmented collateral flow. However, the patient remained neurologically stable, with a thrombosed right cavernous artery and collaterals from the anterior circulation through the anterior communicating artery to bilateral A2 segments and right distal ICA supplied by the right PCom artery. This led us to opt for conservative treatment.

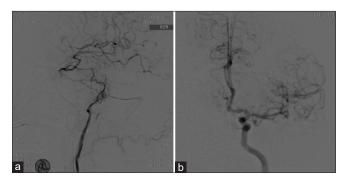


Figure 3: (a) Follow-up cerebral digital subtraction angiography – Right internal carotid artery (ICA) injection – lateral view depicts the truncated proximal ICA with minimal contrast blush within the proximal portion of the thrombosed aneurysm lumen with filling of distal ICA by right posterior communicating artery. (b) Follow-up left ICA injection – AP view shows the filling of bilateral A2 ACA from the left ACA, with no cross flow to the right A1 ACA observed during crosscompression. AP: Antero=posterior, ACA: Anterior cerebral artery

The mechanisms underlying vessel obliteration alongside spontaneous luminal thrombosis in giant aneurysms are not fully understood.^[3,9,12,13] Existing theories suggest local stretching, compression, or distortion of the ICA at this location. Factors such as tough dural folds, bony structures such as the anterior clinoid process or sellar wall, and the natural curvature of the artery in this region facilitate parent vessel deformation, stasis of blood flow, and eventual thrombosis. Notably, the onset of parent vessel occlusion close to the aneurysm, rather than at the carotid bifurcation, hints at the propagation of intra-aneurysmal thrombus into the parent artery.^[2,10,13]

Beyond thrombosis, the journey of intracranial aneurysms continues.^[1-6,10-13] Continuous growth of the aneurysm, distal propagation of thrombus, persistent mass effect, and re-canalization of thrombosed aneurysms are all potential developments as long as parent vessel flow is sustained. Previous studies have reported cases of complete re-canalization of spontaneously thrombosed aneurysms. However, it is worth noting that none of the reported cases involved thrombosed cavernous carotid artery aneurysms with ICA thrombosis.^[1-6,10-13]

While acute vessel occlusion by a thrombosed aneurysm can lead to neurological deficits and ischemic changes, especially in the presence of adequate collateral flow, our case, along with 18 others from the literature, did not exhibit clinical ischemic manifestations despite complete ICA thrombosis.^[8-14] Management of spontaneously thrombosed giant common carotid artery aneurysms and their parent vessels varies, with a dearth of literature on this topic. For asymptomatic, clinically stable patients, a conservative approach with regular follow-ups is often considered, with the hope that they will tolerate ICA occlusion over time. Among the 21 cases reported, 15,

Authors	Age/sex	Clinical features	Angiographic findings	Management	Follow-up	
					Duration	status
Mikabe <i>et al.</i> , 1980	62, F	Left CSS with diminished vision	Lt CCA-giant aneurysm Occlusion of ICA at laceral portion with minimal flow in delayed films	Surgical trapping after an abandoned transcranial exploration.	NA	Improved at discharge, barring 6th nerve palsy
Whittle <i>et al</i> ., 1982	27/M	Rt. sided CSS with proptosis	Giant Rt. CCA aneurysm with proximal occlusion	Aneurysmorrhaphy	3 months	Improved
Gautier, 1986	65/F	Rt. Hemiparesis with aphasia f/b lt CSS	Lt. large CCA aneurysm with ICA occlusion starting from 3 cm from the carotid fork, Right ICA bifurcation, and rt. MCA showed aneurysms	Conservative	1 month	Hemiparesis slightly improved; ophthalmoparesis persisted
Sato <i>et al</i> ., 1990	49/M	Lt CSS-progressed over 12 days	Giant L CCA aneurysm, initial angiogram showed occlusion of ICA beginning 3 cm from carotid fork with delayed film showing some flow in ICA.	Conservative: Complete ICA occlusion after ten days of admission, including the cervical ICA	1.5 months	Improved
	21/M	Visual loss for 5 years with recent onset Lt. hemiparesis, dysarthria	Giant R CCA aneurysm with normal ICA but elevated ipsilateral MCA – 5 years back recent angiogram showed complete ICA occlusion; adequate cross flow into ACA but not ipsilateral MCA	Right STA-MCA bypass	Not specified	Hemiparesis improved
Kurokawa <i>et al.</i> , 2001	60/F	Rt diplopia of 8 months duration	R Giant CCA (Attempted BTO failed), L CCA, Lt. MCA, Lt. ACA aneurysm	Lt MCA/ACA aneurysm clipped Rt ECA-ICA Bypass done, but carotid ligation abandoned	3.5 year	Aneurysm acute thrombosed after 3 years. The patient was improved after 3.5 years.
	50/F	Rt diplopia of Sudden onset	Giant R CCA aneurysm (Attempted BTO failed)	Conservative, readmitted after 2 years with hypopituitarism and new onset oculomotor paresis	2.5 months	Asymptomatic; oculomotor paresis resolved
Tsutsumi <i>et al.</i> , 2002	75/M	Rt CSS of sudden onset	Giant R CCA aneurysm with proximal occlusion with good cross-flow	Conservative	NA	NA
Dehdashti et al., 2003	31/F	Rt. CSS of sudden onset	Giant Rt CCA aneurysm with ICA filling	Conservative	18 months	Symptoms improved a 6 Months of Dissection and complete occlusio of ICA was noted at 18 months to check M angiogram.

(Contd...)

Table 1: (Contin	nued).					
Authors	Age/sex	Clinical features	Angiographic findings	Management		Follow-up
					Duration	status
Perrini <i>et al.</i> , 2005	47/M	6-month h/o diplopia, sudden painful ophthalmoparesis on the day of admission	Giant R CCA aneurysm with sluggish flow in ICA with arrowing at lateral portion	Conservative	6 months	Complete angiographi occlusion at 6 weeks, Pain, ophthalmoparesi better, numbness persisting at 6 months.
Vasconcellos et al., 2009	47/F	Acute onset Rt CSS 6 months back with gradual	Giant Rt CCA aneurysm with proximal ICA block	Conservative	11 years	Complete occlusion; pain resolved, ophthalmoparesis
	44/F	improvement Lt CSS of acute onset, resolved partially at 3 months	Giant Lt CCA aneurysm with proximal occlusion; MCA supplied through p-comm	Conservative	6 years	persisting Complete occlusion; symptomatically bette
	65/F	Rt CSS, Gradually worsened over 3 years	Rt CCA giant aneurysm with 70% occlusion of ICA	Conservative	7 years	Complete angiographic ICA occlusion at 5 years; symptomatical improved at 7 years.
	19/M	Lt CSS with resolution of symptoms after 10 days	Lt CCA giant aneurysm with total ICA occlusion	Conservative	A few months, not specified	asymptomatic
	84/F	Lt CSS	Lt CCA giant aneurysm ICA with occlusion	Conservative	3 months	Pain improved, partial ptosis and ophthalmoparesis persisting
Sastri <i>et al.</i> , 2013	65/F	Lt. CSS Insidious onset for 3 months	Lt CCA giant partially thrombosed aneurysm (on MRI) DSA-complete Lt ICA occlusion	Conservative on aspirin	6 months	Ophthalmoparesis improved, persistent facial pain and numbness
	55/F	GTCS, Lt CSS, left visual loss for 3 months	Lt CCA giant partially thrombosed aneurysm (on MRI) DSA-complete Lt ICA occlusion, contralateral	Conservative on aspirin	3 months	Ophthalmoparesis improved, Vision same
Das <i>et al.</i> , 2018	45/M	Sudden headache, vomiting with transient unconsciousness 1-month back	CCA small aneurysm Rt. Large CCA aneurysm (2.3*2.3 cm), Complete ICA occlusion on the right side with good collateral circulation	Conservative on aspirin	2 months	Neurologically stable
Yamagami <i>et al.</i> , 2021	68/F	Diplopia	Right large CCA aneurysm measuring 22 mm	Endovascular thrombus aspiration followed by flow diverter placement	12 months	Right oculomotor and trigeminal nerve function slight improvement
Abousedu et al., 2023	54/F	Conjunctival injection	Complete thrombosis at the cervical segment of the left ICA	Conservative on aspirin	2 months	Improvement in ophthalmoplegia, ptosis, and facial sensation

(Contd...)

Authors	Age/sex	Clinical features	Angiographic findings	Management	Follow-up	
					Duration	status
Present Case	57/M	Headache, swelling on right side of face while lying, right temporal hallowing	Rt CCA giant aneurysm 3.46*2.72*2.27 cm	Conservative on aspirin	3 months and continuing	Neurologically stable

resonance, GTCS: Generalized tonic-clonic seizure, Lf: Left, Rt: right

including ours, were successfully managed conservatively, four underwent surgical intervention, and one received endovascular treatment [Table 1]. In our case, we initially considered endovascular treatment. However, findings from our DSA indicated obliteration of the distal left ICA beyond the cervical segment, making it inaccessible due to complete thrombosis; moreover, the right distal ICA was filling from the right Pcom artery which was supporting the right MCA as right A1 was hypoplastic. DSA was suggestive of adequate cross perfusion of the right ACA territory from left ACA, right MCA territory from right PCA, and additional supply from meningeal branches of the right external carotid artery. The patient remained neurologically intact due to this robust cross-flow. The patient was started on antiplatelet drugs to prevent further thrombosis. On a follow-up of 6 months, the patient remained neurologically intact without any new-onset deficits, with repeat DSA suggesting a stable course with no further growth of aneurysm or any impairment in collateral circulation. We observed calcification of the aneurysm and remodeling of the surrounding bone.

In the majority of the series reported to date, patients improve at the 6-month mark or remain stable if not deteriorated.^[1-3,9-13] In all these studies, a slow, insidious course without neurological deficit suggests the absence of a thromboembolic phenomenon, which may have a better course and prognosis compared to patients with a thromboembolic phenomenon, suggesting hemodynamically unstable aneurysms with an unpredictable course.^[3,6,9,13,15]

Through this case report, we aim to provide insights and valuable information regarding this rare phenomenon. The presence of unique features, such as cross-flow, calcification, and petrous bone erosion with intracranial and extracranial collaterals to supplement distal ICA flow in one of the largest giant calcified aneurysms reported to date, suggests a chronic nature of the disease not previously documented. We advocate for conservative management in cases with stable hemodynamics, as supported by existing literature, and emphasize the importance of close follow-up for the early detection of disease progression.^[1,4,6,9-15]

CONCLUSION

This case report contributes valuable insights into a rare phenomenon, offering a comprehensive understanding of the intricate interplay between thrombosed giant aneurysms and the vessels they affect. The presence of unique features, such as cross-flow, calcification, and petrous bone erosion, adds further depth to our understanding of this complex condition. We advocate for conservative management in cases with stable hemodynamics, supported by existing literature, and emphasize the need for vigilant follow-up to detect any signs of disease progression. This case adds to the myriad presentations of this entity and opens doors for further research and exploration of the intricate dynamics involved in thrombosed aneurysms.

Ethical approval

Institutional review board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Use of Artificial Intelligence (AI)-Assisted Technology for manuscript preparation

The authors confirm that there was no use of Artificial Intelligence (AI)-Assisted Technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

- 1. Abousedu YA, Saleem A, Alenezi S, Bosnjakovic P, Lazovic L, Alsheikh TM. Spontaneous thrombosis of a giant cavernouscarotid aneurysm with simultaneous ipsilateral complete parent artery occlusion: A rare phenomenon and review of the literature. Arch Clin Cases 2023;10:21-8.
- Das KK, Singh G, Pandey S, Bhaisora KS, Jaiswal A, Behari S. Completely thrombosed giant intracranial aneurysm with spontaneous thrombosis of the parent artery: Is it nature's divine intervention and a self-cure? World Neurosurg 2018;118:132-8.
- 3. Dehdashti AR, de Tribolet N. Giant intracavernous aneurysm thrombosis by spontaneous carotid occlusion. Cerebrovasc Dis 2003;15:301-2.
- 4. Gautier JC, Awada A, Majdalani A. Ophthalmoplegia with contralateral hemiplegia. Occlusion of the internal carotid artery due to thrombosis of an intracavernous aneurysm. Stroke 1986;17:1321-2.
- Hahn CD, Nicolle DA, Lownie SP, Drake CG. Giant cavernous carotid aneurysms: Clinical presentation in fifty-seven cases. J Neuroophthalmol 2000;20:253-8.
- Kurokawa R, Kuroshima Y, Yoshida K, Kawase T. Spontaneous thrombosis of intracavernous internal carotid artery aneurysm and parent artery occlusion in patients with positive balloon test occlusion: Two case reports. Neurol Med Chir (Tokyo) 2001;41:436-41.
- 7. Menon S, Menon RG. Cavernous carotid aneurysms: To do or not to do? J Neurosci Rural Pract 2017;8:284-7.
- Mikabe T, Ogihara R, Tomita S, Kin H, Karasawa H, Watanabe S, *et al.* Giant intracranial aneurysm visualized by prolonged injection angiography - case report (author's transl) No Shinkei Geka 1980;8:749-53.

- 9. Perrini P, Bortolotti C, Wang H, Fraser K, Lanzino G. Thrombosed giant intracavernous aneurysm with subsequent spontaneous ipsilateral carotid artery occlusion. Acta Neurochir (Wien) 2005;147:215-7.
- Sastri SB, Sadasiva N, Pandey P. Giant cavernous carotid aneurysm with spontaneous ipsilateral ICA occlusion: Report of 2 cases and review of literature. J Neurosci Rural Pract 2013;4(Suppl 1):S113-6.
- 11. Sato K, Fujiwara S, Yoshimoto T, Onuma T. Two cases of spontaneous internal carotid artery occlusion due to giant intracranial carotid artery aneurysm. Stroke 1990;21:1506-9.
- 12. Tsutsumi M, Kazekawa K, Tanaka A, Ueno Y, Nomoto Y. Spontaneous thrombosis of a giant intracavernous internal carotid artery aneurysm and ipsilateral internal carotid artery occlusion. Radiat Med 2002;20:261-3.
- Vasconcellos LP, Flores JA, Conti ML, Veiga JC, Lancellotti CL. Spontaneous thrombosis of internal carotid artery: A natural history of giant carotid cavernous aneurysms. Arq Neuropsiquiatr 2009;67:278-83.
- 14. Whittle IR, Williams DB, Halmagyi GM, Besser M. Spontaneous thrombosis of a giant intracranial aneurysm and ipsilateral internal carotid artery. Case report. J Neurosurg 1982;56:287-9.
- 15. Yamagami K, Hatano T, Ando M, Chihara H, Ogura T, Suzuki K, *et al.* Symptomatic cavernous internal carotid artery aneurysm complicated by simultaneous rapid growth of the intra-aneurysmal and parent artery thromboses. NMC Case Rep J 2021;8:177-82.

How to cite this article: Jha VC, Jain R, Sinha VS, Kumar N, Verma G, Dhage N. Spontaneous thrombosis and calcification of giant cavernous carotid artery aneurysm: A rare case and management insights. Surg Neurol Int. 2024;15:98. doi: 10.25259/SNI_805_2023

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