Int Clin Neurosci J. 2022;9:e24 Case Report

doi:10.34172/icnj.2022.24

ICNSJ International Clinical Neuroscience Journal

Management of Traumatic Pseudoaneurysm of the Supraclinoid Internal Carotid Artery using Coil Embolization: A Case Report

Rohit Yadav[1*], S I Sadique[1], Shubhamitra Chaudhari[1]

[1]Department of Neurosurgery, IPGMER & SSKM Hospital, Kolkata, West Bengal, India

Abstract

Traumatic aneurysm of the internal carotid artery (ICA) occurs rarely, with an approximate incidence of 0.15% and 0.40% of total intracranial aneurysms. An interesting case of delayed presentation of pseudoaneurysm of the left ICA in a 61-year-old patient is reported here, who came to us for evaluation of blindness, proptosis, and ophthalmoparesis. This potentially life-threatening condition was successfully managed using coil embolization after complete evaluation and investigations.

Keywords: Head injury; Internal carotid artery; Proptosis; Traumatic pseudoaneurysm.

***Correspondence to**

Rohit Yadav,
Resident, Department of Neurosurgery, IPGMER & SSKM Hospital, Kolkata, West Bengal, India.
Tel: +919921122348,
Email: rohityadav1992@hotmail.com

Published online August 29, 2022

Citation: Yadav R, Sadique SI, Chaudhari S. Management of traumatic pseudoaneurysm of the supraclinoid internal carotid artery using coil embolization: A case report. Clin Neurosci J. 2022;9:e24. doi:10.34172/icnj.2022.24.

Introduction

Traumatic aneurysm of the internal carotid artery (ICA) occurs rarely, with an approximate incidence of 0.15% and 0.40% of total intracranial aneurysms.[1,2] Injury to the ophthalmic artery following head injury may eventually lead to pseudoaneurysm formation. Catastrophic complications may occur because of spontaneous rupture or compression of adjacent vital neural structures because of attainment of massive size by the aneurysm. Compression of optic or oculomotor nerves can lead to ophthalmoplegia and loss of vision.[3] *Unilateral proptosis* or exophthalmos can be uncommonly caused by an aneurysm of the intracranial part of ICA.[4]

A case of delayed presentation of pseudoaneurysm of left ICA in a 61-year-old patient is reported here, who came to us for evaluation of blindness, proptosis, and ophthalmoparesis. This is an interesting case of traumatic pseudoaneurysm of supraclinoid ICA (TPICA) presented to the neurosurgery department.

Case Report

A 61-year-old man presented with a complaint of a progressive decrease in vision of the left eye for 6 years and protrusion of the left eyeball for 9 months and complete vision loss for 3 months. There was a history of head injury in an accident 20 years ago. He had sustained head trauma with a major impact over the left frontotemporal region. The patient did not visit any local hospital then but in the course of time he developed visual problems consulted an ophthalmologist. On examination, right eye visual acuity was 6/9 and left eye perception of light was negative. Extraocular movements were restricted to the left eye and present in the right eye. External examination showed there was proptosis in the left eye with opacity and degeneration of cornea (Figure 1). CT scan of the brain and left orbit was done which did not show any abnormality. Magnetic resonance imaging (MRI) orbit plain and contrast with magnetic resonance (MR) brain angiography showed a large, relatively well-defined, heterogeneously hyperintense lesion arising from extraconal compartment along the superior aspect of left orbit with imaging features of mass effect similar to that of hematoma with a differential diagnosis such as orbital cavernous malformation with intralesional haemorrhages and angiography within normal limits except shifting of anterior cerebral artery (ACA) s to right and left middle cerebral artery (MCA) posteriorly. The diagnosis of apseudoaneurysm arising from the ophthalmic segment of the supraglenoid artery branch of the left ICA was confirmed using digital subtraction angiography (DSA) (Figures 2 and 3).

The patient was then taken for DSA and coil embolization

Figure 1. Proptosis in the Left Eye.

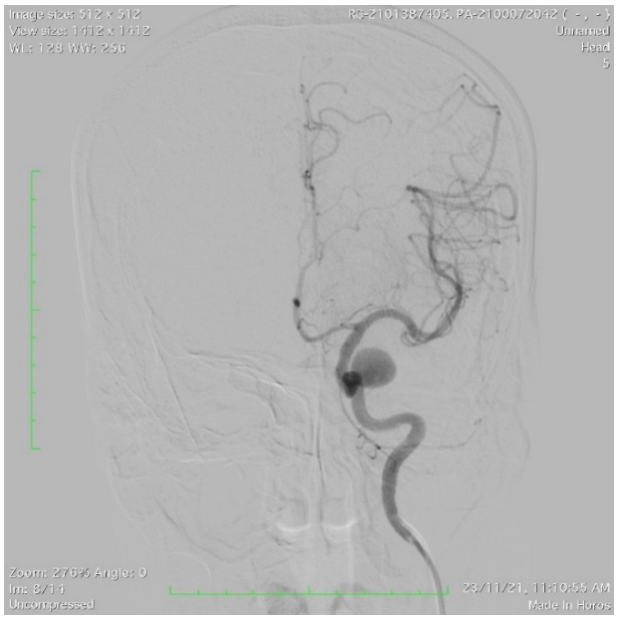

Figure 2. Ophthalmic Branch of the Supraclinoid Segment of the left Internal Carotid Artery.

of pseudoaneurysm and near-complete occlusion of left ICA, pseudoaneurysm was done (Figure 4). Post DSA there was no complication present and a repeat DSA was done after 7 days to look for good occlusion followed by enucleation of the eye (Figure 5).

Discussion

The diagnosis and management of traumatic aneurysm mostly presents a distinctive challenge to surgeons. Depending upon the extent and severity of the primary damage to the arterial wall, histopathologically, traumatic aneurysms can be true, false, mixed, or of dissecting type.

True aneurysms involve disruption of the intima and variable involvement of the internal elastic layer and media, resulting in localised vessel wall weakening and aneurysm formation. False aneurysms occur when all three layers of the vessel wall are disrupted, resulting in the formation of a contained hematoma outside the vessel. This is most similar to the histological type associated with penetrating injuries. Mixed aneurysm is the third histological type, and begins as a true aneurysm and then ruptures, forming hematoma and a false lumen. The anatomic location of the injured vessel and surrounding hard structures is closely related to the development of aneurysm.[5,6] Usually ICA injuries is the result of injury from the clinoid process, pericallosal artery injuries from falx cerebri, and middle cerebral artery injuries from the sphenoid ridge.[7] In our case the patient sustained blunt injury over left frontotemporal region.

Ophthalmic artery is a complex vessel which originates from ICA and runs through a dural sheath on the optic

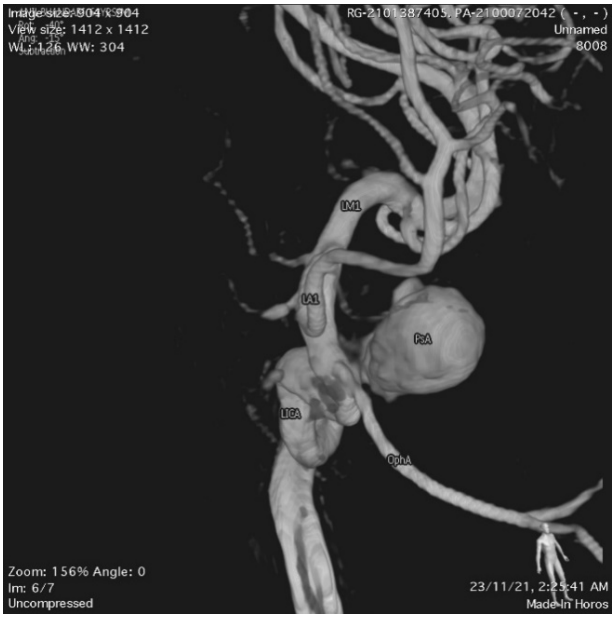

Figure 3. Preoperative 3D DSA.

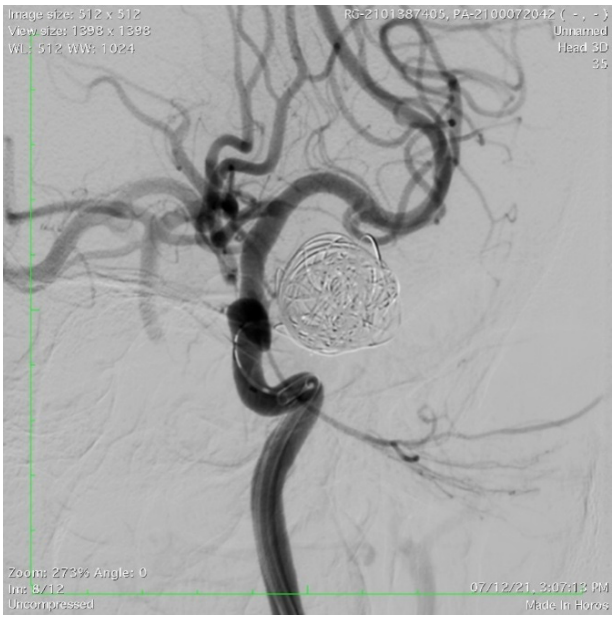

Figure 4. Post-coiling Digital Subtraction Angiography of Pseudoaneurysm.

Figure 5. Enucleation of Eye.

canal floor. The mechanism of traumatic ophthalmic artery aneurysm is not clearly defined but it is proposed that blunt high-speed trauma at the dural penetrating point is the main dynamic force causing focal dilatation or tear of the artery wall. Blood clot formation at injury site provides a seal-off and leads to the formation of a false wall. Usually, it takes 2 to 3 weeks for an aneurysm to rupture after its formation following trauma. Traumatic cerebral aneurysms have high mortality and morbidity. Early diagnosis and timely intervention are mandatory for such lesions.[8,9]

Pathophysiology of Pseudoaneurysm

Intracranial arteries are more vulnerable to the formation of pseudoaneurysms of saccular and fusiform types as they are stiff with thin media and possess a thicker internal elastic lamina.[10] In pseudoaneurysms, haemorrhage from a small, full-thickness arterial rent is adequately contained by the buttressing effect of local brain tissue. An inflammatory reaction occurs in surrounding tissue leading to the development of an epithelial lining and fibrous capsular wall. The apparent lumen of the aneurysm gets contained by an organized extraluminal hematoma which liquefies in approximately one week. There are high chances of rebleeding due to enlargement and weakening of fibrous wall caused by continuous pulsatile forces.[11]

Clinical Course

ICA is generally very unstable and carries a high mortality and may rupture at any minute or can stay silent for years. The usual clinical course is generally delayed. Proptosis and gradual vision loss can occur and sometimes massive bleeding can cause hemodynamic instability and death.[12]

Evaluation

High risk patients with history of penetrating injury over orbitofrontal or craniofacial region or crossing the midline and of blunt trauma with late neurological deterioration, cranial nerve palsy, proptosis and amaurosis should be investigated.[13,14]

Management

The rapid and effective treatment option for the long-term resolution of these lesion is an endovascular occlusion. A neurosurgical procedure such as craniotomy with aneurysm clipping, or permanent ligation of vessels can also be done.[15,16] Pseudoaneurysm was diagnosed 20 years after head injury in this case. Usually, no perception of light in vision 20 years after traumatic neuropathy requires no further evaluation and is generally accepted due to traumatic neuropathy or demyelination of the optic nerve. History of progressive decrease in the visual acuity, proptosis, and ophthalmoplegia require neuroimaging. The presence of pseudoaneurysm of the ICA was revealed in the imaging. Early identification of signs and symptoms of this rare condition can help precluding the adverse complications of head injury.

Conflict of Interest Disclosures

The authors declare that they have no conflict of interests.

Ethical Statement

Written informed consent was obtained from the patient for publication this report.

Funding

The authors declare no financial interest or sources of support.

References

1. Menon G, Hegde A, Nair R. Post-traumatic cavernous carotid pseudoaneurysm with delayed epistaxis. Cureus. 2018;10(7):e3002. doi: 10.7759/cureus.3002.
2. Tunthanathip T, Phuenpathom N, Saehaeng S, Oearsakul T, Sakarunchai I, Kaewborisutsakul A. Traumatic cerebrovascular injury: prevalence and risk factors. Am J Emerg Med. 2020;38(2):182-6. doi: 10.1016/j.ajem.2019.01.055.
3. Ghorbani M, Griessenauer CJ, Wipplinger C, Abdolhoseinpour H, Bahrami R, Asaadi S, et al. Combined Endovascular and Endoscopic Approach for Treatment of Concomitant Sphenoid Sinus Giant Traumatic Aneurysm and Direct Carotid Cavernous Fistulas. World Neurosurg. 2020;134:211-4. doi: 10.1016/j.wneu.2019.10.131.
4. Choi BK, Lee TH, Choi CH, Lee SW. Fusiform intracanalicular ophthalmic artery aneurysm; case report and review of literature. J Korean Neurosurg Soc. 2008;44(1):43-6. doi: 10.3340/jkns.2008.44.1.43.
5. Pandey P, Rayes M, Guthikonda M, Xavier A. Peripheral ophthalmic artery aneurysm associated with multiple intracranial aneurysms: a case report. J Neurointerv Surg. 2010;2(3):211-2. doi: 10.1136/jnis.2009.001107.
6. Li Y, Song WX, Zhang TM, Fu JD, Sun S, Zhao JZ. Intraorbital traumatic ophthalmic artery aneurysm: case report. Neurol India. 2012;60(6):657-60. doi: 10.4103/0028-3886.105213.
7. Dubey A, Sung WS, Chen YY, Amato D, Mujic A, Waites P, et al. Traumatic intracranial aneurysm: a brief review. J Clin Neurosci. 2008;15(6):609-12. doi: 10.1016/j.jocn.2007.11.006.
8. Bhaisora KS, Behari S, Godbole C, Phadke RV. Traumatic aneurysms of the intracranial and cervical vessels: a review. Neurol India. 2016;64 Suppl:S14-23. doi: 10.4103/0028-3886.178032.
9. Mao Z, Wang N, Hussain M, Li M, Zhang H, Zhang Q, et al. Traumatic intracranial aneurysms due to blunt brain

injury-a single center experience. Acta Neurochir (Wien). 2012;154(12):2187-93. doi: 10.1007/s00701-012-1487-x.
10. Ishigami D, Ota T. Traumatic pseudoaneurysm of the distal anterior cerebral artery following penetrating brain injury caused by a crossbow bolt: a case report. NMC Case Rep J. 2018;5(1):21-6. doi: 10.2176/nmccrj.cr.2017-0083.
11. Benoit BG, Wortzman G. Traumatic cerebral aneurysms. Clinical features and natural history. J Neurol Neurosurg Psychiatry. 1973;36(1):127-38. doi: 10.1136/jnnp.36.1.127.
12. Shim YS. Coil embolization of traumatic ophthalmic artery aneurysm: case report. Korean J Neurotrauma. 2022;18(1):98-102. doi: 10.13004/kjnt.2022.18.e2.
13. Al-Jehani HM, Alwadaani HA, Almolani FM. Traumatic intracranial internal carotid artery pseudoaneurysm presenting as epistaxis treated by endovascular coiling. Neurosciences (Riyadh). 2016;21(1):60-3. doi: 10.17712/nsj.2016.1.20150514.
14. Zheng Y, Lu Z, Shen J, Xu F. Intracranial pseudoaneurysms: evaluation and management. Front Neurol. 2020;11:582. doi: 10.3389/fneur.2020.00582.
15. Bassi ST, Noronha V, Halbe S. A time bomb defused, in time! A traumatic optic neuropathy to be wary of. Neuroophthalmology. 2019;43(1):32-5. doi: 10.1080/01658107.2018.1458886.
16. Kankane VK, Warade AG, Misra BK. Extracranial-intracranial high-flow bypass for post-traumatic cavernous carotid pseudoaneurysm presenting with epistaxis: case report. Neurol India. 2019;67(2):485-90. doi: 10.4103/0028-3886.257994.