www.jcimcr.org

JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 6

Partially thrombosed internal carotid artery: A diagnostic dilemma

***Corresponding Author: Avani Kanojia**

Department of Radiodiagnosis, Era's Lucknow Medical College and Hospital, Lucknow, India.

Email: avanikanojia1997@gmail.com

Received: Mar 23, 2025

Accepted: Apr 09, 2025

Published: Apr 16, 2025

Archived: www.jcimcr.org

DOI: www.doi.org/10.52768/2766-7820/3556

Abstract

Acute or chronic thrombosis of the Internal Carotid Artery (ICA) is a rare but life-threatening condition that can result in ischemic stroke, Transient Ischemic Attacks (TIAs), or significant neurological deficits. The ICA is a major blood vessel supplying oxygenated blood to the brain, and its occlusion can lead to devastating consequences, including cerebral infarction, hemodynamic insufficiency, and embolic complications. Acute thrombosis occurs suddenly, often due to embolism, arterial dissection, or hypercoagulable states, leading to abrupt-onset neurological symptoms such as hemiparesis, hemianesthesia, aphasia, or visual disturbances. It requires urgent intervention, including thrombolysis or mechanical thrombectomy. Chronic thrombosis develops over time, typically secondary to atherosclerosis or vessel narrowing, allowing collateral circulation to compensate. Patients may remain asymptomatic or experience gradual cognitive decline, TIAs, or progressive stroke-like symptoms.

Keywords: Internal carotid artery; Thrombosis; Aneurysm; Neurological deficit; Compression.

Introduction

Partially thrombosed aneurysms are a diverse group of complex aneurysms characterized by an organized intraluminal thrombus and a solid mass. While their size can range from small to giant, they are most commonly found in the large to giant category. In the anterior circulation, these aneurysms often present with symptoms related to mass effect on cranial nerves. The specific neurological symptoms depend on the aneurysm's size and location. Compression of the oculomotor nerve (CN III), trochlear nerve (CN IV), or abducens nerve (CN VI) can lead to ophthalmospasms or ophthalmoplegia, frequently seen with aneurysms of the cavernous sinus and posterior communicating artery (Coma). Additionally, compression of the optic nerve (CN II) may result in decreased visual acuity and visual field deficits, which are most commonly associated with carotid ophthalmic and superior hypophyseal aneurysms. Giant aneurysms of the anterior communicating artery (AcomA) can lead to a frontal syndrome, while Middle Cerebral Artery (MCA) aneurysms may result in dysphasia. In the posterior circulation, giant aneurysms have the potential to compress the brainstem. Large basilar tip and superior cerebellar artery (SCA) aneurysms are often associated with cranial nerve III (CN III) palsy, whereas Posterior Inferior Cerebellar Artery (PICA) aneurysms may compress the trigeminal or facial nerve, leading to corresponding neurological deficits [1-3]. The presence of a thrombus can significantly influence both the risk associated with an aneurysm and its progression. Studies on abdominal aortic aneurysms have already demonstrated that a considerable clot burden correlates with increased aneurysm growth rates [4]. Magnetic Resonance (MR) imaging techniques have the ability to visualize thrombi in detail, allowing for the assessment of their structure and composition. Additionally, these techniques can provide valuable insights into the age of the thrombus, which may aid in understanding its stability and potential clinical implications [5].

Case report

A 40-year-old female, previously healthy, presented to the emergency department with a sudden-onset neurological

deficit that began three hours prior to arrival. The symptoms included Right-sided hemiparesis (weakness in the right upper and lower limbs), with a Medical Research Council (MRC) grade of 2/5 in both extremities, Facial droop affecting the right side, suggesting Lower Motor Neuron (LMN) type facial palsy and Dysarthria, with slurred speech but no significant language impairment. She had no history of hypertension, diabetes, dyslipidemia, smoking, alcohol consumption, recent infections, or previous cerebrovascular events. There was no history of trauma, fever, headache, neck pain, visual disturbances, seizures, or loss of consciousness. Her family history was unremarkable for cardiovascular or cerebrovascular diseases, and she was not on any oral contraceptives or anticoagulant therapy. She denied any recent long-distance travel or prolonged immobility that could predispose her to a hypercoagulable state. On general examination, her blood pressure was 124/80 mmHg, heart rate was 76 bpm, and there were no signs of carotid bruit, limb asymmetry, or systemic illness. A focused neurological examination revealed - Glasgow Coma Scale (GCS): 15/15 (fully conscious and oriented), Cranial nerve examination: Right-sided facial weakness (House-Brackmann Grade III), Motor system: Right upper and lower limb power: 2/5 (severe weakness), Muscle tone: Mildly reduced on the right side, Reflexes: Brisk (hyperreflexia) on the right side with a positive Babinski sign (suggesting an upper motor neuron lesion). Sensory examination revealed no significant loss of touch, pain, or vibration sense. She was unable to walk independently due to weakness. Given her sudden-onset focal neurological symptoms, an acute ischemic stroke due to large-vessel occlusion was suspected, prompting urgent neuroimaging. Magnetic Resonance Imaging (MRI) revealed a giant saccular aneurysmal dilatation involving the intra-cavernous segment of the right Internal Carotid Artery (ICA). The aneurysm measures approximately 25.3 x 23.5 x 23.4 mm and demonstrates distinct signal characteristics indicative of both a patent lumen and a partially thrombosed sac. The peripheral rim of the aneurysm exhibits altered signal intensity, appearing hyperintense on T1-weighted and FLAIR sequences while being hypointense on T2-weighted imaging. Angiographic studies show contrast enhancement along the rim, confirming the presence of a patent lumen. The central portion of the aneurysmal sac demonstrates heterogeneous signal characteristics, with a heterogeneously hyperintense appearance on T1-weighted imaging, suggesting the presence of subacute blood products. On T2-weighted sequences, it appears hypointense, likely due to hemosiderin or chronic thrombus deposition. FLAIR imaging reveals a heterogeneously hypointense signal, indicating mixed blood components. Additionally, Susceptibility-Weighted Imaging (SWI) demonstrates blooming artifacts, further confirming the presence of thrombus within the aneurysmal sac. The aneurysmal dilation exerts a significant mass effect, resulting in the supero-lateral displacement of the adjacent right temporal lobe, which may contribute to focal neurological symptoms. It also compresses cranial nerves traversing the cavernous sinus, including the ophthalmic (V1) and maxillary (V2) divisions of the trigeminal nerve (CN V), potentially causing facial pain, paresthesia, or sensory deficits. Additionally, involvement of the oculomotor nerve (CN III) and trochlear nerve (CN IV) may lead to ophthalmoplegia, ptosis, or diplopia. The right Middle Cerebral Artery (MCA) is also affected, raising concerns about potential ischemic effects or vascular compromise. Furthermore, the aneurysm exhibits supero-medial abutment of the optic chiasma, which could result in visual field defects, particularly bitemporal hemianopia if significant compression occurs. The findings are consistent with a giant partially thrombosed saccular aneurysm of the intra-cavernous segment of the right ICA, exhibiting a patent lumen with peripheral enhancement and central thrombus formation. The aneurysm is exerting mass effect on adjacent neurovascular structures, with potential clinical implications including cranial nerve dysfunction, cerebrovascular compromise, and compressive optic neuropathy.

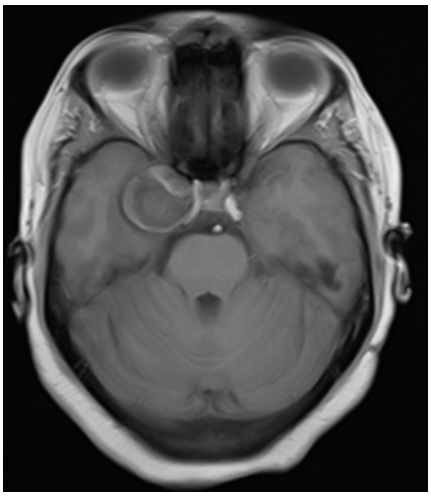

Figure 1: T1- Weighted axial MRI image showing a saccular aneurysmal dilatation of the right internal carotid artery having a peripheral hyperintense rim (blood) with hypointense center.

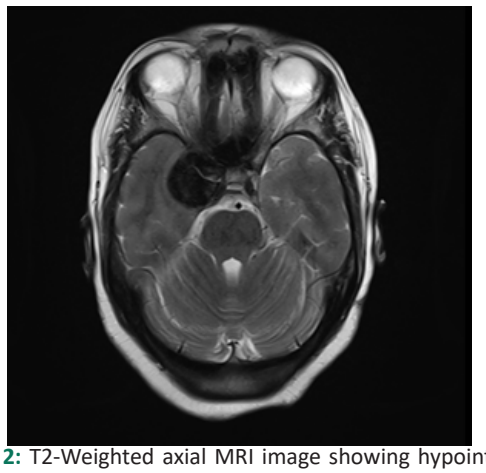

Figure 2: T2-Weighted axial MRI image showing hypointensity in the aneurysmal dilatation.

Discussion

A giant thrombus within the Internal Carotid Artery (ICA) is an uncommon but serious vascular pathology with potentially devastating neurological consequences. It poses a significant risk of ischemic stroke due to arterial occlusion or distal embolization, which can lead to cerebral infarction and long-term disability. Additionally, the presence of a large thrombus can exert a mass effect on adjacent neurovascular structures, leading to cranial nerve compression, vascular compromise, and altered cerebrovascular hemodynamics. This condition may arise due to underlying atherosclerosis, aneurysmal dilation, arterial dissection, vasculitis, or hypercoagulable states, all of which contribute to thrombus formation. Clinically, patients may present with neurological deficits ranging from Transient Ischemic

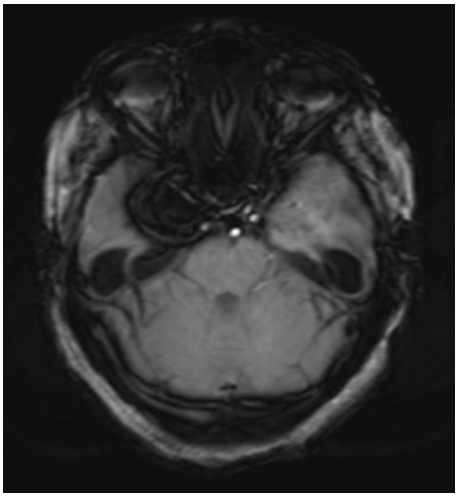

Figure 3: Susceptibility-Weighted Imaging (SWI) MRI - The image demonstrates a large thrombus within the intra-cavernous segment of the right ICA, appearing as a markedly hypointense (black) area with blooming artifact on SWI, consistent with hemosiderin deposition and chronic thrombus formation.

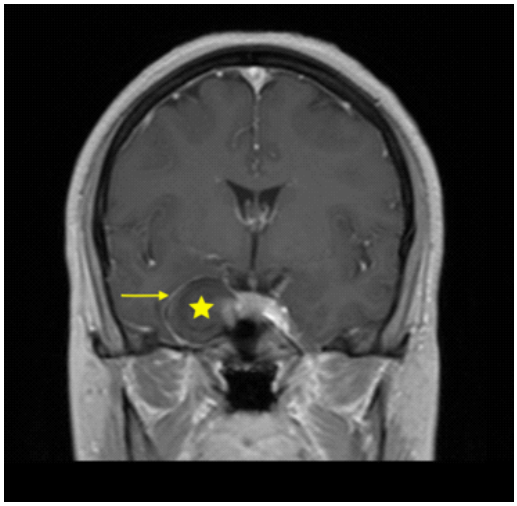

Figure 4: Post-Contrast MR Angiography (MRA) of a Giant Internal Carotid Artery (ICA) Thrombus - The image reveals a giant saccular aneurysmal dilation in the intra-cavernous segment of the right ICA, with a central non-enhancing thrombus (yellow star) surrounded by a rim of contrast-enhancing patent lumen (yellow arrow).

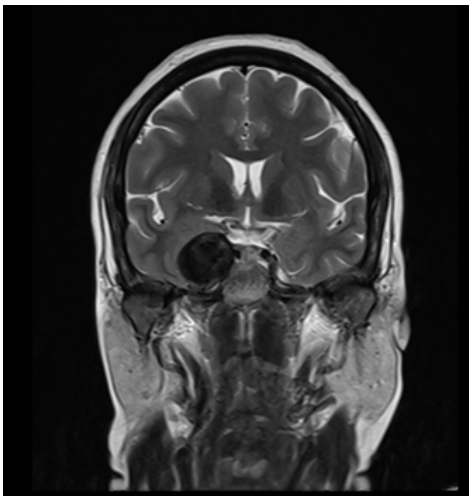

Figure 5: T2-Weighted coronal MRI of a giant Internal Carotid Artery (ICA) thrombus.

Attacks (TIAs) to acute ischemic stroke, as well as symptoms of cranial nerve dysfunction if the thrombus extends into the cavernous sinus. Advanced neuroimaging techniques such as MRI, MRA, CT Angiography (CTA), and Digital Subtraction Angiography (DSA) are crucial for early detection, accurate localization, and characterization of the thrombus. Early diagnosis allows for prompt medical, endovascular, or surgical intervention, reducing the risk of infarction and neurological deterioration. This case emphasizes the critical role of timely recognition and appropriate management strategies in preventing catastrophic neurological outcomes.

References

1. Mansour N, Kamel MH, Kelleher M, Aquilina K, Thornton J, et al. Resolution of cranial nerve paresis after endovascular management of cerebral aneurysms. Surgical neurology. 2007; 68(5): 500-4.
2. Kazekawa K, Tsutsumi M, Aikawa H, Iko M, Kodama T, et al. Internal carotid aneurysms presenting with mass effect symptoms of cranial nerve dysfunction: efficacy and imitations of endosaccular embolization with GDC. Radiation medicine. 2003; 21(2): 80-5.
3. Stiebel-Kalish H, Maimon S, Amsalem J, Erlich R, Kalish Y, et al. Evolution of oculomotor nerve paresis after endovascular coiling of posterior communicating artery aneurysms: a neuro-ophthalmological perspective. Neurosurgery. 2003; 53(6): 1268-74.
4. Speelman L, Schurink GW, Bosboom EM, Buth J, Breeuwer M, et al. The mechanical role of thrombus on the growth rate of an abdominal aortic aneurysm. Journal of vascular surgery. 2010; 51(1): 19-26.
5. Corti R, Osende JI, Fayad ZA, Fallon JT, Fuster V, et al. In vivo noninvasive detection and age definition of arterial thrombus by MRI. Journal of the American College of Cardiology. 2002; 39(8): 1366-73.