Case Report

Partially thrombosed internal carotid artery: A diagnostic dilemma

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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.

Key words: Aneurysm, Internal carotid artery, Thrombosis

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 the 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 ophthalmoparesis or ophthalmoplegia, frequently seen with aneurysms of the cavernous sinus and posterior communicating artery. In addition, 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 can lead to a frontal syndrome, while middle cerebral artery 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 aneurysms are often associated with cranial nerve III (CN III) palsy, whereas posterior inferior cerebellar artery 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 imaging (MRI) techniques have the ability to visualize thrombi in detail, allowing for the assessment of their structure and composition. In addition, these techniques can provide valuable insights into the age of the thrombus, which may aid in understanding its stability and potential clinical implications [5].

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CASE REPORT

A 40-year-old female, previously healthy, presented to the emergency department with a sudden-onset neurological deficit that began 3 h before arrival. The symptoms included right-sided hemiparesis (weakness in the right upper and lower limbs), with a Medical Research Council grade of 2/5 in both extremities, facial droop affecting the right side, suggesting lower motor neuron 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

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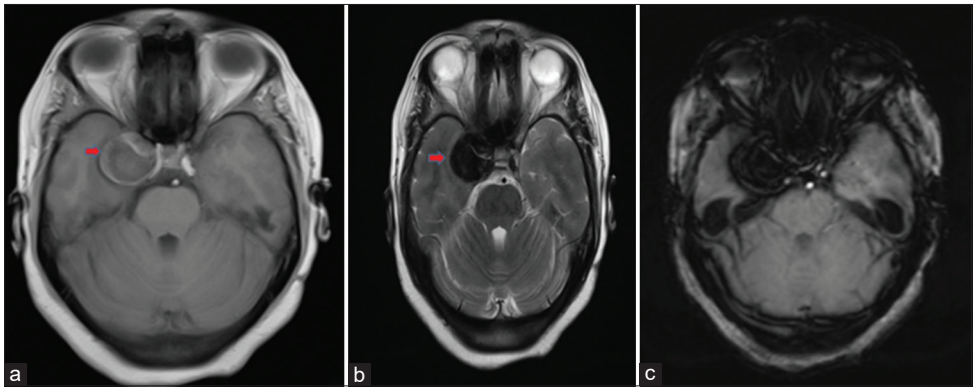

Figure 1: (a) T1-weighted axial magnetic resonance imaging (MRI) image showing a saccular aneurysmal dilatation of the right ICA having a peripheral hyperintense rim (blood) with hypointense center; (b) T2-weighted axial MRI image showing hypointensity in the aneurysmal dilatation; (c) Susceptibility-weighted 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

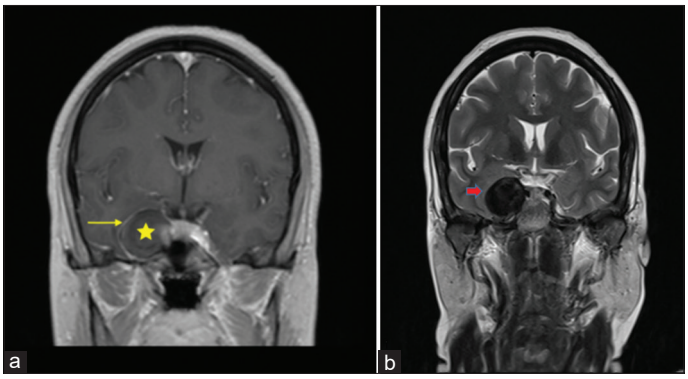

Figure 2: (a) Post-contrast magnetic resonance angiography of a giant internal carotid artery (ICA) thrombus - The image reveals a giant saccular aneurysmal dilation in the intra-cavernous segment of the right ICA, with a central non-enhancing thrombus (yellow star) surrounded by a rim of contrast-enhancing patent lumen (yellow arrow); (b) T2-weighted coronal magnetic resonance imaging of a giant ICA thrombus

immobility that could predispose her to a hypercoagulable state. 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.

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 × 23.5 × 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 fluid-attenuated inversion recovery (FLAIR) sequences while being hypointense on T2-weighted imaging (Figs. 1 and 2). 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. In addition, susceptibility-weighted imaging demonstrates blooming artifacts, further confirming the presence of a thrombus within the aneurysmal sac. The aneurysmal dilation exerts a significant mass effect, resulting in the superolateral 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. In addition, involvement of the oculomotor nerve (CN III) and trochlear nerve (CN IV) may lead to ophthalmoplegia, ptosis, or diplopia. The right middle cerebral artery is also affected, raising concerns about potential ischemic effects or vascular compromise. Furthermore, the aneurysm exhibits superomedial 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 exerts a mass effect on adjacent neurovascular structures, with potential clinical implications including cranial nerve dysfunction, cerebrovascular compromise, and compressive optic neuropathy.

DISCUSSION

A giant thrombus within the 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. In addition, 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.

Early restoration of blood flow in an obstructed vessel offers a promising approach for the active treatment of acute ischemic

stroke [6]. Thrombolytic therapy in intracranial vascular occlusion aims to preserve at-risk brain tissue rather than restore damaged areas. Leptomeningeal collaterals can prevent infarction after major artery blockage, but progressive brain swelling may cause collateral failure, enlarging the infarct. Early recanalization helps counter this, so early infarct signs on computed tomography (CT) are not a contraindication to thrombolysis. Increased radiolucency of the gray matter indicates irreversibly damaged brain tissue, becoming detectable within the first 2 h of symptom onset [7].

Recanalizing therapy may be beneficial if the parenchymal hypodensity affects only a portion of the at-risk tissue volume [8].

CONCLUSION

Advanced neuroimaging techniques such as MRI, magnetic resonance angiography, CT angiography, and digital subtraction angiography 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.

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