SHORT REPORT

Extracranial Internal Carotid Artery Mycotic Aneurysm: A Case Report

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Abstract Mycotic aneurysms of the extracranial carotid arteries (MCAs) are extremely rare. They usually appear as an enlarging pulsatile neck mass with no specific signs and symptoms, and they can lead to severe morbidity and mortality if left untreated. We report a case of a saccular thrombosed MCA in a 68-year-old man, presented as a non-pulsatile enlarging mass. The patient did not have any clinical signs of infection, and he was treated with resection of the MCA and synthetic patch reconstruction of the carotid bifurcation. Postoperative microbial cultures revealed Streptococcus parasanguinis. We review and discuss the literature regarding the clinical presentation, diagnosis and treatment options of MCAs.
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Mycotic aneurysms of carotid arteries (MCAs) today are extremely rare, they usually appear as an enlarging neck mass and often are difficult to diagnose. They have non-specific signs and symptoms, an unclear aetiology and can lead to severe morbidity and mortality if left untreated.

We report an MCA case resulting from Streptococcus parasanguinis, treated with resection of the aneurysm and reconstruction of carotid bifurcation with a synthetic patch.

Case report

A 68-year-old man was admitted to our hospital with an expanding mass on the left side of the neck. He had no history of trauma, neck infection or endocarditis. Physical examination revealed a tense, hard and non-pulsatile mass, measuring about 5 × 5 cm, on the left side of the neck. Auscultation failed to reveal a bruit. Magnetic resonance (MR) imaging showed a mass close to the carotid bifurcation. MR angiography revealed occlusion of the right internal carotid artery and tight ulcerated stenosis of the left internal carotid artery (Fig. 1).

The patient was scheduled for an elective operation. The operative plan included exploration and resection of the mass by a laryngologist, followed by conventional...
endarterectomy of the left carotid bifurcation by a vascular surgeon. Complete dissection of the mass revealed that it was a 5 × 5 thrombosed saccular aneurysm which involved the carotid bifurcation. The systolic internal carotid back-pressure was 60 mmHg. Arteriotomy of the carotid bifurcation revealed the absence of atherosclerotic stenosis and that the possible plaque ulceration was in fact the neck of a thrombosed saccular aneurysm. The aneurysm was resected and the left lateral wall of the common and internal carotid arteries was debrided to obtain a normal tissue. A polytetrafluoroethylene (PTFE) patch measuring 25 mm in length was used to restore the flow to the carotid bifurcation arteries. No intraluminal shunt was used and the cross-clamp time was 30 min.

The postoperative course was uncomplicated. Microscopic examination revealed mild chronic atherosclerotic changes of the carotid arterial wall and the presence of inflammatory infiltration and arterial degradation of the resected aneurysm’s wall (Fig. 2). Microbial cultures of the aneurysm’s sac demonstrated S. parasanguinis sensitive to amoxicillin–clavulanic acid. This set the definite diagnosis of an MCA. The patient took 1 g of this antibiotic twice daily for 1 month. The follow-up regimen included clinical, laboratory and duplex ultrasound examination at the time of patient’s discharge and at the first and third months. Three months postoperatively the patient is in excellent condition without clinical or laboratory findings of recurrent infection and with patency of left carotid arteries.

Discussion

The presence of a pulsatile neck mass associated with fever and pain are strongly correlated with clinical diagnosis of an MCA. The laboratory diagnosis is made with either a positive culture or an evidence of organisms on histologic examination of the arterial wall. Differential diagnosis includes neck tumours, carotid body tumour, enlarged lymph nodes and a redundant or kinked carotid artery, but in cases of non-pulsatile thrombosed aneurysm diagnosis might be difficult.1–3

Figure 1  MR tomography shows the pseudoaneurysm (left) and MR angiogram of carotid arteries (right). The arrow indicates the neck of the saccular pseudoaneurysm and the external compression of the left internal carotid lumen that was misdiagnosed as tight atherosclerotic ulcerated stenosis.

Figure 2  Light microscope pathological examination (hematoxylin–eosin stained, ×100) of the pseudoaneurysm’s wall, consisted from inflammatory infiltrated fibroblastic tissue (Fibr) in close relation with the surrounding adipose tissue (Ad). Thr indicates the presence of endoluminal thrombus.
The surgical management includes resection of the MCA and restoration of vascular continuity by some type of reconstructive technique. The use of venous patch or venous interposition graft is the best option to prevent short- and especially long-term infections and recurrences. Unfortunately, in our case, the final diagnosis was set postoperatively and we used a synthetic patch. However, the short-term follow-up showed no evidence of recurrent infection and the patient is in a strict surveillance protocol. Relevant literature search showed a few MCA case reports but this is the first with a thrombosed non-pulsatile MCA, with no clinical signs of local or systematic infection.

Ligation of carotid arteries might be unavoidable when revascularisation is technically impossible. In that case a carotid back-pressure of 50 mmHg indicates adequate collateral flow, but back-pressure of 70 mmHg is safer.

In conclusion surgical treatment is always indicated to resect the MCA and to revascularise the internal carotid artery by some reconstructive technique, usually with autologous material.

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**References**