La Prensa Medica Argentina

Case Report

A Case of Giant Extracranial Carotid Artery Aneurysm

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Abstract

Extracranial carotid aneurysms are uncommon. Most patients remain asymptomatic. Local pain and a pulsating mass in the neck are the most frequently described symptoms. Most carotid artery aneurysms are caused by atherosclerosis, other common causes include trauma and infection. Surgical management of a 80-year-old female patient with a giant extracranial internal carotid artery aneurysm is presented in this report .

Introduction

Extracranial carotid artery aneurysms are an uncommon entity, comprising 0.4% to 4% of all peripheral artery aneurysms [1,2]; only 0.1% to 2% of all the carotid surgery procedures are performed for aneurysms [3].

Local pain and a pulsating mass in the neck are the most frequently described symptoms [1,4,5]. Patients might also present with ischemic stroke, transient ischemic attack, amaurosis fugax, due to thromboembolism; or symptoms of damage to cranial nerves related to the compression by the aneurysm. Most patients, however, remain asymptomatic.

Although most carotid artery aneurysms are caused by atherosclerosis, other common causes include trauma and infection. Less frequent causes are cystic medial necrosis, Marfan syndrome, Takayasu arteritis, and idiopathic medial arteriopathy [2].

We present a giant extracranial internal carotid artery (ICA) aneurysm which was treated surgically.

Case Report

A 80-year-old woman was admitted to our surgical unit with a diagnosis of extracranial internal carotid artery (ICA) aneurysm. The patient presented with a large, growing, pulsatile mass on the left side of the neck. She reported persistent symptoms of dysphagia, and transient hypophonia; there was no history of cerebrovascular symptoms or cervical trauma. Physical examination revealed a pulsatile mass on the left side of the neck with a palpable thrill and systolic bruit. MRI and MRI-angiography revealed a wide necked, saccular, "diverticulum-like" aneurysm at the origin of the left ICA, developing from the side of the carotid wall in a diverticular shape, with a longitudinal diameter of 18 mm and a transverse diameter of 25 mm (Figure 1,2).

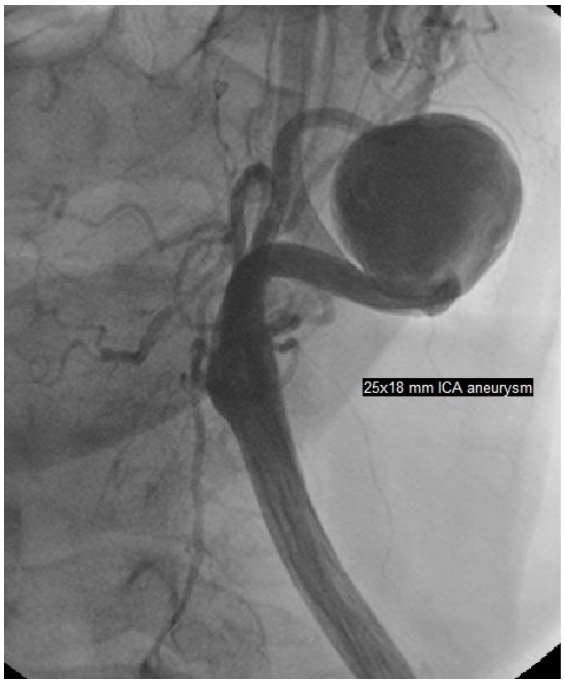

Figure 1: Axial MRI image of the aneurysm

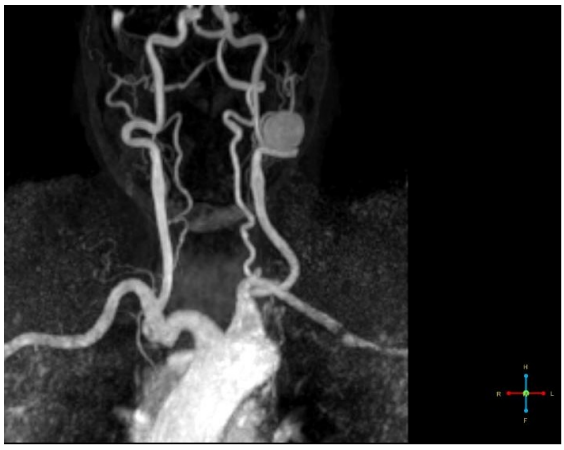

Figure 2: MRI-angiography showing the aneurysm

Patient was scheduled for surgical repair. Endovascular greft stenting had not been considered due to high degree tortiosity of the vessel. Due to her anginal symptoms and high coronary risk profile, we performed conventional coronary and bilateral carotid angiography before continuing with surgery. There was a non-

significant lesion in the left circumflex artery and a giant aneurysm at the origin of the left ICA(Figure 3).

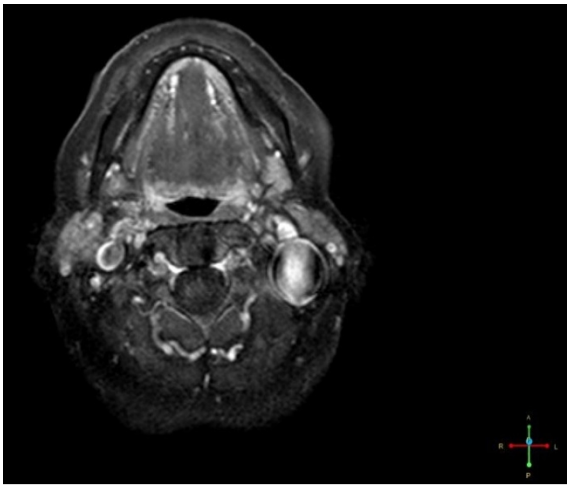

Figure 3: Conventional angiographic image of the aneurysm

The right ICA and both of the vertebral arteries were patent. Under general anesthesia, patient's neck was slightly extended, and the head was turned right and placed on a foam ring. Cerebral ischemia was monitored during the operation by means of somatosensitive evoked potentials and bispectral index. A left longitudinal neck incision was performed to explore the left common, external, and internal carotid arteries. Extensive manipulation was avoided to prevent distal embolization of any debris from the aneurysm. Carotid shunt was not used since no signs of cerebral ischemia were detected throughout the operation. Aneurysm was arising from the medial wall of the ICA. Cross-clamps were placed on the ICA, distal and proximal to the aneurysm. Aneurysm was resected (Figure 4).

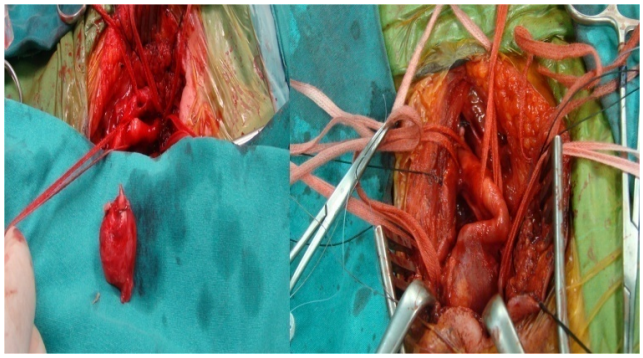

Figure 4: Intraoperative view of the aneurysm

Both free ends of the artery could be elongated taking advantage of the tortuousity. End-to-end anastomosis of the native artery was possible without using an interposition graft. Total cross-clamp time was ten minutes. Her postoperative course was uncomplicated and on the 3rd day after surgery the patient was discharged from the hospital in a good ambulatory condition. Pathological evaluation of the surgical specimen revealed a pseudoaneurysm. Histopathological examination showed the disappearance of elastic fibers in the tunica media replaced by collagen fibers in the wall of aneurysm (Figure 5A,B).

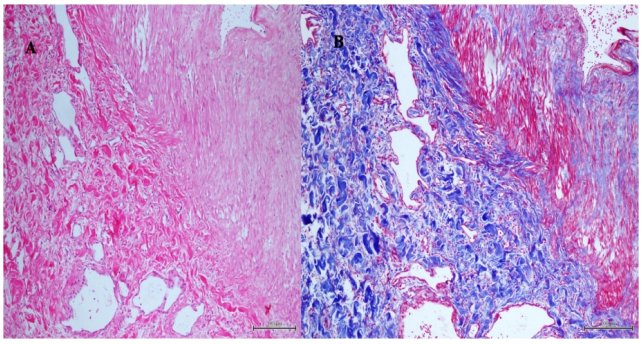

Figure 5A: Histopathological examination demonstrating disappearance of elastic fibers in the tunica media replaced by collagen fibers in the wall of the aneurysm (x40, H&E). 5B: Masson's trichrome stain (x100) and rich capillary channels

Control CT angiography in her second year of follow up, showed a patent anastomosis without any recurrences (Figure 6).

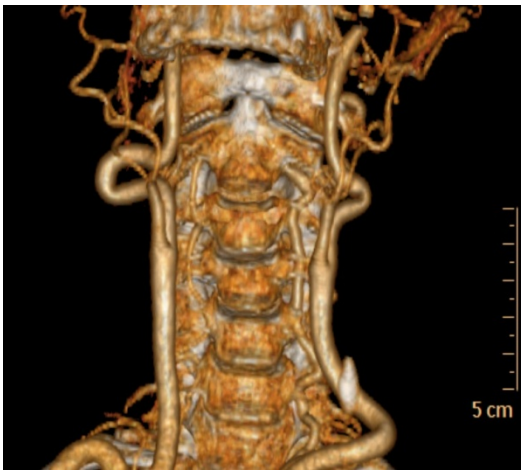

Figure 6: Second year control CT angiographic image showing patent anastomosis, without any recurrence of the pseudoaneurysm

Discussion

Carotid and vertebral pseudoaneurysms are uncommon lesions that may occur as sequelae of blunt trauma, cancer/radiation necrosis, or mycotic infection. Historically, treatment of pseudoaneurysms has been primarily surgical [6]. However, surgery of pseudoaneurysms located near the skull base is technically challenging and has been associated with high morbidity/mortality rates [7,8]. More recently, endovascular techniques have become more appealing as multiple studies have provided encouraging results [6,9-10].

Extracranial internal carotid artery aneurysms are defined as localised increases of the calibre of more than 50% as compared with the reference values of the ICA (0.55±0.06 cm in men; 0.49±0.07 in

women). They are very rarely seen and they may be classified like true or pseudoaneurysms according to their different aetiologies.

Pseudoaneurysms are mainly secondary to previous endoarterectomy with the onset of symptoms ranging from 1 month to 15 years after the original carotid procedure; other contributing factors of pseudoaneurysms may be trauma (car accidents, stab-wounds, iatrogenic central venous cannulation) and infections: patch complications or recurrent tonsillitis or pharingitis reaching the carotid artery wall following peritonsillar abscess with subsequent ischemia of the wall leading to acute rupture or causing septicemia and invasion of vasa vasorum [11].

True aneurysms are most commonly due to atherosclerosis or fibromuscular dysplasia. Atherosclerotic degeneration is assumed to be the most frequent etiology [12,13]. Atherosclerotic Aneurysms (AAs) are usually found in patients aged 50 to 70 years old with a male/female ratio of 1.9/1 [14]. AAs may involve various portions of the carotid artery: they tend to be located at bifurcation and at the proximal ICA, size ranging from 1.5 to 5.0 cm, whereas dysplastic aneurysms are often associated with chronic dissection, and are located at more distal segments [15].

Other rare contributing factors to aneurysm formation include neck irradiation, neurofibromatosis, Marfan's Syndrome, Behcet's Syndrome and Takayasus's arteritis [16]; 30% to 60% of extracranial ICA aneurysms are symptomatic for thrombo-embolic focal or non focal symptoms; more rarely cranial nerve compression may result in neurological deficits. Spontaneous progression of extracranial ICA aneurysms is associated with a higher risk of mainly thromboembolic neurological complications; other adverse events may be rupture, with massive haemorrhage, and nerve compression. Conservative treatment is based on anti-coagulant or anti-aggregant therapy. A non-surgical approach may be indicated in young patients with asymptomatic, traumatic or spontaneous dissecting aneurysms. In many cases, however, medical treatment is considered to be ineffective or even dangerous, with a persistent risk of haemorrhage [17].

At present, the indications for intervention in patients with extracranial carotid aneurysms have not been completely brought to light. In the event of symptoms, an intervention is clearly indicated. To date, the risk of complications of an asymptomatic aneurysm and its relation to issues like aneurysmal diameter and plaque instability have not been studied. The choice between endovascular repair and conventional surgery can depend on various factors, including the type of aneurysm, comorbidities, symptoms, and the experience and preference of the surgeon. The relatively low incidence of these lesions makes it difficult to obtain sufficient experience with endovascular procedures for carotid aneurysm. However, growing proficiency with carotid stenting for stenotic lesions might provide adequate interventional skills. Intraprocedural complications from carotid stent placement are attributable to thromboembolism. McCready et al, [18] describe 2 of 4 patients who developed transient ischemic attacks secondary to thromboembolism in middle cerebral artery branches following covered-stent placement for an ICA pseudoaneurysm.

The most frequent surgical procedures for AA's are represented by: total resection of the aneurysm with direct end-to-end anastomosis or interposition of a prosthetic graft or the saphenous vein. In our case we performed surgical repair with direct end-to-end anastomosis. Endovascular greft stenting has not been considered due to increased tortuosity of the vessel. The patient course was uneventfull.

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