

Carotid artery stenting in patients with brain meningioma



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The association between carotid artery stenosis and brain meningioma is rather rare. Indeed, data in literature about this association are scarce and potential risks of carotid artery stenting using different protection devices are not known. We describe the endovascular treatment of severe carotid stenosis in two patients with intracranial meningioma.

KEY WORDS: Brain meningioma, Carotid artery stenosis, Carotid artery stenting

Introduction

Meningioma is generally a benign lesion and represents the second most common primary neoplasm enclosing 18% of all intracranial tumors. Because most of meningiomas do not produce symptoms, the true incidence is probably underestimated¹. Presenting signs and symptoms depend on the size and location of the lesion and include headache, seizure, and neurologic deficits due to local mass effect².

Coexistence of a brain tumour and a severe carotid stenosis is rarely reported in the literature. Herein we describe the endovascular treatment of three severe carotid stenosis in two patients (one bilateral) with intracranial meningioma.

Case Reports

PATIENT N. 1

A 69-year-old female patient with history of hypertension, dyslipidemia was admitted to our unit for evaluation of bilateral asymptomatic carotid stenosis. The duplex scan showed a type III calcified plaque according to Gray-Weale classification³ in the left internal carotid artery (ICA) with stenosis > 80% and peak systolic velocity (PSV) > 230 cm/sec and type II soft plaque with ulceration in right internal carotid and stenosis > 70% with PSV of 215 cm/sec.

Contrast-enhanced computer tomography (CT) confirmed severe bilateral carotid stenosis and highlighted the presence of parafalcine lesion with diffusely enhanced content (Fig. 1). We decided to investigate this lesion with magnetic resonance imaging (MRI) that revealed an intracranial meningioma. The consultant neurosurgeon excluded treatment of this lesion.

Under local anesthesia via a percutaneous transfemoral access, we implanted a 7 mm X 30 mm Carotid Wallstent (Boston Scientific, Natick, Massachusetts, USA) in the left ICA after position of distal embolic

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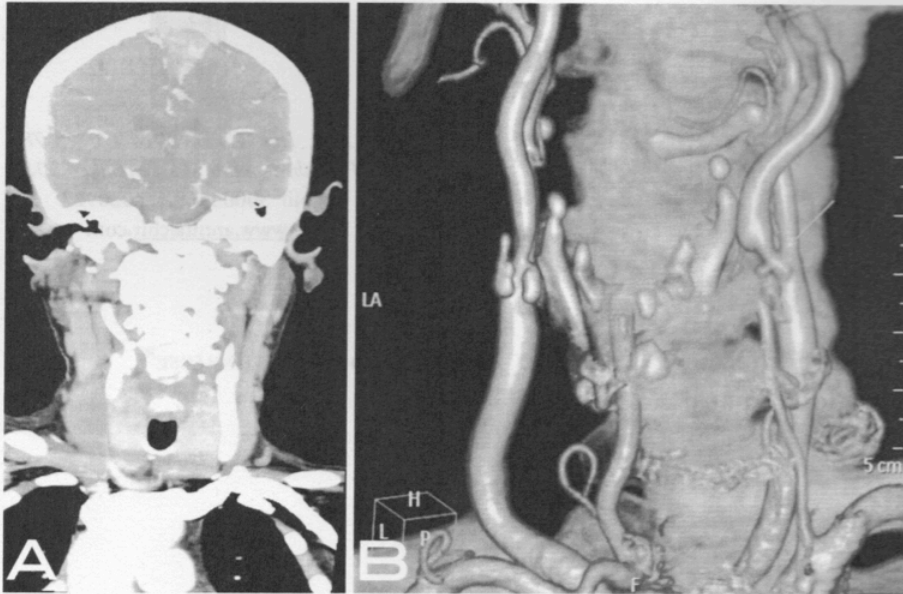


Fig. 1: (A) Preoperative CT scan showing frontal falx meningioma and bilateral ICA stenosis. (B) Posterior view of volume-rendering image highlighting calcification of left carotid plaque and ulceration of the right carotid plaque (white arrow).



Fig. 2: Digital subtraction angiography. (A) Right common carotid injection via Mo.Ma. device (Invatec, Roncadelle, Italy) showing a tight stenosis of the ICA. (B) Poststenting (6-8 x 30 mm Protegè Rx (EV3, Minneapolis, Minnesota, USA)) right carotid angiogram demonstrating no residual stenosis. Left 7 x 30 mm monorail Carotid Wallstent (Boston Scientific, Natick, MA, USA), previously implanted, is noted in both images (black arrows).

protection device (Spider FX 5mm, EV3, Minneapolis, Minnesota, USA) using 120 cc of non-ionic contrast medium (Iodixanol (Visipaque®)).

The postoperative course was uneventful and the patient was discharged on dual antiplatelet therapy the day after the procedure. A contralateral carotid artery stenting (CAS) procedure was scheduled within 2 months.

Therefore, at readmission, a right carotid stenting under proximal endovascular flow blockage for cerebral protection (9 Fr Mo.Ma Ultra, Invatec, Roncadelle, Italy) was planned because of a type II soft plaque. A percutaneous transfemoral approach was achieved and a 6-8 mm x 30 mm Protegè Rx (EV3, Minneapolis, Minnesota, USA) was deployed (Fig. 2) using 90 cc of non-ionic contrast medium (Iodixanol (Visipaque®)). During procedure, the

patient complained clamping intolerance (total clamping time of 5 minutes), with severe headache and contralateral leg pain resolved after 24 hours of antiedematous therapy (dexamethasone 4mg/6h). Postoperative MRI didn't show any acute cerebral lesion or peritumoral edema and the patient was discharged on postoperative day four in good condition. She was doing well 18 months after the last procedure.

PATIENT N. 2

A 64-year-old female patient with history of coronary artery disease, hypertension, dyslipidemia, COPD and smoking, was admitted to our unit for evaluation of an

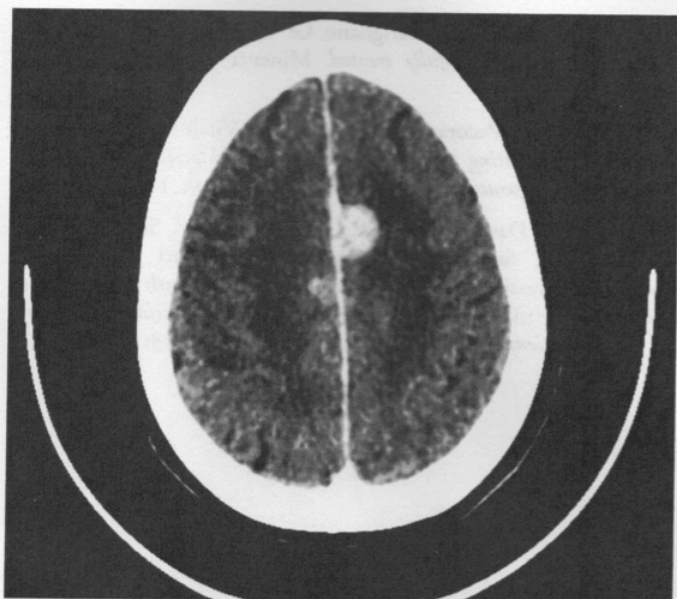


Fig. 3: Preoperative CT scan of brain showing a mass adjacent to the falx cerebri in the left frontal lobe.

asymptomatic right ICA stenosis. The duplex scan showed a type III calcified plaque at the origin of right ICA determining stenosis >80% with PSV of 244 cm/sec. The subsequent contrast-enhancement CT scan confirmed severe right carotid stenosis and revealed the presence of parafalcine mass with diffusely enhanced content (Fig. 3). MRI indicated that an intracranial meningioma was the most likely diagnosis. She was seen by a neurosurgeon who advised no treatment of meningioma. Under local anaesthesia, a 7 mm x 40 mm Carotid Wallstent (Boston Scientific, Natick, Massachusetts, USA) was deployed in the left ICA after position of distal embolic protection device (Spider FX 5mm EV3, Minneapolis, Minnesota, USA). Total contrast medium amount was 140 cc of Iodixanol (Visipaque®). The patient was discharged the day after this uncomplicated procedure on dual antiplatelet therapy. Follow-up with clinical and imaging examinations at 16 months showed no sign of restenosis.

Discussion

The real effect of cerebral meningioma in patients undergoing carotid procedures has not been extensively evaluated. Several authors have supposed that both general anaesthesia⁴ and changes in cerebral blood flow (i.e. during carotid cross-clamping) increase the risk of cerebral edema in patients with brain meningioma⁵. Recently, Sun and Ross reported two patients who experienced fatal hemorrhagic infarction of a meningioma after an otherwise uncomplicated cardiac surgery. They conclude that possible causes of tumor swelling and infarction may depend to relative hypotension while on

bypass resulting in poor perfusion to the tumor, decreased plasma osmolality with a resulting increase in peritumoral brain edema, a hypercoagulable state after reversal of anticoagulation at the end of the procedure, and increased blood brain barrier permeability during cardiopulmonary bypass⁶.

Merlo *et al.* reported the successful surgical management of severe carotid stenosis in two patients with brain tumor. In their opinion, barbituric administration and temporary intraluminal shunt placement is mandatory in order to avoid cerebral perfusion deficit during carotid clamping⁷.

In the last ten years, CAS has emerged as less invasive therapy for extracranial internal carotid artery disease avoiding the use of general anaesthesia and prolonged carotid clamping. At our institution, CAS with embolic protection device is the technique of choice in patients with contralateral severe stenosis or occlusion and in patients at high surgical risk⁷. High risk for surgery is defined by SAPHIRE criteria⁸.

Although our experience is based only on two patients, we assumed that CAS with distal embolic protection device is feasible and safe in presence of brain meningioma; on the contrary the use of proximal cerebral protection device should be deeply evaluated in these patients. To our knowledge, this is the first paper reporting these particular findings.

Further studies with larger patients cohorts are advocated to elucidate the influence of meningioma on neurologic complication after treatment of carotid artery stenosis.

Riassunto

Il meningioma è un tumore cerebrale piuttosto frequente, benigno in oltre il 90% dei casi.

La reale incidenza è probabilmente sottostimata a causa della sua scarsa sintomatologia clinica.

In letteratura i pochi lavori che riportano esperienze di chirurgia cardiaca e vascolare in pazienti con meningioma ipotizzano che le modificazioni del flusso, dovute o al clampaggio dei vasi cerebro-afferenti o all'anestesia generale, aumentino il rischio di edema cerebrale post-operatorio.

Scopo di questo lavoro è riportare e discutere il trattamento endovascolare con stent di stenosi carotidiche in due pazienti con meningioma cerebrale.

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