

liver function parameters remained constantly elevated but in a tolerable range, without sudden increases, as previously described (Fig 1). AchR-antibodies had their ups and downs, however they also remained stable until the last follow-up in March, 2011 (Fig 2). Since the detection of PSC, the patient has never complained about liver symptoms. Total bilirubine did not exceed a maximal value of 3.0 mg/dL during five years. Since the last myasthenic crisis, she remained clinically stable, with a persistent slight ptosis on the right side.

PSC is a chronic, inflammatory disease of the bile ducts, complicated by fatigue, pruritus, fibrotic biliary strictures, liver cirrhosis, and liver failure necessitating liver transplantation, cholangiocarcinoma, or colorectal cancer³. The etiology of PSC is unknown but autoimmune mechanisms and genetic factors seem to play a pathogenetic role³. Although there is no causative treatment yet available, patients may profit from endoscopic therapy of biliary stenoses and inflammatory bowel disease⁴.

The presented report shows that in case of MG and collateral PSC, liver-toxic immunosuppression with AZT is tolerable and effective even over a longer period of time if the dosage is adapted according to the impaired liver functions. Low-dose AZT has a sufficient therapeutic effect on MG without deterioration of PSC. Possibly, AZT was beneficial even for PSC, since the patient exclusively required ursodesoxycholic acid, PSC did not progress so far, and AZT has also been recommended as a treatment of PSC⁵. Immunosuppressive agents other than AZT were considered, but they were rejected because AZT is the only approved immunosuppressive drug for MG and since other agents are potentially liver-toxic as well. Continuation of steroids was also considered since they may have a beneficial effect on PSC⁵ and on MG, but they were lastly stopped for their side effects, their ineffectivity on the AchR-antibody titers, and since PSC did not deteriorate after their discontinuation.

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Rare case of carotid artery occlusion due to thrombosis of a giant cerebral aneurysm. The role of cerebral revascularization.

Caso raro de oclusão da artéria carótida devido à trombose de um aneurisma cerebral gigante. O papel da revascularização cerebral.

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Conflict of interest: There is no conflict of interest to declare.

Received 16 August 2011; Received in final for 06 September 2011; Accepted 13 September 2011

Spontaneous thrombosis of a giant cerebral aneurysm is a recognized phenomenon, however it becomes rare when the thrombosed aneurysm is associated with the occlusion of its parent artery^{1,2}. The best management strategy is not defined yet. Theoretically, it should

be directed to alleviating mass effect related symptoms caused by the aneurysm itself and to preventing cerebral ischemia.

We describe a case of carotid artery occlusion and discuss the role of cerebral revascularization.

CASE REPORT

A 69-year-old woman had suffered from left painful ophthalmoplegia with a sudden onset. She had consulted her ophthalmologist, who suspected a left cavernous sinus syndrome. A computed tomography of the head was obtained, and the patient was referred to our service with a diagnosis of brain tumor.

On admission, the patient was conscious and the left cavernous sinus syndrome was confirmed. The initial computed tomography (CT) scan showed a high-density parasellar round lesion extending to the left middle cranial fossa. Magnetic resonance imaging revealed a giant thrombosed aneurysm of the left cavernous internal carotid artery (Fig 1). Further investigation with a four-vessel cerebral angiography (Fig 2) obtained 10 days after the onset of presentation revealed the finding of left internal carotid artery (ICA) occlusion with good cross filling in

the left-side circulation through the anterior communicating artery. The patient was treated conservatively and her symptoms gradually improved. She was discharged on antiplatelet treatment.

At the 3-month follow-up consultation, the patient was complaining of some brief episodes of language disturbance related to verbal expression.

After demonstration of cerebral flow asymmetry by a single photon emission computed tomography (SPECT) study, the patient underwent extra-intracranial bypass surgery using the technique described by Yasargil³ to anastomose the left superficial temporal artery to the middle cerebral artery. Postoperative course was uneventful and control cerebral angiography confirmed bypass patency.

At follow-up consultation 3-years later, the patient was asymptomatic and CT angiography (Fig 3) showed that the bypass remained patent.

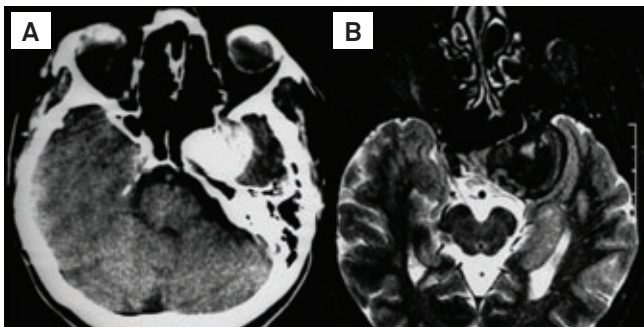


Fig 1. (A) The computed tomography scan showing a high-density parasellar round lesion extending to the left middle cranial fossa. It was first misdiagnosed as a brain tumor. (B) Axial T2 weighted image revealing the giant thrombosed aneurysm.

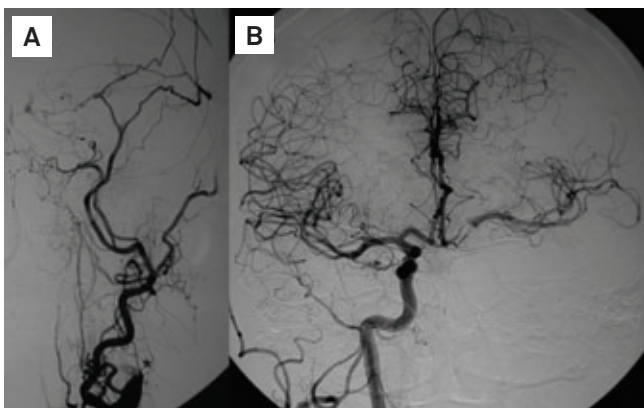


Fig 2. (A) Oblique view of the left carotid angiogram demonstrating occlusion (,) of the proximal internal carotid artery in the neck. (B) Frontal view of the right carotid angiogram revealing good cross filling in the left side through the anterior communicating artery.

DISCUSSION

The angiographic finding of an ICA occlusion in the case of a parasellar cerebral aneurysm is very fortunate if there is good cross filling to the contralateral side and the patient is asymptomatic. We suggest that it cannot be considered a “self-treated lesion” unless a functional cerebral blood flow study (transcranial Doppler, positron emission tomography or SPECT) is within normal limits. When considering the indication of bypass surgery for the aim of flow augmentation, patient selection includes clinical criteria, such as recurrent transient ischemic attack and ce-



Fig 3. Postoperative computed tomography angiography showing a patent left-side superficial temporal artery-middle cerebral artery (STA-MCA) bypass.

rebral fluid flow study criteria of reduced cerebrovascular reserve capacity⁴.

A report has been published in which aneurysmotomy and thrombectomy of a thrombosed giant intracavernous carotid aneurysm were performed and produced mass effect relief with symptomatic improvement². Similarly, our

patient had remission of mass effect symptoms, but with conservative management.

Our experience with this case has convinced us that a close follow-up is paramount in a patient with an occluded internal carotid artery since surgical intervention may be necessary.

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