Bacterial Cavernous Sinus Aneurysm Treated by Detachable Balloon Technique

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We describe a patient who developed bilateral cavernous sinus septic thrombosis secondary to a suppurative lesion on the left cheek. Despite clinical improvement, left oculomotor symptoms recurred suddenly. A carotid artery aneurysm within the cavernous sinus was diagnosed by means of magnetic resonance imaging and confirmed by digital angiography. Follow-up angiograms showed an initial decrease in the aneurysm size, with subsequent enlargement. A latex contrast-filled balloon was successfully placed within the aneurysm, preserving the carotid parent artery blood flow. Our case illustrates the usefulness of the detachable balloon technique in the treatment of bacterial aneurysms of the cavernous sinus as an alternative treatment to carotid artery ligation. (Stroke 1989;20:1751-1754)

Suppurative lesions in the middle third of the face, commonly due to Staphylococcus aureus, are apt to spread into the cavernous sinus, causing septic thrombosis. The clinical picture may be quite severe, and septic thrombosis often leads to death. Commonly associated with sphenoidal sinusitis,1 10% of cavernous sinus thrombophlebitis cases arise from odontogenic infections,2 particularly before antibiotics became available.

In the past, angiography was essential to confirm diagnosis,3,4 but recently computed tomography (CT) has been considered a reliable tool.5-7 The mortality rate reaches 30%, while 40% of those with cavernous sinus thrombophlebitis have a full recovery and 30% suffer neurologic sequelae such as loss of visual acuity and oculomotor impairment.8

We describe a patient with bilateral cavernous sinus involvement who developed a bacterial aneurysm of the intracavernous carotid artery, which was not disclosed at CT scanning and which was successfully treated by intra-arterial balloon placement.

Case Report

A 19-year-old man originally presented with a furuncle on his left cheek that worsened during the course of 1 week until the entire left side of his face was affected by cellulitis. Surgical drainage was then carried out, and antibiotics were prescribed but not taken. He was admitted to the University Hospital 3 days later after exhibiting local aggravation and fever accompanied by anorexia, hyperthermia, nausea, and vomiting.

On examination, the patient was alert and well oriented to time and space. There was severe swelling of his entire left cheek, extending to the frontal area and the ipsilateral side of his neck, together with a submaxillary adenopathy and a peripheral facial palsy, both on the left side. Initial laboratory data showed 15,700 leukocytes/mm³ with mostly polymorphonuclear cells.

The patient was put on 12 g/day cefotaxime, 1 g/day amikacin, and 1.2 g/day rifampin for up to 24 days. However, he became drowsy 2 days after the first dose and developed bilateral ptosis and IV and VI nerve palsies as well as a right mydriasis and neck rigidity. Bilateral basal lung hypoventilation was also evident. A CT scan was normal, but cerebrospinal fluid (CSF) from a lumbar puncture revealed 200 mg/dl protein, 66 mg/dl glucose, and 330 leukocytes/mm³ (90% polymorphonuclear cells). A CSF culture grew no organisms. Within 24 hours the patient’s oculomotor symptoms progressed to bilateral total ophthalmoplegia with mild proptosis and normal fundus oculi. Respiratory impairment worsened, with right basal lung condensation signs and pronounced dyspnea. Blood cultures grew S. aureus.

During the next few days, the patient became more alert and his ophthalmoplegia abated somewhat, but his fever persisted and his lung function deteriorated. A second lumbar puncture yielded xanthochromic CSF and 180 leukocytes/mm³ with 70% neutrophils, 47 mg/dl glucose, and 180 mg/dl...
protein but negative cultures. Pleural drainage material disclosed purulent hematic fluid with 96% glucose, pH 7.34, lactic dehydrogenase 931 IU, 4,360 leukocytes/mm³, and 3.5 g% of protein. An echocardiogram proved normal. At 10 days' follow-up (8 days after onset of symptoms), despite progressive clinical improvement, the patient’s left oculomotor symptoms abruptly recurred. Magnetic resonance imaging (MRI) disclosed a multilobular carotid artery aneurysm within the cavernous sinus that was confirmed by digital angiography (Figure 1). Follow-up chest x-ray films and CSF analyses were normal.

After 6 weeks the patient was discharged with normal neurologic findings except for a mild left ophthalmoparesis. Given evident clinical improvement during the fourth week, intravenous antibiotic therapy was replaced by 4 g/day cephalexin for 30 days and 0.6 g/day cephalexin plus 1.2 g/day rifampin for 12 days, both by oral route. Angiograms taken 20 and 40 days after discharge showed a progressive decrease in aneurysm size (Figure 2), but a third angiogram performed 19 weeks after the onset of symptoms showed enlargement of the aneurysm to its original size (Figure 3) without clinical worsening.

The patient was given a single 1-g cephalothin dose, and a contrast-filled balloon was placed within the aneurysm sac, preserving the carotid artery blood flow (Figure 4) without inducing further symptoms. Sequential skull x-ray films showed progressive balloon shrinkage over the following months. At 6 months' follow-up, a fifth angiogram demonstrated complete disappearance of the aneurysm, with residual carotid artery irregularities within the cavernous sinus. At that time no neurologic abnormalities could be detected.

Discussion

Bacterial aneurysms are caused mainly by septic emboli secondary to bacterial endocarditis. However, there are also extravascular sources of emboli following arterial wall infections by adjacent foci as well as cryptogenic origins of emboli that lack evidence of septic processes. As a whole, bacterial aneurysms account for 2.5–6.2% of intracranial aneurysms.10,11

Although its course through the cavernous sinus would appear to render the carotid artery highly susceptible in the event of cavernous septic thrombosis, there have been only 22,12–23 documented cases of bacterial cavernous sinus aneurysms thus far, with approximately half of the cases occurring in children. Most of the cases have been attributable to facial S. aureus infection. The fact that many such cases have been detected in less developed countries leads one to conjecture that the true incidence would be much higher if MRI, CT, and angiographic facilities were routinely available.

Before CT scanning, the differential diagnosis of orbital cellulitis was difficult and depended on the presence of severe bilateral oculomotor involvement and/or associated meningitis.22 Recently, CT has been able to discriminate infection restricted to
FIGURE 3. Left carotid arteriogram performed 19 weeks after onset of symptoms demonstrating enlargement of aneurysm.

FIGURE 4. Left carotid roentgenogram showing detachable balloon within aneurysm sac, with preservation of internal carotid artery blood flow.

Typically, our patient presented with ophthalmoplegia with ptosis and proptosis secondary to facial staphylococcal infection. Bilateral oculomotor disturbances together with meningeal involvement supported a diagnosis of cavernous sinus septic thrombosis. Nevertheless, CT failed to show filling defects or other abnormalities consistent with this entity.4-6,26,27 Despite clinical and transient ophthalmologic improvement, the patient’s left oculomotor symptoms markedly worsened 8 days after their onset. MRI disclosed abnormalities suggestive of a left carotid artery aneurysm within the cavernous sinus; the aneurysm was subsequently confirmed by digital angiography.

The fact that a CT scan was normal whereas an MRI scan 20 days later patently demonstrated a bacterial cavernous sinus aneurysm should be stressed. Furthermore, serial angiography proved invaluable for follow-up and timing of surgery, since bacterial aneurysms are known to disappear,9,13,17,19 enlarge,7,15,16,19 or develop thrombosis19 during antibiotic treatment. Moreover, serial angiography has been advocated to preclude the risk of life-threatening rupture,10,19,28,29 even on suspicion of asymptomatic aneurysms secondary to infectious foci as, for example, due to bacterial endocarditis.

Surgical treatment for cavernous sinus aneurysms is difficult and is generally intended to exclude or occlude the malformation while sparing blood flow through the parent artery. Up to now most bacterial cavernous sinus aneurysms have been treated by carotid ligation, a procedure still employed for unclippable aneurysms,30 although associated morbidity is >28%.31 The detachable balloon device was first described by Serbinenko32 in 1974 and became widely known after the valuable contribution of Debrun et al33 to this technique. Such treatment for giant intracranial aneurysms, including those involving the cavernous segment of the carotid artery, is aimed at avoiding neural compression or carotid cavernous fistulae.34 Fox et al34 reported residual neurologic impairment in 1.5% but mortality in none in a series of patients receiving the detachable balloon.

Our case illustrates the usefulness of MRI in diagnosing aneurysms of the cavernous sinus in the absence of CT evidence when septic thrombosis is suspected. However, serial angiograms allow diagnostic confirmation and radiologic follow-up, thus enabling surgical planning when there is undue enlargement of the aneurysm despite antibiotic treatment. The detachable balloon technique has proven
to be a simple and reliable occlusive procedure quite feasible for bacterial aneurysms when the acute stage of arterial wall inflammation has abated.

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