Fatal Subarachnoid Hemorrhage from an Inflammatory Cavernous Carotid Artery Aneurysm: Failure of Conservative Treatment After Early Diagnosis

—Case Report—

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Abstract

Inflammatory carotid artery aneurysm is a rare complication of acute paranasal sinusitis. A 50-year-old female presented with a ruptured giant carotid artery aneurysm secondary to infection of the sphenoid sinus and cavernous sinus. She had been healthy until 5 days before admission, when she developed orbital phlegmon and meningitis. She received antibiotic therapy for 10 days. Computed tomography (CT) of the brain 2 days after admission showed no abnormality. However, repeat CT on day 6 showed a round isodense mass in the suprasellar cistern suggesting a cerebral aneurysm. Twelve days after admission, she suffered a fatal subarachnoid hemorrhage. Cerebral angiography revealed a giant left cavernous carotid artery aneurysm with a very irregular shape. Autopsy found sphenoid sinusitis and osteomyelitis extending into the cavernous sinuses. Diagnosis of bacterial inflammatory aneurysms before rupture is very important. Appropriate surgical intervention should be considered if there is enlargement of the original aneurysm or appearance of a new aneurysm indicating a potentially dangerous situation.

Key words: inflammatory cerebral aneurysm, subarachnoid hemorrhage

Introduction

Inflammatory cerebral aneurysms due to septicemia and meningitis usually arise in the small distal branches of the cerebral vessels. A few inflammatory cavernous carotid artery aneurysms secondary to paranasal sinusitis have been reported. Aneurysms of the intracavernous carotid artery usually cause cavernous sinus syndrome with abnormal neuroimaging findings. Rupture of a cavernous carotid artery aneurysm usually causes a carotid cavernous fistula and rarely results in subarachnoid hemorrhage.

We report an unusual inflammatory aneurysm arising primarily from the cavernous portion of the internal carotid artery, but with extension into the intradural space, which resulted in a fatal subarachnoid hemorrhage.

Case Report

A 50-year-old female had been in good health until 5 days before admission, when she experienced right retrobulbar pain due to orbital phlegmon. She was admitted to the Department of Ophthalmology of our institution. On admission, her body temperature was 38.7°C, pulse 92/min, and blood pressure 136/70 mmHg. Physical examination revealed right exophthalmos with chemosis. No bruit was audible over the eyes or head. Heart sounds were normal. Neurological examination revealed right abducens nerve palsy. No other neurological abnormalities were present. Initial peripheral blood studies revealed a white blood cell count of 19,300/mm³ with

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89% neutrophils, and a markedly elevated C-reactive protein level. She was treated with high doses of intravenous antibiotics. The fever subsided and the swelling of the right eyelid decreased. However, she developed meningitis and was referred to the Department of Neurology.

Precontrast and postcontrast computed tomography (CT) of the head 2 days after admission showed no abnormality. A lumbar puncture yielded slightly turbid cerebrospinal fluid (CSF) under an elevated opening pressure of 200 mmH₂O. Analysis of the CSF showed 238 cells/mm³ (32% neutrophils and 68% lymphocytes), 1.01 g/l of protein, and 0.58 g/l of glucose. Gram staining showed no organisms. Assays for syphilis, latex agglutination for cryptococcal antigen, and smear for mycobacteria were negative. Blood and CSF cultures for bacteria, fungi, and viral studies were all negative. She received antibiotic therapy for 10 days with an improvement in her clinical condition and laboratory findings. Precontrast CT scan of the head 6 days after admission showed a new, round isodense mass in the suprasellar cistern suggesting the formation of a cerebral aneurysm. Magnetic resonance (MR) imaging on day 7 revealed an abnormal signal void in the left cavernous sinus (Fig. 1) and in the suprasellar cistern. Her clinical condition continued to improve over the following days.

On day 12 she complained of a severe headache. Her condition suddenly deteriorated, and required in-

Fig. 1 Postcontrast T₁-weighted MR image, coronal view, 7 days after admission demonstrating an abnormal signal void in the left cavernous sinus (arrow).

Fig. 2 Left internal carotid angiograms, anteroposterior (left) and lateral (right) views, demonstrating a giant cavernous carotid artery aneurysm (arrow) projecting superiorly and medially, and extending into the intradural space.

Fig. 3 Photomicrographs of the autopsy specimen of the left internal carotid artery just proximal to the aneurysm showing loss of continuity of the media (arrows) with marked intimal proliferation (asterisk) (upper: Masson and elastica van Gieson stain, × 5), and infiltration of polymorphonuclear leukocytes into the adventitia (lower right corner) (lower: HE stain, ×50).
tubation and mechanical ventilation. She developed fixed, dilated pupils. CT revealed diffuse, thick subarachnoid hemorrhage and a frontal intrahemispheric hematoma. She was referred to the Neurosurgery Department for surgical treatment. Cerebral angiography revealed a giant left cavernous carotid artery aneurysm extending into the intradural space (Fig. 2). Clinical signs of brain death persisted without surgical intervention, and she died 10 days later.

Autopsy revealed sphenoid sinusitis and osteomyelitis of the sphenoid bone. The major organs were grossly and histologically normal except for pulmonary edema due to prolonged brain death. A large clot was present in the left frontal base. However, the aneurysm was obscured because the cerebrum was diffusely softened due to autolysis. The left internal carotid artery just proximal to the aneurysm showed destructive tissue change. Histological examination of the artery revealed infiltration of polymorphonuclear leukocytes in the adventitia and loss of continuity of the media with marked intimal proliferation (Fig. 3). Evidence of residual meningitis with venous sinus thrombosis was present over the length of the subarachnoid space. We could not find any causative organisms.

Discussion

Inflammatory intracranial aneurysms are often unsuspected until a devastating hemorrhage occurs. Serial angiography in 30 patients has shown that the aneurysm resolved completely in 13 patients and decreased in size in five patients. However, enlargement of the original aneurysm or appearance of a new aneurysm occurred in 12 patients. Therefore, inflammatory aneurysms have an unpredictable clinical course.

Aneurysms may develop by 7 days after a septic embolus to the brain in experimental subjects treated with antibiotics and as early as 3 days following septic embolization in subjects not receiving antibiotic therapy. There is a 10-day mean delay from the onset of warning signs or symptoms (sudden and severe headache, focal neurological deficits, or seizures in patients with bacterial endocarditis) to the development of a catastrophic hemorrhage or the angiographic demonstration of a cerebral aneurysm. It is important to emphasize that although intracranial bacterial aneurysms do resolve or decrease in size in some patients, the mortality associated with antibiotic therapy alone is still high. Possibly as many as half of all bacterial inflammatory aneurysms do not resolve with antibiotic therapy alone. In our patient, clinical improvement occurred with effective antibiotic therapy. However, the medical therapy could not prevent the process of aneurysm formation and subsequent rupture.

The precise mechanism of inflammatory aneurysm formation is not known. Extra-arterial infections, which cause bacterial aneurysms, include meningitis and cavernous sinus thrombophlebitis. The histopathology of inflammatory intracranial aneurysms includes early changes in the adventitial layer and the media of the arterial wall. Destructive change in the media is followed by aneurysm formation due to hemodynamic stress.

Carotid ligation is indicated when the patient can tolerate a carotid occlusion test. Giant carotid artery aneurysms in the cavernous portion are hard to access surgically, but can be approached by intravascular surgery. Diagnosis of bacterial inflammatory aneurysms is essential in the nonruptured stage. In our patient, we could not offer intravascular surgery in the acute stage of subarachnoid hemorrhage because of the high risk of ischemic complications. We conclude that carotid occlusion is the best therapeutic modality if there are features indicative of potentially dangerous complications.

References

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