Cerebral Hemorrhage from a Mycotic Aneurysm Developing During Appropriate Antibiotic Therapy

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SUMMARY A patient with bacterial endocarditis had headaches, cerebrospinal fluid pleocytosis and normal cerebral angiograms. Fifteen days later, while on appropriate antibiotic therapy, he developed an intracerebral hematoma due to a mycotic aneurysm. Mycotic aneurysm is an infrequent but serious complication of bacterial endocarditis. An aneurysm should be considered whenever a patient with bacterial endocarditis has neurologic symptoms even when the patient is receiving antibiotics.

CEREBRAL MYCOTIC ANEURYSM occurs in 5-10% of patients with bacterial endocarditis. 1-4 Like the other neurological complications of this disease, it may be the presenting feature of the illness. 1, 4 Frequently, the patient has no neurologic symptoms until the aneurysm ruptures. We report a patient in whom a mycotic aneurysm developed late in the course of subacute bacterial endocarditis despite appropriate antibiotic therapy.

A 41-year-old man was admitted because of a 3 month history of fever, malaise, arthralgias and skin rash. He had a rectal temperature of 38.5°C, a systolic murmur, and splenomegaly. Cefazolin sodium and gentamicin sulfate were administered to treat the presumed bacterial endocarditis. Blood cultures subsequently grew gram-positive cocci, later identified as having characteristics of both streptococcus viridans and enterococcus. The cefazolin was discontinued and penicillin, 12 million units per day, was begun on the 3rd hospital day. A neurology consultant saw the patient because of a mild, diffuse headache. Neurological examination results were normal. Lumbar puncture on the 12th hospital day yielded clear, colorless cerebrospinal fluid (CSF) under a pressure of 220 mm CSF. The CSF contained 156 red blood cells (RBC), 143 white blood cells (WBC), 84% mononuclears. The protein was 39 mg % and glucose, 36 mg %. Serum glucose was 107 mg %. Right and left carotid and left vertebral angiograms were normal on the 16th hospital day (fig. 1). A repeat lumbar puncture, 2 days after the angiogram, revealed an opening pressure of 120 mm CSF, 37 RBC, 93% mononuclear cells, a protein of 92 mg %, and a glucose of 42 mg %. Serum glucose was 119 mg %. Stains and cultures from both spinal fluid specimens were negative.

The headache subsided within a week. The organism originally isolated was found to be sensitive to penicillin, and gentamicin was discontinued. Numerous repeat blood cultures were negative.

On the 31st day of antibiotic therapy, the patient suddenly developed a severe right-sided headache, depressed consciousness and left hemiparesis. A computerized tomographic scan and right carotid angiogram showed an intracerebral hematoma secondary to a ruptured mycotic aneurysm (fig. 2). Review of the initial angiogram showed no disease in the area of the subsequent aneurysm. The hematoma was surgically evacuated and the aneurysm resected after clipping the dilated vessel. Postoperative angiography showed no residual aneurysm, and the remainder of the intracerebral vessels were normal. The patient was eventually transferred to a rehabilitation hospital. The severe left hemiparesis persisted.

Discussion

Rupture of a mycotic aneurysm is a life-threatening complication of bacterial endocarditis. 1-9 The aneurysm frequently presents by causing an intracerebral hemorrhage, commonly early in the course. Hemorrhage can occur later and may not happen until months after diagnosis and treatment of the endocarditis. 8 When this occurs, it has been assumed that the aneurysm formed before therapy or early in its course, and became symptomatic when it ruptured.

Antibiotics have improved the prognosis of patients with bacterial endocarditis. However, the incidence of neurological complications has not been significantly reduced. 1-7 The development of a cerebral mycotic aneurysm is presumed to begin with a septic embolus that lodges in a cerebral artery. Then local infection weakens the arterial wall and an aneurysm develops. 8 Experimentally, mycotic aneurysms have been produced in dogs by embolization of cerebral vessels using bacteria-coated particles. 8 Without treatment those aneurysms developed and ruptured within four days of the embolus. Treatment with an antibiotic to which the organism was sensitive did not prevent development of an aneurysm but prevented early rupture.

The aneurysm in our patient developed between the 16th and 31st days of antibiotic therapy. The early headache and spinal fluid abnormalities indicated an active central nervous system process but no aneurysm was found on the arteriogram. We presume the patient had a clinically silent septic embolus to a branch of the right middle cerebral artery some time after the first angiogram. A repeat study probably would have shown the aneurysm and would have prompted consideration of surgery to prevent rupture of the
vessel. Repeat angiography was not done because the patient's neurological symptoms abated and the general symptoms and signs of endocarditis improved. The antibiotic therapy did not prevent the formation of the aneurysm but may have retarded its development and rupture.

This case emphasizes that physicians should consider a mycotic aneurysm in a patient with bacterial endocarditis, even when the patient is on antibiotics and has minimal neurologic symptoms. It also raises the issue of how vigorously the diagnosis of mycotic aneurysm should be pursued, especially in the relatively asymptomatic patient.

References
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