Case Reports

Giant Basilar Aneurysm in the Course of Subacute Bacterial Endocarditis

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We describe a man aged 42 years with mitral valve regurgitation who suffered from subacute bacterial endocarditis caused by *Streptococcus morbillorum*. The clinical picture began with a toxic syndrome. Five months later, the patient had an embolic episode and a right rostral pontine stroke, which was followed a few days later by an adversive focal seizure on the right. Despite antibiotic treatment, he suffered complete third nerve palsy. Arteriography, magnetic resonance imaging, and computed tomography of the brain showed a giant aneurysm in the rostral end of the basilar artery; the aneurysm was clipped. We discuss the clinical features, radiology, and characteristics of this aneurysm as a unique case of a giant bacterial aneurysm in the vertebrobasilar system. *(Stroke 1990;21:1625–1627)*

During the course of infective endocarditis, in 20–40% of cases1,2 compromise of the nervous system occurs, causing stroke, abscess, mycotic aneurysm, or subarachnoid or intracerebral hemorrhage. These forms are derived from emboli produced by endocardial vegetations and are the main factors associated with increased mortality.1 Embolism is the most frequent and important neurologic complication1-4 and a late manifestation of subacute bacterial endocarditis.2 Embolism usually occurs in the middle cerebral artery or its distal branches2-5; brain stem localization is rare. Mycotic aneurysm is the next most frequent complication of endocarditis, appearing mainly in the distal branches of the middle cerebral artery.

**Case Report**

Our patient was a 45-year-old man with a medical history of moderate mitral regurgitation. He was a heavy drinker and smoker (40 cigarettes/day) and had gout. Five months before admission, he began a toxic syndrome with joint pain, asthenia, anorexia, weight loss of 8–10 kg, and anemia. He came to our hospital with an acute picture consisting of dizziness, vomiting, instability, pulsatile left oculofrontal headache, left gaze diplopia, left hemiparesis, and left hemifacial paresthesias. He had a normal arterial blood pressure, a normal body temperature, and a systolic murmur at the cardiac apex and the left sternal border that radiated into the axilla and the base of the heart. The neurologic examination showed a dysarthric patient with a right horizontal gaze paresis and a right internuclear ophthalmoplegia that led to the one-and-a-half syndrome,6 a vertical nystagmus appearing during upward gaze, a left hemiparesis (facial 3/5, arm 3–4/5, and leg 4/5) with the plantar reflexes in flexion, a left cerebellar deficit, and left arm and face sensory loss.

An initial nonenhanced computed tomogram (CT scan) of the brain was normal (Figure 1). The results of urine and blood analyses were normal except for an erythrocyte sedimentation rate of 113 mm/hr; hematocrit was 31%, hemoglobin was 10.2 g/dl, leukocyte count was 11,290/mm3, platelet count was 431,000/mm3, and fibrinogen concentration was 405 mg/100 ml. A chest roentgenograph showed cardiomegaly due to enlargement of the left chambers. The electrocardiogram was normal with a QRS axis of –30°.

On the sixth day of hospitalization, the patient began to have a severe occipital headache and later developed a focal seizure on turning his head and eyes to the right, followed by a secondary generalized tonic-clonic seizure.

His leukocyte and platelet counts rose to 18,910/mm3 and 591,000/mm3, respectively, and *Streptococcus morbillorum* grew in six blood cultures. An echocardiogram demonstrated the presence of vegetations in the mitral valve with severe mitral regurgitation, minimal tricuspid regurgitation, and enlargement of the left ventricle with preserved cardiac output (Figure 2). Syphilis, *Brucella*, *Toxoplasma*, and human immunodeficiency virus serologies were negative. Hepatitis B surface antigen assay was positive, and anti-hepatitis B surface antigen assay was negative.
Beginning on the 15th day of hospitalization, the patient was treated with penicillin G (24 million units i.v./day in divided doses at 4-hour intervals) for a month and streptomycin (2 g i.m. every 12 hours) for 2 weeks. Six days after the onset of antibiotic treatment, he had the sudden onset of complete paralysis of the third cranial nerve with ptosis, nonreactive mydriasis, and paresis of the superior, inferior, and internal rectus and inferior oblique muscles. Magnetic resonance imaging of the brain revealed an aneurysm approximately 2 cm in diameter in the midbrain, with ischemic areas in the pontine region. Brain arteriography confirmed an aneurysm approximately 2 cm in diameter, located in the distal basilar trunk. A CT scan also revealed the giant basilar
aneurysm as having a transverse diameter of 2.5 cm and an anteroposterior diameter of 2.0 cm (Figure 3). The patient subsequently had surgery, during which the arachnoid around the aneurysm was found to be pathological, friable, and whitish. The aneurysm was successfully clipped.

Nine days after surgery, a control angiogram showed slow circulation in the posterior fossa and perfusion of the posterior cerebral artery by the internal carotid artery. The patient's progress was favorable, but residual deficits consisted of emotional lability, left third cranial nerve palsy, incomplete right ptosis, right internuclear ophthalmoplegy, ataxic gait, left hemiparesis (4/5), hypoesthesia, and the cerebellar syndrome.

Discussion

Our patient's clinical picture began with a toxic syndrome, followed 5 months later by a stroke in the right superior medial pontine area (ventral and tegmental). The pathogenetic mechanism was a basilar artery embolism during the course of subacute bacterial endocarditis with occlusion of both paramedian and long circumferential branches. In addition to a stroke, embolism can produce focal seizures,1 as seen in our patient. The vertebrobasilar location of such embolisms is rare, appearing in only 10.52% of 38 cases of one series.1 In 90% of cases, embolism occurs in the middle cerebral artery or its distal branches,2,2 and the process rarely affects large arteries.3

Although our patient was under antibiotic treatment, he suffered complete paralysis of the left third cranial nerve due to compression of its peripheral trunk by a giant aneurysm arising from the distal basilar artery. This lesion appeared 21 days after the pontine stroke, strongly suggesting that the aneurysm was secondary to a previous septic embolism of cardiac origin, although the possibility of a superinfection and growth of a preexistent, small aneurysm not visible on the admission CT scan cannot be ruled out.7

Mycotic aneurysm appears clinically in 2% of all cases of bacterial endocarditis and in 5–10% at autopsy.2 Experimentally, mycotic aneurysm has been noted 24–48 hours after inoculation of a microorganism.8 The middle cerebral artery is four times more frequently involved than either the anterior or the posterior cerebral artery, and there are only three reports of mycotic aneurysm in the vertebrobasilar system.9–11

Our patient is a unique case of giant mycotic aneurysm in the vertebrobasilar system. An intracranial aneurysm >2.5 cm in diameter is defined as giant,12 although it could appear to be 1 cm in diameter on arteriography or CT.12,13 Giant intracranial aneurysms are rare since they represent only 5% of all intracranial aneurysms,14,15 and 40% are located in the vertebrobasilar system,11,16 most frequently the site of branching of the basilar artery.15 Other cases of aneurysms located in the branching and distal basilar artery presented with paralysis of the third cranial nerve,17–19 which is close to the site of basilar branching.18

Mycotic aneurysms can remit after correct antibiotic treatment, but our patient was submitted to surgery because of the persistence of the aneurysm despite antibiotic treatment and because compression was causing headache and paralysis of a cranial nerve.5

We believe that despite pathologic confirmation, our patient had a giant bacterial aneurysm in the vertebrobasilar system that appeared during the course of subacute bacterial endocarditis and that was probably secondary to cardiogenic embolism.

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References


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