Mycotic Aneurysm Caused by *Acinetobacter* in Infective Endocarditis - Case Report

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Introduction

The mycotic aneurysm (MA), also known as pseudoaneurysm is a rare inflammatory neurovascular injury, accounting for 0.7%-6.5% of all intracranial aneurysms. In initial studies, mortality was 60% to 90% due to its high potential to rupture, resulting in subarachnoid and intracerebral hemorrhage, and 12% to 32% in a review of recent literature. Clinical history and pathological findings are peculiar and varies considerably among the cases described in the literature [1]. Treatment of mycotic aneurysm is divided into drugs, surgery or endovascular approach. We present the first case described in the literature to secondary intracranial AM Infective Endocarditis (IE) by *Acinetobacter baumannii* from initial clinical onset, investigation and treatment.

Case Report

Man, 33 years old, had had a fever for one month (39°), nausea, vomiting, diarrhea, headache, and weight loss of 10 kg. One day of his admission to the University General Hospital he developed sudden worsening of headache, aphasia, right hemiparesis, seizures, neck stiffness and Hunt Hess IV.

Computed tomography (CT) and Magnetic Resonance Imaging (MRI) showed subarachnoid hemorrhage and ischemia in the left temporo-parietal region (Figure 1 A and B).

Upon the admission cerebral angiography revealed disruption of PM in the M4 segment of the left middle cerebral artery and embolization of same was made (Figure 2 A, B, C and D).

On clinical suspicion of IE to cerebral angiography Transthoracic echocardiography (TTE) was performed, which revealed tricuspidized aortic valve and with vegetation in his ventricular, mobile face, measuring 1.49 x 0.52 cm, and moderate valvar insufficiency (Figure 3 A and B) and normal dimensions of the heart cavities.

Keywords

Aneurisma Infectado; Acinetobacter; Endocardite.

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Manuscript received on May, 13th, 2013; accepted August, 11th, 2013.

Blood cultures were positive for *Acinetobacter baumannii* in more than two blood cultures with time interval of 12 hours, CBC with leukocytosis. Negative serology for human immunodeficiency virus. No history of previous hospitalizations or use of immunosuppressive drugs.

Initially, he was treated with oxacillin and amikacin without clinical improvement. After blood culture results, imipenem was set for 45 days, and he had remission of fever soon in the first 48 hours with significant improvement of symptoms and maintenance of mild reduction of motor strength in the upper right arm. Blood cultures showed no bacterial growth after a week of appropriate antibiotic therapy. After 18 days of treatment, vegetation showed a reduction to ETT (0.50 x 0.39 cm) (Figure 2C) and without changing the size of the cavities. Evolved after six months with complete remission of symptoms without motor deficit. Currently presents aortic valve thickening with moderate impairment, and normal cavities without valve replacement indication.

Discussion

Endocarditis by Acinetobacter is rare in both native valves as prosthetic valves, being typically characterized by acute onset and aggressive course. Mortality is higher in native valves compared to prosthetic valves, perhaps by the low index of suspicion and delayed initiation of treatment.[2].

The diagnosis of infective endocarditis, based on clinical history and the Duke modified classification, included two major criteria: presence of blood culture positive for *Acinetobacter baumannii*, present in two blood cultures, with an interval of 12 hours, and clear vegetation to Transthoracic echocardiography. The minor criteria were mycotic aneurysm evidenced by cerebral angiography and fever.

Echocardiography is essential for diagnosis, monitoring and important tool in making therapeutic decisions in patients with infective endocarditis. TTE can be useful in demonstrating intracardiac masses or vegetations, paravalvar abscesses and new valve insufficiencies, besides demonstrating lesions that lead to infective endocarditis and rheumatic valve disease, bicuspid aortic valves, congenital disorders and other conditions.

Echocardiography should be performed in all patients with moderate or high suspicion of infective endocarditis.

The Transesophageal Echocardiography (TEE) has better spatial resolution and is more sensitive in detecting EI

DOI: 10.5935/2318-8219.20140005

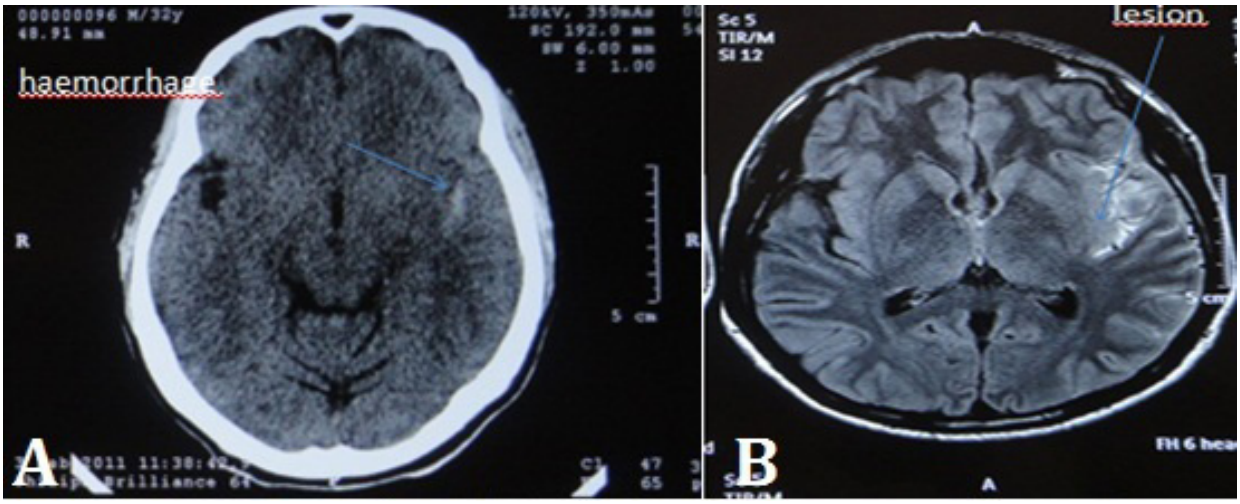

Figure 1 - *A - Computed tomography: of the brain parenchymal temporal hemorrhage . B - Cranial magnetic resonance imaging: hemorrhage with perilesional edema in the left temporal lobe.*

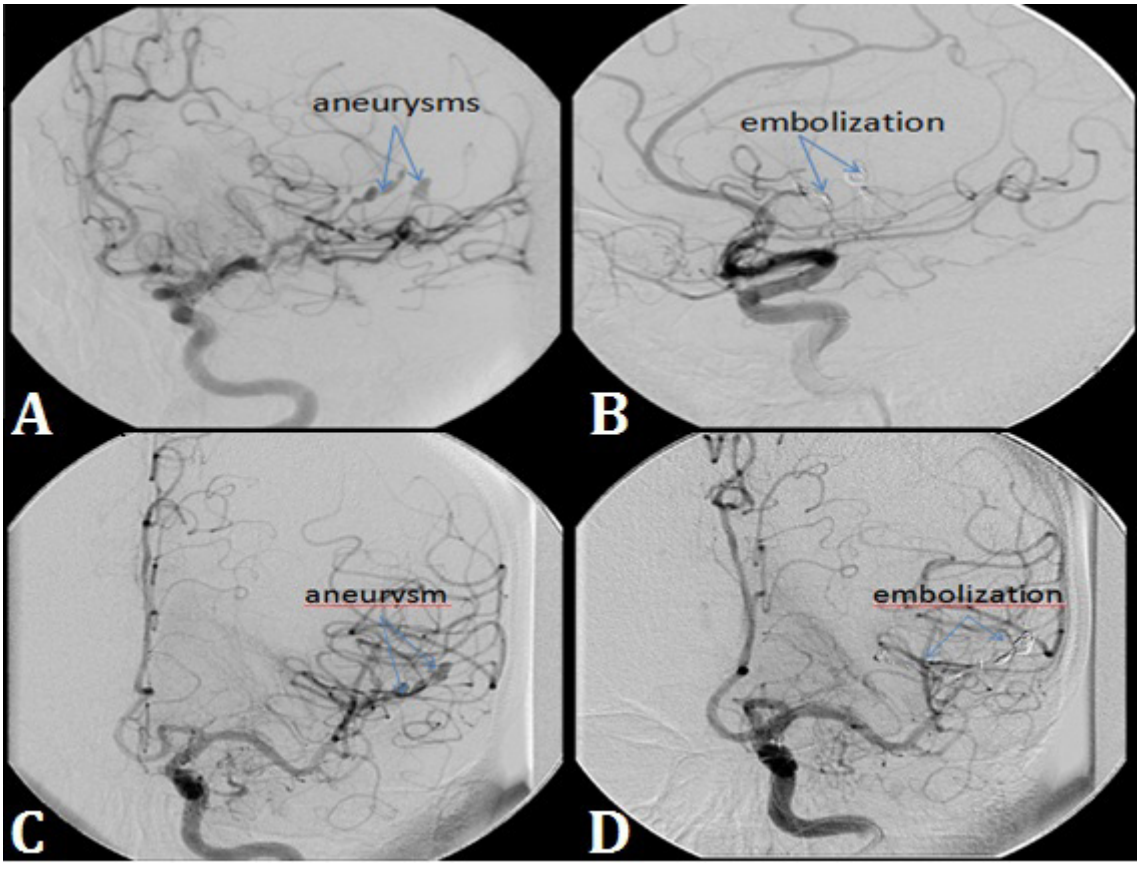

Figure 2 - *A - Cerebral angiography in profile pre-embolization cerebral aneurysm. B - Cerebral angiography in profile after embolization aneurysm C - Cerebral angiography in anteroposterior view: pre-embolization cerebral aneurysm. D - Cerebral angiography in anteroposterior view: post-embolization cerebral aneurysm.*

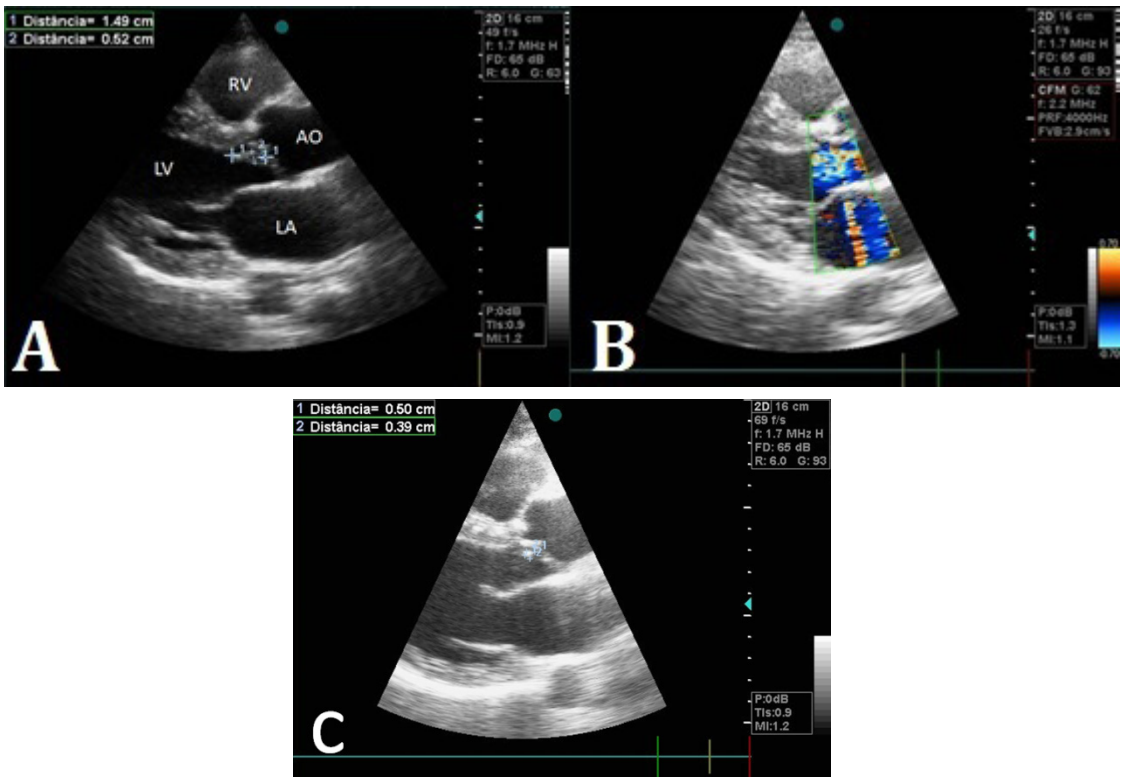

Figure 3 - *A - Transthoracic echocardiography: parasternal view of vegetation in the aortic valve. B - color Doppler: Aortic Insufficiency C-Transthoracic echocardiography: parasternal view of aortic valve vegetations 18 days after initiation of antibiotic therapy.*

compared with Transthoracic echocardiography. Reynolds et al. showed a sensitivity of only 55% to TTE when compared to TEE in detecting vegetations in 50 patients with 51 vegetations in native valves with proven valvar infectious endocarditis. The TTE sensitivity in detecting aortic valve vegetations was 50% (12/24); mitral 62% (16/26), and for tricuspid valve 0% (0/1). TTE failed detecting large vegetations and five of these measured more than 10 mm, when there is more risk of embolic phenomena. The sensitivity was 0% in the detection of vegetations <5 mm, 50% from 5 to 10 mm, and 84% for vegetations> 10 mm[3].

Despite the high sensitivity of transesophageal echocardiography, it should initially be employed in cases of limited Transthoracic window (e.g.,: obese, chest deformity, mechanical ventilation) in valvular prostheses in previous valvular abnormality (including previous endocarditis), staphylococcal bacteremia, bacteremia caused by common bacteria such as *Streptococcus viridans* endocarditis. Transesophageal echocardiography should be performed in cases of clinical suspicion of IE and not viewing the TTE[4] vegetations. Even with TTE and TEE, false negative results can be produced in the case of vegetations that have already been embolized and in the small vegetations. In our case, it was not necessary to perform transesophageal echocardiography because of easy demonstration of valvular vegetation and excellent patient outcomes through serial TTE.

Neurological complications can occur in 20% to 40% of patients with IE[5]. The AM represents a small and dangerous subgroup of these complications, result of the lodging of the septic embolus in a vessel consequent to the friability of cardiac vegetations in the effectiveness of IE. The emboli may occlude the vessel, causing or promoting myocardial infection of the arterial wall. The theory of the *vasa vasorum* is the most widely accepted as a pathological mechanism of formation of MA. Its basis lies in the fact that emboli causes infection and inflammation of the vessel adventitia, damaging its structure, and in the consequent formation of the aneurysm lumen high internal pressure against the damaged arterial wall. Intracranial MA tend to occur in the more distal portions of the middle cerebral artery involving the second and third branches near the brain surface. This pattern helps to distinguish them clinically from saccular aneurysms occurring most commonly at the base of the brain and Willis Circle[6].

The most common etiologic agent is *Staphylococcus aureus*, accounting for approximately 50% of cases[7]. Jarret F. et al. have shown that Gram negative organisms are implied in 35% of MA cases and are associated with the highest incidence of rupture (84% versus 10%) and mortality (84% versus 50%), when compared to Gram positive organisms.[8]

Clinical manifestations of intracerebral MA are peculiar to the affected area, which may present initially with

headache, fever, vomiting, eye paralysis, convulsions, behavioral changes, hemiparesis, drowsiness and loss of consciousness.

Several imaging studies can be used to identify intracerebral MA, such as CT not contrasted of the skull, which can provide important information. This technique has a sensitivity of 90% to 95% in the identification of intracerebral hemorrhage and can indirectly identify the location of MA. Magnetic resonance angiography with or without contrast and contrast angiography may be used and have a sensitivity of 90% to 95% in detecting intracerebrais[9] MA. Both have limitations in the detection of aneurysms smaller than 5 mm, and contrast angiotomography presents risk of kidney insufficiency.[10]

Hess et al [11], in the evaluation of 101 patients with IE and proven high probability, asymptomatic, found abnormalities in 78 patients (71.5%) under evaluation by brain MRI. The most frequent changes were acute ischemic lesions (40 patients, 37%) and cerebral microbleeds (62 patients, 57%). Eight patients had subarachnoid hemorrhages, three patients with microabscesses, three patients with small cortical hemorrhages, and three had mycotic aneurysm.[11]

With regard to cerebral aneurysms, angiography is the gold standard method, confirming the exact location of the aneurysm, detecting extravasation and arterial occlusions, and assisting in surgical planning.

Because of the rarity of intracranial MA, there are no randomized studies to guide treatment and management, these being restricted to clinical experience and case series. The treatment is based on prolonged antibiotic therapy, endovascular or surgical treatment. Medical intervention uniformly accepted is antibiotics for at least six weeks. The initial choice of antibiotic should be based on the most commonly implicated organism in clinical circumstances of the patient, and subsequently replaced based on the antibiogram of a positive blood culture. The efficacy of treatment should be based on clinical and laboratorial improvement. The MA not ruptured may experience spontaneous thrombosis and can only be resolved completely with antibiotics treatment [4]

Peters et al [13] in a review of intracranial MA secondary to endocarditis showed that the most relevant factor in therapeutic considerations of cerebral aneurysm refers to the fact that it is intact or broken. The mortality is higher in cases where there is rupture as compared to non-disrupted ones (24% vs. 9%). Among the patients with aneurysm rupture, mortality was higher in those treated with antibiotics compared to those treated with antibiotic therapy and surgery (49% *versus* 12%) [12]. For these reasons, intact aneurysms can be treated with isolated antibiotycotherapy, but, whenever possible, broken aneurysms should be conducted with antibiotycotherapy combined to surgery or endovascular treatment[13]

Endovascular treatment has developed rapidly, and because of the ease of access to distal aneurysms, provides less cerebral ischemic damage when compared to proximal aneurismal lesions. Dohme et al. report that in treating 13 patients with endocarditis associated with rheumatic valvar disease and broken mycotic aneurysm, all were treated successfully and presented only two deaths due to mass caused by intracerebral hemorrhage occurred before the procedure. There were no complications during the procedure, and eight patients survived without any neurological sequel. The surgical treatment of intracerebral MA may be more harmful when compared to drug treatment alone. The loss of surgical treatment can be explained by the more distal location and depth of MA. The location of these aneurysms is difficult because of the hematoma, and bleeding dissection can cause rebleeding due to fragility of the aneurysm. Surgical treatment may be an option after patient stabilization and evacuation of intercerebral hematomas[14].

The *Acinetobacter baumannii* is a Gram-negative anaerobic bacterium that has been implicated in infections in critically ill patients with impaired immune response, in colonization and infection of major surgeries, major trauma, major burns, premature, previously hospitalized patients, especially in unit of intensive care, mechanical ventilation, invasive procedures and previous antimicrobial treatment.

Acinetobacter sp. has been responsible for infections acquired in the community as infections of the urinary tract, medium otitis, meningitis and endocarditis. These cases may evolve in a striking manner, with high rates of bacteremia and mortality from 40% to 64%[15].

Unfortunately, the clinical impact of *A. baumannii* is related to its growing resistance to antimicrobial, being reason of large concern, and that which led it to be called as the negative Gram bacteria *"Staphylococcus aureus meticilina resistant"* (MRSA).

The neurological clinical picture associated with aneurysms of the distal branches of the middle cerebral artery with ruptured cerebral angiography enhances the possibility of mycotic aneurysm secondary to infective endocarditis proven by vegetation detected by echocardiography and reinforced by blood culture Endovascular treatment associated with specific antibiotic therapy for Acinetobacter was essential for good clinical outcome of the patient.

The clinical case is important because there are no cases reported in the medical literature of secondary MA to EI by *Acinetobacter baumani*. It should be highlighted the good progress of the patient, despite being a rare disease caused by Gram-negative bacteria, besides the severity and high mortality. Early diagnosis and clinical suspicion are important for a favorable outcome.

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