An Uncommon Case of Streptococcus Pyogenes Endocarditis Causing Intra-Cranial Mycotic Aneurysms

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Abstract
Intra-Cranial Mycotic Aneurysms [ICMA] or infection aneurysms are rare and represent less than 10% of the neurological complication of infective endocarditis. The most common causative organisms are alpha-hemolytic streptococcus of the viridans group and staphylococcus aureus, respectively responsible for 50% and 10% of ICMA.

We report a case of group A beta-hemolytic streptococcus – streptococcus pyogenes- native mitral valve endocarditis complicated with intracranial mycotic aneurysm. A review of the existing literature revealed that acute IE caused by streptococcus pyogenes has rarely been reported, only 40 cases since 1940, of which no case was complicated with intracranial mycotic aneurysm.

Keywords: Infective endocarditis; Streptococcus pyogenes; Intracranial mycotic aneurysm

Introduction
Infective endocarditis is an uncommon infectious disease with an annual incidence that varies from 3 to 7 per 100 000 person-years. Although relatively rare, IE continues to be characterized by increased morbidity and mortality through its complications [1]. Neurologic complications related to IE are diverse (ischemic, hemorrhagic and infective), and are the most frequent extracardiac complications, occurring in 17 to 82% of patients with left-sided IE [2]. Mycotic aneurysm are rare inflammatory neurovascular lesions, comprising 0.7 – 5.4% of all intracranial aneurysms. S.viridans and S.aureus are the most common organisms that cause IE. Therefore, they are the two most frequently associated pathogens with CMAs in the course of IE[3]. Streptococcus pyogenes, whilst known to infect immunocompromised patients, is a rare cause of endocarditis and there are no published reports of this organism causing intracerebral mycotic aneurysms. We report a case of streptococcus pyogenes endocarditis complicated within intracranial haemorrhage in the setting of cerebral mycoticaneurysm.

Case Presentation
An 18-year-old female was admitted with a one-month history of persistent fever, associated with shortness of breath and precordialgia. She had previously received some unspecified treatment with no apparent amelioration of symptomatology. She reported a history of dental procedure, undergone one year earlier. Physical examination, showed a temperature 38.5°C, (Blood pressure: 120/70, Heart rate= 100 beats/minute, respiratory rate=20 breaths per minute). Cardiovascular examination revealed an intensive mitral holosystolic heart murmur. Osler nodes were observed on fingers andtoes. Biological test revealed anaemia of chronic inflammation, blood cell count= 10500/μL, platelet count= 360000/μL, prothrombin time-international normalized ratio=1.23, C- reactive protein=98mg/dL, creatinine=6mg/L and estimated glomerular filtration rate of 138.5 mL/mn. The urine analysis was negative for infection. Electrocardiogram demonstrated sinus tachycardia, and Chest x-ray demonstrated cardiomegaly with features of left atrial enlargement.

Blood cultures were found to be positive for streptococcus group A (Streptococcus pyogenes). A transthoracic echocardiogram, along with a Trans oesophageal echocardiography, documented a severe mitral regurgitation and a 16×6mm vegetation, attached to the anterior mitral leaflet and 8 mm vegetation implanted on the tricuspid valve was also noted (Figure 1and 2) Pan-computed tomography (CT) scans revealed features of splenic infarction, with no other sign of secondary lesions elsewhere. She met two major and three minor modified Duke Criteria and was diagnosed with definite endocarditis. The neurological examination was steadily normal, and ECG was undergone daily, during antibiotic therapy. A Trans oesophageal echocardiography was repeated 9thday of
antibiotic therapy, it showed three calcified masses implanted on the two leaflets of the mitral valve, with the following dimensions (14mm, 13mm, 6mm), and suspected a perforation of the anterior mitral leaflet. A decision of cardiac surgery was taken.

16th day of antibiotic therapy, she reported headache and drowsiness. Cranial CT scans showed an intracerebral hemorrhage located in the left frontal and insular lobes with manifest signs of cerebral herniation (Figure 3).

Discussion

Beta haemolytic streptococci, which include serogroups A, B, C and G, cause a wide variety of infections including cellulitis, necrotising fasciitis, bacteremia and infective endocarditis [4]. Although BHS endocarditis is relatively uncommon, it is very aggressive, with a high rate of cardiac valve destruction, cardiac abscess formation and systemic embolization [4]. Of the different subgroups, group B streptococcus (S. agalactiae) is the most common cause of BHS endocarditis. BHS Endocarditis due to groups C and G are less common, but presents similarly to that caused by group B BHS [4]. It is noted that Group A β-hemolytic Streptococcus pyogenes is the least common cause of BHS endocarditis [4]. Forty cases of endocarditis caused by Streptococcus pyogenes in children and adults have been reported since 1940, with a median age of 32 years, ages ranging from four months to 80 years. The mortality rate was 24% (62.5% before 1990 and 15.4% after 1990) and was mainly due to cardiac failure and/or septic shock; most patients recovered after antibiotic therapy [50]. In our case, the outcome was dramatic, due to a neurologic complication, namely, intracranial hemorrhage. Intracranial Mycotic aneurysm, which results from the septic embolism of vegetation in the cerebral circulation, has been reported secondary to infective endocarditis, with an estimated range between 2% and 10%. The most common causative organisms being Streptococcus viridans, Streptococcus group D and Staphylococcus aureus species [6]. The clinical presentation of unruptured ICMAs is variable and lacks specificity: Fever, headache, convulsions and focal deficit. Ruptured ICMAs are responsible of a clinical profile of cerebral or subarachnoid hemorrhage: headache, loss of consciousness, intracranial hypertension and focal deficit [7]. The diagnosis of ICMAs is possible on brain CT scan that visualises the indirect signs. Cerebral angiography, cerebral MRI and/or cerebral angiography remains the benchmark examination, in particular for the diagnosis of small ICMA [7]. In our case, cerebral angiography gave evidence of two aneurysms. Mortality associated with rupture of intracranial mycotic aneurysm, leading to subarachnoid hemorrhage [5].
Haemorrhage or intracerebral haemorrhage, is reported to be as high as 80%[6]. There are, however, no published reports of S. Pyogenes causing intracerebral mycotic aneurysms. Treatment of intracranial mycotic aneurysms is challenging and to date, there are no standard guidelines. Antibiotic therapy is an essential component of the treatment and studies have reported variable rates of resolution in response to antimicrobial therapy [6]. Taking into consideration the variable response to antibiotics and the high rate of mortality associated with rupture of intracranial mycotic aneurysms, some authors have advocated more aggressive treatment, using endovascular or open surgical treatment, to permit securing of aneurysms and timely cardiac valve replacement [6]. Furthermore, timing of cardiac surgery is difficult to determine, owing to the cerebral damage that may be amplified by heparinization and hypotension during the cardiopulmonary bypass or postoperative anticoagulant therapy. There are currently no guidelines in the treatment of Intracranial mycotic aneurysms in the setting of IE, rendering its management extremely challenging.

Conclusion

Group A β-hemolytic Streptococcus pyogenes IE is a rare condition, however, it can be lethal through extra cardiac complications, which is approved through our case which confirms its virulent character. There are currently no guidelines in treatment of Intracranial mycotic aneurysms in the setting of IE, rendering its management extremely challenging.

References
