CASE REPORT

Infective Endocarditis Caused by *Streptococcus agalactiae* in a Native Aortic Valve, Complicated by Meningitis and Cerebral Embolism

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Abstract

We present a case of infective endocarditis associated with community-acquired *Streptococcus agalactiae* in an immune competent patient. The endocarditis affected the native aortic valve with perforation of the coronary cusp and was complicated by a cerebral embolism. The use of intravenous ampicillin produced a satisfactory clinical and echocardiographic recovery despite not receiving a valve replacement. In addition to reporting an extremely rare case, this paper confirms that the opportune identification of endocarditis caused by *S. agalactiae* and the selection of appropriate antibiotics can prevent the necessity of cardiac surgery, usually required in such cases.

Key words: Group B *Streptococcus*, cardiac infection, bacterial infection

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Introduction

*Streptococcus agalactiae* or group B *Streptococcus* (GBS) are facultative gram-positive diplococcus originally isolated in cows with bovine mastitis. In pregnant women it may cause bacteremia, chorioamnionitis, septic abortions, urosepsis and cellulites. In newborns it is associated with neonatal sepsis and meningitis (1). The prevalence of GBS infections varies from one country to another. In Mexico it is estimated that up to 90% of women of childbearing age have had contact with this bacteria (2). Many people are asymptomatic carriers; however the incidence of clinical infection is very low. Clinical GBS infections in adults in many cases are associated with contamination in hospitals or maternal and infant care units. Among the infections associated with GBS, infective endocarditis represents a very rare, yet extremely important phenomenon due to it’s high mortality rate if it does not receive aggressive and opportune treatment, usually including valve replacement surgery (3). It has been reported that even with surgery the mortality rates for this disease reach 50% (4).

We present the case of an individual without evidence of immune suppression, who developed native aortic valve endocarditis associated with community acquired GBS which was complicated by perforation of the left coronary cusp and a cerebral embolism. The patient showed a notable recuperation after 6 weeks of antibiotic therapy without heart surgery.

Case Report

The patient, a 39-year-old man, was a resident of the city of Colima, Mexico; no risk factors for GBS infection were reported. He was admitted to the Hospital Regional Universitario in Colima in April 2007 after three days of fever and headache followed by ataxia, vertigo, and diplopia, numbness of the right half of the face and body and confused speech. The physical examination revealed fever of 38.5°C, a heart rate of 80 per minute, breath rate was 24 per minute, and blood pressure 100/70 mmHg. The patient was somnolent, with presence of disartrhia, discreet right hemiparesis...
of the face and body with paresis of the left VI cranial nerve pair, osteotendinous hyperreflexia, horizontal nystagmus and nuchal rigidity. The chest auscultation revealed an intense mesosystolic murmur in the aortic area that radiated to the neck. The rest of the examination was practically normal.

The laboratory results reported hemoglobin of 16.3 g/dL, hematocrit 44%, leucocytes 8,800/mm³ with 88% neutrophils, and 2.6% lymphocytes, platelets 41,000/mm³. Blood glucose 78 mg/dL, serum creatinine 0.9 mg/dL, urea 29.5 mg/dL, serum Na 134 mEq/L, K 3.3 mEq/L. Liver function tests were normal as was a urine analysis. The examination of cerebrospinal fluid showed slightly cloudy liquid, with proteins 94 mg/dL, glucose 43 mg/dL, 22 cells of which 70% were polymorphonuclear cells and 30% were mononuclear cells. The Gram, Ziehl Nielsen and India ink stains were negative as well as the culture. An electrocardiogram showed signs of left ventricle hypertrophy with an ischemic area in the left ventricular free wall. The echocardiogram revealed the presence of multiple vegetations on bicuspid aortic valve, perforation of the left coronary cusp and important valvular insufficiency. The left ventricle had an ejection fraction of 64%. A cranial computed tomography revealed a hypodense area in the left temporo-parietal region that was enhanced with the administration of gadolinium suggestive of an infarction. The first of a unique series of three blood cultures, taken at an interval of 2 hours each and processed with MicroScan³, was positive for "Streptococcus agalactiae" (group B "Streptococcus") sensitive to ampicillin, penicillin, clindamycin and ceftriaxone; it was resistant to vancomycin, tetracycline and imipenem.

The initial treatment was with intravenous vancomycin for a week which was changed to intravenous ampicillin 2 g every 6 hours plus intravenous ceftriaxone 2 g every 8 hours for two weeks due to the involvement of the central nervous system, continued for four weeks with ampicillin alone to complete treatment for infective endocarditis. Aminoglycosides were avoided because of the risk of renal toxicity. After the modification there was a rapid reduction of fever and a steady improvement of the neurological deficit. An aortic valve replacement was proposed but the patient rejected on several occasions and for this reason only parenteral antibiotics were continued. After six weeks of the aforementioned treatment the patient was discharged with significant clinical improvement. Three months after his discharge the patient was seen in the outpatient consult. He arrived walking without difficulty with only discreet disarthria. There was a notable reduction in the aortic murmur with no evidence of heart failure. A new echocardiogram showed disappearance of the vegetations and the perforation of the coronary cusp with an ejection fraction of 78%. Seven months later, he was hospitalized again with signs of heart failure; however the new echocardiogram showed only a moderate reduction of ejection fraction to 60%, without evidence of vegetations. The patient has continued in observation as an outpatient, treated only with digoxin 0.25 mg/day and acenocoumarin 5 mg/day.

Discussion

This the first case of endocarditis associated with GBS reported in Latin America. At the same time, it is one of few cases of this type of endocarditis complicated with a cerebral embolism and meningitis, especially in an immune competent individual.

The disappearance of the valve lesions with only antibiotic therapy and no valve replacement is notable. This indicates that the opportune recognition of this microorganism and the adequate use of antibiotics based on the pattern of sensitivity in vitro, can dramatically improve the outcome of a usually fatal pathology (4, 5).

Ischemic cerebral lesions are a relatively common complication of bacterial endocarditis; however there are only two previous reports of this phenomenon in GBS endocarditis (6). They are due to the detachment septic emboli from the vegetations on the cardiac valve. The presence of GBS meningitis in newborns is a relatively frequent pathology; however, its presence in adults with infective endocarditis, as in this case, represents a phenomenon reported only in five previous cases in the international literature (7). The meningitis is undoubtedly caused by bacterial dissemination to the central nervous system which can explain the rapid resolution of the problem with the use of high doses of Ampicillin and Ceftriaxone, which are considered therapy first and second choice, respectively, for S. agalactiae.

Up until now all GBS strains have proven susceptible to penicillin, as in the present case (8). However, the notable increase in the incidence of serious infections by GBS in recent years increases the suspicion of the emergence of mutated strains that may be more aggressive and resistant, whereas in this case the strain was resistant to vancomycin (1, 9).

This patient had none of the predisposing risk factors traditionally considered for the acquisition of this bacteria such as diabetes mellitus, renal diseases, alcoholism, smoking, obesity, HIV infection, cancer or previous neurological problems (1, 4, 6), which might be explained by the fact that it has been reported to present in a small proportion of patients (approximately 10%) who can develop it without these risk factors (1). However, the discovery of a bicuspid aortic valve most certainly contributed to the colonization after GBS bacteremia. Taking into consideration all of the previous factors, GBS should be added to the germs considered to cause endocarditis, not just in pregnant women and newborns, but also in immune competent adults, especially in communities with a high prevalence of GBS asymptomatic carriers, as is the case in Mexico (2). In these areas the use of the anti-GBS vaccine will most certainly have important benefits.
References