Group B streptococcus endocarditis associated with multiple pulmonary septic emboli

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Abstract

Endocarditis is a rare presentation of group B streptococcal infection. Its association with pulmonary septic embolism was only barely studied and limited data is available up to date. Multiple septic emboli is a common complication of bacterial endocarditis, but only a few cases have been documented in relation to group B streptococcus. We present the case of an 87 year old female patient with multiple underlying conditions that predisposed the development of bacterial endocarditis secondary to group B streptococcus and subsequently multiple pulmonary septic emboli. The patient was treated with ceftriaxone and azithromycin with good response and complete recovery without any further complications. In the event of a diagnosed case of group B streptococcus endocarditis, there should be a low threshold for the suspicion of septic pulmonary emboli especially in cases with right valves involvement.

Introduction

Streptococcus agalactiae, also known as group B Streptococcus (GBS) is a gram positive, facultative anaerobic bacteria associated with infections in three different populations: i) Newborns: GBS is the main cause of sepsis and meningitis; ii) Pregnant woman: It is an important etiologic agent of chorioamnionitis, endometritis, septic abortion and bacteremia1 and iii) Healthy individuals: GBS rarely causes infection in this group and when it occurs is almost always associated to underlying disease like diabetes mellitus, cardiovascular disease, malignancy and immunodepression.2,4

The incidence of endocarditis secondary to GBS is still considered a rare, especially when associated with severe disease and high mortality. With the documentation of endocarditis with complication of multiple septic emboli secondary to tricuspid valve vegetations in this elderly woman, we hope to add more knowledge on this issue since limited documentation has been done on it.

Case Report

An 87 year old female with history of hypertension and heart failure was admitted to the hospital because of two weeks history of fever, chills, loss of appetite and abdominal discomfort. She denied cough, dyspnea, chest pain, palpitations or diaphoresis. Two weeks prior admission she visited her primary care physician with the same complaints and was prescribed oral cefuroxime. The patient complied with taking of the medication but had no improvement in her condition. She had a history of atrial fibrillation and hypothyroidism and received prophylactic warfarin and levothyroxine for treatment of the respective conditions. Sick sinus syndrome was diagnosed ten year prior to presentation and pacemaker was placed for control. She had no history of alcohol intake, smoking or use of intravenous drugs. She lived with 24 hours home aid. On examination she had a temperature of 104°F, blood pressure 195/64, the pulse 75 beats per minute, the respiratory rate was 19 cycles per minute and the oxygen saturation was 99% while the patient was breathing ambient air.

At general examination she was in mild respiratory distress and oriented in person, place and person. Pallor was noted on inspection of the mucous membranes and the skin and the presence of a pacemaker generator noted on the left anterior chest. Auscultation of the chest revealed an irregular rhythm and a non radiated grade II/VI holosystolic murmur best heard in mitral and tricuspid areas. Basal crackles were heard bilaterally on lung auscultation. No peripheral edema, jugular venous distention, Osler’s nodes, splinter hemorrhages or Janeway’s lesions were noticed. The rest of the physical exam was within normal limits.

A chest x ray was done showing cardiomegaly and perihilar infiltrates bilaterally (Figure 1).

An electrocardiogram revealed atrial fibrillation with 2 to 3 paced beats and without ischemic changes.

A complete blood cell count showed important leucocytosis (26.2x10⁹/L) with 83% neutrophils and normocytic anemia (hemoglobin 8.6 g/dL and MCV 85fL). Liver and basic metabolic profile within normal limits. Prothrombin time was 47 seconds and 3.8 of INR. Blood was drawn for culture and sent to microbiology laboratory.

The blood culture results was positive for Group B streptococcus, sensitive to penicillin, cephalosporins, quinolones and macrolides.

The presence of the murmur, fever and the blood culture raised the suspicion of the possibility of endocarditis. Transthoracic and trans-esophageal echocardiogram were ordered and these showed a left ventricle that was moderately dilated with severe left ventricular systolic dysfunction. The left ventricular ejection fraction was estimated to be 25%. The right ventricle appeared severely dilated and hypokinetic and a large vegetation was found in the aortic valve.

Treatment for suspected pneumonia was initiated with azithromycin and Ceftriaxone. Levothyroxine, furosemide, metoprolol and simvastatin were continued for the management of hypothyroidism, cardiac failure, hypertension and hypercholesterolemia respectively.

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tricuspid valve arising from its annulus measuring 1.2 cm in its greatest dimension.

A computerized tomograph of the chest showed the presence of multiple sizes, scattered lesions in the right lung; some with spiculations and cavitation. These lesions were mostly in the peripheral regions of the lungs, with the largest in the apex of the right lung (Figure 2, 3).

The diagnosis of acute endocarditis secondary to Group B Streptococcus associated with multiple pulmonary septic emboli was then done.

The antibiotic regimen was continued and after three days of treatment the fever started to settle and the rest of symptoms to improve.

The white blood cell count decreased to normal levels by the fourth day of hospitalization and the patient was discharge, completely asymptomatic on the seventh day of hospitalization. Oral antibiotics were prescribed to complete 4 weeks of treatment.

**Discussion**

Endocarditis is a rare, severe presentation of GBS infection. It accounts for only a minority of all cases of bacterial endocarditis (1.7%) and is mainly found in patients with history of alcoholism, cirrhosis, diabetes and cancer. In the last 45 years, more than 150 cases were reported in English and Spanish literature with only a minority (5 cases) in the last decade. It has been described in all age groups with a higher incidence in adults with risk factors and pregnant women. Most of the studies demonstrated a slightly predominance in females. The virulence of the bacteria is reflected in the high rates of mortality that vary from 20-45% as documented by studies and also valve compromise. The patients usually present with severe disease associated with early development of large vegetations on valves. The aortic and mitral valves are frequently involved, with a majority of cases reporting the involvement of the tricuspid and pulmonary valves. The infection usually progresses aggressively and is complicated with metastatic emboli formation.

More uncommon complications like endophthalmitis, pericarditis, myocardial abscess and massive pulmonary hemorrhage have been anecdotically reported in the literature.

Natural penicillins remain as the best option of treatment for GBS since no in vitro resistance or production of betalactamase has been documented to date. The addition of an aminoglycoside is still controversial and most of the effects of its use are only based in the clinical outcomes of reported cases. The use of penicillins alone appears to be effective for management but has not been proven clinically. Most of the cases reported in literature combined at least one natural penicillin or cephalosporin with an aminoglycoside as initial treatment, therefore, no protocol exist for the use of antibiotics in the rare cases of GBS endocarditis and most of the guidelines are based on the experience and outcomes of reported cases.

Septic pulmonary embolism (SPE) is an uncommon condition that generally presents with an insidious onset of fever, respiratory symptoms, and lung infiltrates. Actually, SPE is especially associated with the use of indwelling catheters, immunodeficiencies and IV drug use, pelvic thrombophilia, as well as suppurative processes.

The association of septic pulmonary emboli and endocarditis is well recognized especially when the right chambers of the heart are involved.

The diagnosis is usually made with radiologic investigations; the most helpful diagnostic procedure is the computerized tomography (CT) scan, typically showing cavitations or gas in nodular peripheral lesions regardless of the primary cause of the emboli.

This case followed the typical radiologic pattern on CT scan but the clinical signs and symptoms were barely manifested. Group B streptococcus is known for the large extent of metastatic emboli formation in relation to endocarditis, but there is limited information in the electronic literature of cases presenting with septic pulmonary emboli.

In the largest case series study published in the literature, Sambola et al., described 30 cases of GBS endocarditis in a 24 years period, reporting embolic complications in 50% of the cases but only one patient with septic pulmonary embolism. Contradictorily, another study found that GBS endocarditis with involvement of the tricuspid valve is associated with septic pulmonary embolism in approximately 86% of cases.

This association remains controversial and more case series studies are necessary to achieve conclusions. In our believe the presence of septic pulmonary embolism is common in all endocarditis with right valves involvement and apparently there is no additional factor to this specific bacteria to not predispose to this complication. In contrast the typical aggressiveness of the bacteria make the possibility of septic embolism early in the progression of the disease.

**Conclusions**

In the event of a diagnosed case of group B streptococcal endocarditis, there should be a low threshold for the suspicion of septic pulmonary emboli especially in cases with tricuspid valve involvement and should prompt the initiation of appropriate diagnostic workup for the early diagnosis of septic pulmonary embolism even in the absence of typical pulmonary symptoms.

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