ACUTE GROUP G STREPTOCOCCAL ENDOCARDITIS: 
CASE REPORT AND REVIEW OF THE LITERATURE

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Though initially considered a non-pathogenic normal flora, group G beta-hemolytic streptococci has increasingly been recognized as a cause of aggressive and frequently fatal infections. Over the past two decades, more than 40 cases of infective endocarditis caused by group G beta-hemolytic streptococci were reported in the literature. Common noticeable features reported were acute rapidly progressive and often complicated course, frequent involvement of normal valves, and lack of response of the organism to antimicrobial treatment inspite of in vitro sensitivity. We have recently encountered a case of rapidly progressive and complicated group G streptococcal endocarditis inspite of adequate early antimicrobial therapy. To the best of our knowledge, this is the first case to be reported from Saudi Arabia.

Case Report

A 32-YEAR-OLD ERITERIAN FEMALE presented with a four-day history of fever, pain, and swelling of both ankles and knees, generalized myalgia, and weakness, particularly in the limb girdle muscles. There was no history of dyspnea, skin rashes, and oral or genital ulcers. She gave a history of dysuria but not urethral or vaginal discharge. The past medical history was unremarkable, in particular for valvular and congenital heart diseases and for arthritis. Physical examination revealed an ill looking, lethargic, febrile, young female with evidence of mild bilateral conjunctivitis, mild mitral regurgitation, bilateral ankle and knee arthritis, and profound weakness and tenderness of the pelvic girdle muscles. The initial leukocyte count was 69,000 per JLL with 85% segmented neutrophil, 9% monocyte, and 6% lymphocyte.

The platelet count was 37,000 per JLL. There was mild normochromic normocytic anemia, negative direct and indirect Comb's tests, and a high titre of cold agglutinins. The Zeta erythrocyte sedimentation rate in lh was 60 mm. The C3, C4, and CH50 complements were within normal values. The rheumatoid factor, antinuclear antibodies, and anti-ds DNA were negative. Urine microscopy revealed a white count of 300/mL, red cell count of 20/mL and few bacteria, however, the urine culture revealed no growth. A two-dimensional and Doppler echocardiography revealed a large 1 x 1 cm vegetation attached to the posterior mitral valve leaflet. Transesophageal echocardiography revealed a large 1 x 1 cm vegetation attached to the anterior mitral valve leaflet, a larger 1.2 x 1.3 cm highly mobile vegetation attached to the posterior mitral valve leaflet, and mild mitral regurgitation. On the day of presentation and after collection of blood and urine for culture, the patient was empirically started on intravenous crystalline penicillin G, 4 mega units every 6 h and gentamicin, with a loading dose of 80 mg followed by 60 mg every 8 h, the dose was later adjusted to! maintain a therapeutic level. The three sets of blood cultures taken on the day of presentation
grew group G beta-hemolytic streptococci. Identification was based on biochemical characteristics and serological grouping (Streptex, Murex Diagnostics, England). The organism was sensitive to penicillin G, gentamicin, amikacin, ampicillin, and cephalosporins. Both the minimum inhibitory concentration (MIC) and minimum bactericidal concentration (MBC) were 0.01 mg/mL. Blood cultures, after 5 days of antimicrobial therapy, grew the same organism (group G beta-hemolytic streptococci) with the same sensitivity pattern. On the sixth day of hospitalization, the patient was still febrile and the chest x-ray revealed new right lower lobe alveolar infiltrate. Ceftazidime, 2 gm intravenously every 8 h, was added to the treatment. Repeated blood cultures in the 8th day of admission failed to grow any organisms after prolonged incubation, and tests for brucella, typhoid, and paratyphoid were negative. Ultrasound of the abdomen failed to show intraabdominal abscesses, and total body gallium and leukocyte-labelled scans were negative. Two days later, due to lack of response to the antimicrobial therapy (persistent fever and leukocytosis) and deterioration of the general condition of the patient, it was decided that the mitral valve to be replaced. A few hours before the surgery, the patient developed severe headaches followed by deterioration of the level of consciousness. A CT scan of the brain revealed a large right temporoparietal intracerebral hemorrhage with brain edema with no midline shift. Because of this complication and the risk of fatal intracerebral hemorrhage during cardiopulmonary bypass, the surgery was cancelled and conservative treatment instituted. The patient remained febrile. Four days later, the patient expired after another massive intracerebral bleed and herniation.

Discussion

Group G beta-hemolytic streptococcus was first described in 1935 by Lancefield and Hare as a non-pathogenic normal flora of the vagina. It was later recovered from the pharynx, intestine, and skin of healthy humans. The organism, identified by means of beta-hemolysis on blood agar and failure to grow in the presence of bile and agglutination with group specific antisera following antigen extraction, was subsequently isolated from patients with pharyngitis, meningitis, puerperal sepsis, pneumonia, peritonitis, cellulitis, arthritis, wound infection, and endocarditis. The incidence of group G streptococcal infection is rare, accounting for approximately 9% of all streptococcal infections. Over the past decade, an increasing number of reports describing severe infection due to group G streptococci have been published. This might reflect improved laboratory techniques rather than a true increase in the incidence. The hypothesis that the increased group G streptococcal infections are due to change in the virulence of the organism needs further studies. Septicemia was the most frequently reported infection. The incidence of group G streptococcal endocarditis reported in the recent literature is very variable, ranging from 0% to 8% of all cases of endocarditis and 8.4% of beta-hemolytic streptococcal endocarditis. Group G streptococcal endocarditis has been described in all age groups but more frequently in those above 50 years. It affects almost equally both immunocompetent and immunocompromised individuals. The incidence of group G streptococcal endocarditis was higher in patients with underlying malignancy, diabetes mellitus, and alcoholic liver cirrhosis.

Both normal and abnormal valves are affected. The aortic valve was the most frequently affected normal valve, followed by the mitral valve and the tricuspid valves. Of the abnormal valves, the mitral valve is commonly affected followed by the aortic valve. Associated valvular lesions are mitral regurgitation, mitral valve prosthesis, aortic valve prosthesis, and aortic regurgitation. The only reported case of tricuspid group G streptococcal endocarditis was in an intravenous drug abuser. The commonest portal of entry of the organism was the skin, followed by the respiratory and genitourinary tracts. A patient had clinical and laboratory evidence of urinary tract infection, however, the organism was not isolated from the urine. Acute group G streptococcal endocarditis is more common than the subacute form. Most of the patients presented within one week of the onset of symptoms. Like
the other forms of acute infective endocarditis, the symptoms are nonspecific and point to severe septicemia.5,22,24,26 It might initially be confused with one of the connective tissue disorders. The disease usually progresses rapidly with a high rate of complications. Of notice is the severe central nervous system complications, namely embolic cerebrovascular accidents and associated group G streptococcal meningitis. Patients with associated meningitis usually presents with mental confusion and generalized petechiae and can easily be confused with meningococcal meningitis.4,8,10,16,22,24 Our patient had two episodes of intracerebral hemorrhage, the second was complicated by cerebral herniation and death. This complication, most likely due to ruptured mycotic aneurysm, was not previously reported in group G streptococcal endocarditis. Other frequent complications are valve destruction4,5,10,12,13,18-20,22 with consequent development or worsening of heart failure and arterial embolisms.4-7,9,10,15,16,19,20,22 The leukocyte count, ESR, and CRP are usually combatable with acute bacterial infection. The blood cultures were positive in all reported patients and the organism was invariably sensitive to penicillin when tested.4-3o However, the in vivo response in patients with endocarditis is poor and delayed with consequent development or worsening of heart failure and arterial embolisms.4-7,9,10,15,16,19,20,22 The leukocyte count, ESR, and CRP are usually combatable with acute bacterial infection. The blood cultures were positive in all reported patients and the organism was invariably sensitive to penicillin when tested.4-3o However, the in vivo response in patients with endocarditis is poor and delayed with consequent development or worsening of heart failure and arterial embolisms.4-7,9,10,15,16,19,20,22 The leukocyte count, ESR, and CRP are usually combatable with acute bacterial infection. The blood cultures were positive in all reported patients and the organism was invariably sensitive to penicillin when tested.4-3o However, the in vivo response in patients with endocarditis is poor and delayed with consequent development or worsening of heart failure and arterial embolisms.4-7,9,10,15,16,19,20,22

Conclusion

Group G streptococcal endocarditis is a severe potentially fatal disease, characterized by a high rate of complications and delayed poor in vivo response to antimicrobial therapy inspite of good in vitro sensitivity. Treatment with a combination of benzylpenicillin and aminoglycoside is recommended. Early valve replacement should be considered, particularly if the response to the antimicrobial therapy is unsatisfactory.

References