Group B Streptococcal Endocarditis of Tricuspid Valve*

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We report three cases of group B streptococcal endocarditis of the tricuspid valve. Two patients were intravenous drug abusers. In the literature review, and including our cases, ten patients had group B streptococcal endocarditis of the tricuspid valve. Half of the patients were intravenous drug abusers. Four of the other patients had underlying conditions. All patients were treated with a penicillin with or without an aminoglycoside. Three patients underwent tricuspid valve surgery. The overall mortality was 20 percent. Both patients who died received medical therapy only.

(Chest 1991; 100:569-71)

**MIC = minimum inhibitory concentration**

Infective endocarditis due to group B Streptococcus (Streptococcus agalactiae) usually involves the mitral and aortic valves. Tricuspid valve endocarditis is mainly found in intravenous (IV) drug abusers and the infecting organism is *Staphylococcus aureus* in the majority of cases. We report herein three cases of tricuspid valve endocarditis due to group B streptococcus, and review the literature on this subject.

**Case Reports**

**Case 1**

A 22-year-old IV drug abuser was admitted to the hospital with a four-day history of fever, chills, subternal chest pain on coughing, and greenish sputum. She had a history of several episodes of tricuspid valve endocarditis due to *Haemophilus parainfluenzae*, *Streptococcus mitis* plus *Streptococcus sanguis* II, and *Streptococcus acidominimus* with septic pulmonary emboli.

The temperature was 30°C. There was decreased breath sounds over the right lung base. There was grade 3/6 holosystolic murmur over the left subternal border that increased with inspiration. Pertinent laboratory data were as follows: hemoglobin, 14.2 g/dl; hematocrit, 43.8 percent; white blood cell count, 15,900/cu mm, with 79 percent segmental forms, 4 percent band forms, 12 percent lymphocytes, 5 percent monocytes; 
P<0.03 mg/L, 25 mm Hg; PCO_, 27 mm Hg; pH, 7.32; HCO_, 22.8 mmol/L; and O_2 saturation, 98.3 percent on room air. Two of the two blood cultures grew group B Streptococcus susceptible to penicillin, ampicillin, cephalothin, vancomycin, erythromycin, and clindamycin.

The patient was treated with penicillin G 5 million units intravenously every 6 h. She also received one week of gentamicin therapy. Ventilation-perfusion lung scan showed high probability of pulmonary emboli. Two-dimensional (2D) echocardiogram showed a large mobile shaggy echo-dense mass 1.5 to 2.0 cm compatible with vegetation on the tricuspid valve with significant tricuspid regurgitation. The patient’s condition improved but she never became completely afebrile. She had several episodes of chest pain with hemoptysis. The patient died on the 27th day after massive hemoptysis.

**Case 2**

A 13-year-old girl was admitted to the hospital because of the findings of a vegetation on the tricuspid valve on 2D echocardiogram. Three days before hospital admission, she went to the emergency department with acute anterior chest pain. The chest roentgenograms showed an enlarged heart. She was given cephalaxin and was referred to a cardiologist who performed the 2D echocardiogram. At the time of hospital admission, she had no complaint. She gave a history of being treated for Lyme disease several times during the past three years. The symptom was mainly intermittent arthralgia. She had no history of exposure to ticks. She was afebrile. She had no pericardial rub. There was a soft grade 1/6 systolic murmur over lower left sternal border. The lungs were clear. Liver and spleen were not palpable. There were no skin lesions.

One of three blood cultures grew group B Streptococcus susceptible to penicillin (minimum inhibitory concentration [MIC] <0.03 mg/L), ampicillin (MIC <0.125 mg/L), cefazolin, clindamycin, erythromycin, vancomycin, and resistant to tetracycline. The chest roentgenograms showed an enlarged heart but clear lung fields. Repeated 2D echocardiogram showed slight pericardial effusion and a small tricuspid vegetation. The erythrocyte sedimentation rate (ESR) was 35 mm/h. The hemoglobin was 11.3 g/dl and hematocrit was 35.2 percent. The white blood cell count was 10,600/cu mm. Lyme disease antibody was negative.

She was treated with penicillin G 5 million units IV every 6 h and later discharged from the hospital to complete a 4-week course of intravenous penicillin G therapy. Her treatment course was uncomplicated.

**Case 3**

A 32-year-old IV drug abuser was admitted to the hospital with a one-week history of fever and nonproductive cough. She also complained of left knee pain and back pain. She has a history of *S. aureus* tricuspid valve endocarditis three months before hospital admission.

Her temperature was 39°C. The lungs were clear. There was a grade 2/6 pansystolic murmur over the lower left sternal border. There was right saccral tenderness. The left knee was swollen. The hemoglobin was 11.8 g/dl, and white blood cell count was 9,600/cu mm with 69 percent segmental forms, 1 percent band forms, and 30 percent lymphocytes. Six of six blood cultures grew group B Streptococcus susceptible to penicillin (MIC, 0.06 mg/L). A 2D echocardiogram showed a 0.5-cm vegetation on the tricuspid valve. The bone scan was normal. The patient refused arthrocentesis of her left knee. Initial chest roentgenogram was normal. Repeated chest roentgenogram when she had chest pain showed left lung infiltrates. The patient was treated with IV penicillin G 2 million units every 4 h for 45 days with gentamicin for the first 2 weeks. She was well at six-month follow-up.

**Review of the Literature**

There have been seven cases of group B streptococcal tricuspid valve endocarditis without involvement of other valves reported in the literature. The important clinical features of these cases together with three cases from this report are listed in Table 1. The ages ranged from 13 to 65 years. There were three men and seven women. Eight of the ten patients have acute illness. Only five of these patients had a history of IV drug abuse. Two had diabetes mellitus (cases 6 and 7); one was after cesarean section (case 1); one was after saline solution–induced abortion (case 2); one had breast cancer (case 4); and two had history of alcoholism (cases 4 and 7). Nine patients had clinical evidence of septic pulmonary emboli. Vegetations were seen on the tricuspid
valve by 2D echocardiography in six patients. With the exception of the patient with mixed S aureus infection, all patients were treated with either penicillin with or without an aminoglycoside. Tricuspid valve surgery was performed in three patients. Two patients died (mortality, 20 percent) and both received medical therapy only.

**Discussion**

In recent years, the group B Streptococcus assumes an increasing role as an important pathogen in causing serious infections in adults, including bacteraemia and endocarditis.1-3,9-11 That most patients who have group B streptococcal endocarditis have involvement of the aortic and mitral valve is not surprising since right-sided endocarditis is uncommon in patients who are not IV drug abusers.10 Of the ten patients with group B streptococcal endocarditis reviewed herein, 50 percent did not have a history of IV drug abuse. One of these five patients had diabetes mellitus, one was after cesarean section, one had cancer of the breast and alcoholism, and one had both diabetes mellitus and alcoholism. In contrast, only one of the five IV drug abusers had an underlying condition, that being after a saline solution abortion. It is known that tricuspid valve endocarditis is common in IV drug abusers.13 Although many organisms can cause infective endocarditis of the tricuspid valve in IV drug abusers, S aureus remains the most common cause and group B Streptococcus is a rare infecting organism.3,8,13 Polymicrobial tricuspid valve endocarditis is not uncommon in IV drug abusers.14 Only one patient in this review had polymicrobial endocarditis with S aureus.

Penicillin with or without an aminoglycoside remains the drug of choice in the treatment of group B streptococcal endocarditis.12 However, despite the fact that group B Streptococcus is susceptible to penicillin, endocarditis caused by this organism is associated with high mortality (42 to 43.5 percent).1,2 In contrast, the mortality of group B streptococcal endocarditis of the tricuspid valve in this review is only 20 percent. Tricuspid valve endocarditis, in general, is known to be associated with low mortality.2,12

Early surgery has been recommended for left-sided group B streptococcal endocarditis.5,5 The indication for valve surgery in tricuspid valve endocarditis has not been established. Surgery was performed in three of the ten patients in this series. One of the patients who was reported herein died of massive pulmonary hemorrhage that has been reported as a complication of tricuspid valve endocarditis.16 This patient also had a large vegetation, which by itself is not an indication for surgery.16 Surgery was considered in this patient, but she died before it was done. In retrospect, tricuspid valvulectomy might have prevented recurrent pulmonary emboli and hemoptysis as a cause of death in this patient.

In summary, group B Streptococcus is capable of causing tricuspid valve endocarditis both in IV drug abusers and in patients who have no history of IV drug abuse. The mortality is less than half of the mortality of group B streptococcal endocarditis of the mitral or aortic valve.

**References**

6 Dressler FA, Roberts WC. Infective endocarditis in opiate addicts: analysis of 80 cases studied at necropsy. Am J Cardiol 1989; 63:1240-57
8 Jemsek JG, Gentry LO, Greenberg SB. Malignant group B

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**Table 1— Group B Streptococcal Endocarditis of Tricuspid Valve**

<table>
<thead>
<tr>
<th>Case/Age, yr/Sex</th>
<th>Presentation</th>
<th>IV Drug Abuse</th>
<th>Pulmonary Emboli</th>
<th>2D Echocardiogram</th>
<th>Medical Therapy</th>
<th>Surgery</th>
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*R = recovered; D = died; NS = not stated; PR = present report; IV = intravenous; and 2D = two dimensional.

†This patient had a mixed infection with Staphylococcus aureus.
Myocardial Infarction due to Intracoronary Thrombi without Significant Coronary Artery Disease in Systemic Lupus Erythematosus*

Ali H. Katom, M.D.; and Harry R. Gibbs, M.D.

Acute myocardial infarction is a potentially fatal complication of SLE. Reported mechanisms include atherosclerosis, arteritis and coronary arterial spasm. The following case report presents a fourth possible cause; intracoronary thrombus with angiographically normal coronary arteries in a patient with active lupus and AMI.

(Chest 1991; 100:571-72)

AMI = acute myocardial infarction; SLE = systemic lupus erythematosus; CK = creatine kinase; ANA = antinuclear antibody; DNA = deoxyribonucleic acid; ESR = erythrocyte sedimentation rate

A 26-year-old black woman with a four-year history of SLE was admitted with a three-day history of abdominal discomfort and nausea, followed by pleuritic chest pain. There was no history of hypertension, hypercholesterolemia or Raynaud's phenomenon. Her current medications included prednisone, aspirin and indocin. She had not taken birth control pills. Cardiac examination disclosed no abnormalities.

Electrocardiography revealed diffuse ST segment elevation, suggestive of acute pericarditis. Initial serum CK level was 3,200 U/L, with 15 percent MB fraction. Subsequent levels were significantly lower, but remained above normal. Serial ECGs showed a decrease in R wave progression. The ANA level was 1:2,800; anti-DNA was 221 (1 to 160); and ESR was 36 mm/h. Lupus anticoagulant and anticardiolipin antibodies were negative. Technetium 99m stannous pyrophosphate imaging revealed 2 to 3+ positivity localized to the apex. Thallium 201 chloride imaging showed decreased apical activity. Two-dimensional echocardiography demonstrated distal septal hypokinesis and apical dyskinesis. Symptoms resolved with nitrate therapy. Angiography, performed on the tenth hospital day,

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FIGURE 1. Left anterior oblique view of the left anterior descending artery, showing a proximal filling defect (arrow).

FIGURE 2. Left anterior oblique view three months later showing resolution of the thrombus.