Case Report

Group B *Streptococcal* endocarditis

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ABSTRACT

Group B *Streptococcus* can cause early onset neonatal disease. Beyond neonatal life, group B *Streptococci* are unusual pathogens. It can cause septicemia, epiglottis, fascitis, and endocarditis. A male Saudi child with group B endocarditis who has congenital heart disease is discussed.

Keywords: *Streptococcus*, endocarditis.

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Streptococcus Group B (*Streptococcus agalactie*) was known at the end of the 19th century as a cause of bovine mastitis but it was not until the 1930s that its association with human disease was recognized. Infections with *Streptococcus* Group B have been reported with increasing frequency and have now replaced group A *streptococci*, the scourge of the pre 1940s, as the major streptococcal pathogens in neonates and young infants. In the neonatal period, group B *streptococcus* is a major pathogen causing meningitis and bacteremia. Beyond the neonatal period, it has been reported to cause epiglottitis,[1] necrotizing fasciitis,[2] and osteomyelitis.[3] Infective endocarditis is an unusual presentation of group B *streptococcal* infection. We report a case of group B *streptococcal* endocarditis in a 2.5-year-old child with congenital heart disease and review the English medical literature.

Case Report. ***Patient One.*** A 30-month old Saudi boy with complex congenital heart disease (double outlet right ventricle, ventricular septal defect, pulmonary valvular and subvalvular stenosis with transposition of the great vessels and severe proximal left pulmonary stenosis) underwent total repair utilizing Rastelli procedure at the age of 21-months. He had an uneventful immediate post-operative course with no major complication. Five months later, he presented with fever associated with poor appetite and decreased activity of one-month duration. Physical examination showed an ill looking child with respiratory rate of 44 breaths/minute, heart rate (HR) 160 beats/minute, temperature 39°C and blood pressure 80/50 mm Hg with oxygen (O_2) saturation 80%. Cardiovascular examination revealed an active precordium with a loud systolic murmur, grade 3/6 heard all over the chest. Liver was 4 cm below the costal margin with no palpable spleen. Central nervous system examination revealed a conscious child with no signs of meningitis or focal neurological deficit. Complete blood count showed white blood cell 19.7×10^9/L (polymorph neutrophils 36.4%, lymphocytes 47%), hemoglobin 10.3 gm/dl platelets 35×10^9/L, erythrocyte sedimentation rate (ESR) 10mm/hour, prothrombin time (PT) 17.8 seconds (normal 11.9-14.3 seconds), partial

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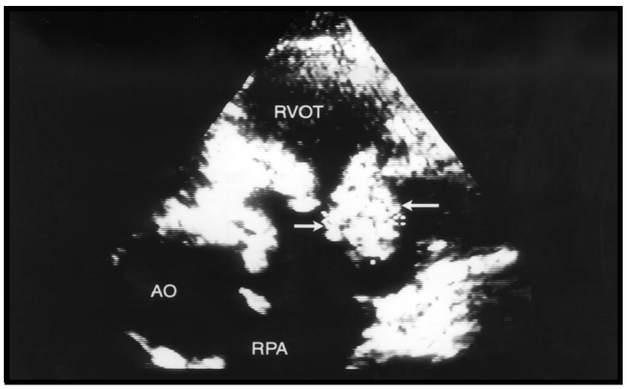

Figure 1 - Two-dimensional echocardiogram showing large vegetation at the pulmonary valve (arrow). RVOT - right ventricular outlet tract, AO - aorta, RPA - right pulmonary artery.

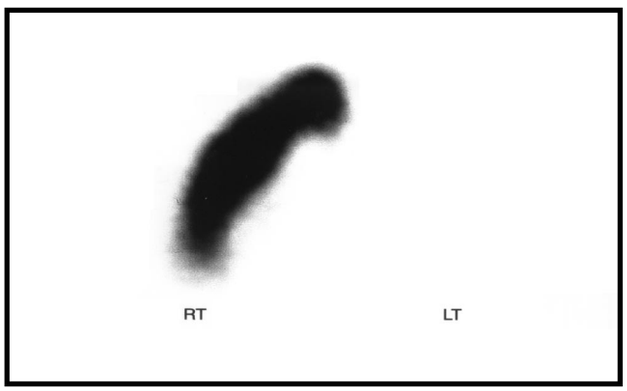

Figure 2 - Ventilation perfusion scan showing marked decreased perfusion to the left lung. RT - right, LT - left.

thromboplastin time (PTT) 46.9 seconds (normal 34.7-42.2 seconds). Blood culture on 3 consecutive occasions grew group B β-hemolytic *streptococcus*. Echocardiogram showed vegetation on the pulmonary valve (**Figure 1**). The patient was initially commenced on vancomycin, gentamicin, and ceftazidime. In-vitro susceptibility testing however showed that the organism was sensitive to penicillin (MIC 0.032 microgram/ml), and ceftriaxone (MIC 0.047 microgram/ml). The antibiotics were therefore, changed to penicillin and gentamicin. The fever subsided and the patient's general condition improved during the first week of therapy. On the 8th day of hospitalization, he suddenly developed acute respiratory symptoms with tachypnea and tachycardia. A diagnosis of pulmonary embolism was entertained, and the patient was transferred to the intensive care unit and mechanically ventilated. Echocardiogram showed that the vegetation had been dislodged from the pulmonary valve. Ventilation perfusion scans showed a marked decrease in perfusion of the left lung (**Figure 2**). Cardiac catheterization showed vegetation on the homograft at the pulmonary valve, with severe left pulmonary artery stenosis. Due to hemodynamic instability, surgical intervention was recommended and the homograft was replaced. The pulmonary valve appeared infected. The patient did well post-operatively. He received 4 weeks of intravenous antibiotics. At follow-up after discharge from the hospital, he remained well.

Discussion. Group B *streptococcus* can cause early onset neonatal disease (age <7 days) or late onset disease (>7 days). Beyond neonatal life, group B *streptococci* are unusual pathogens. Our case illustrates an invasive group B *streptococcal* infection in a child with congenital heart disease, causing bacteremia, and endocarditis. There is limited experience with invasive group B *streptococci* beyond extremes of age. This patient had a smouldering course before presentation with prolonged fever and thrombocytopenia. In addition, after commencing therapy his course was complicated by the development of pulmonary embolism due to dislodgment of vegetation. Infective endocarditis is an uncommon manifestation of group B *streptococcal* disease. It is a disease of the elderly with 45% of cases having been reported in those 60 years of age or older.[4] Underlying heart diseases were found in more than one half of the patients. Other conditions that predispose patients to group B *streptococcal* endocarditis include diabetes mellitus, malignancy, alcoholism, cancer, pregnancy, intravenous drug abuse and genitourinary disease. Penicillin with or without aminoglycoside remains the preferred regimen for the treatment of group B *streptococcal* endocarditis. Pedunculated vegetations may require surgical intervention. The overall mortality for all treated patients is 50%. Mortality in those treated with antibiotics alone was 60%, while in those treated with both antibiotics and surgery was 29%.[5]

A review of the English medical literature (1966-1999) using computerized Medline search revealed 5 cases of group B *streptococcal* endocarditis in children beyond the neonatal period.[4-8] It can involve any valve and produce large pedunculated vegetations, which may dislodge and produce emboli. The important clinical features of these cases are summarized in **Table 1**. Ages ranged from 45-days to 13-years, with equal sex distribution. Vegetations in the involved valves were demonstrated by 2-dimensional echocardiogram in all patients. Penicillin alone or combined with gentamicin was used in the majority of patients.

In conclusion, group B *streptococcal* endocarditis is unusual in infants and older children, with only 6 cases beyond the neonatal period reported including our patient. Antibiotic therapy is the mainstay of

Table 1 - Summary of case reports in literature.

Author	Age	Sex	Valve involved	Echo		Medical therapy and duration	Surgery	Outcome
				Cardiac legion	Vegetation			
Al-Soub et al[4]	8 years	Male	Mitral	MR	Large	Penicillin 6 weeks, Gentamicin 2 weeks	No	Survived
Sledge et al[5]	7 months	Female	Tricuspid	TR	Large tricuspid	Ampicillin 6 weeks, Gentamicin 6 weeks	Yes	Survived
Horigome et al[6]	20 months	Female	Mitral & aortic	AR and MR	Small aortic	Penicillin Streptomycin	No	Died after 7 days
Moral et al[7]	1.5 months	Male	Pulmonary	NL	NL	Not stated	Yes	Survived
Watana & Habte-Gaber[8]	13 years	Female	Tricuspid	NL	NL	Penicillin 4 weeks	No	Survived
Al Shahrani et al (this study)	30 months	Male	Pulmonary	NL	NL	Penicillin 4 weeks	Yes	Survived

MR - mitral regurgitation, TR - tricuspid regurgitation, VSD - ventricular septal defect, AR - aortic regurgitaton, NL - no lesion

management; however, surgery may be considered in patients with large or pedunculated vegetations that imposes a risk for embolization. The course of this patient was complicated by pulmonary embolism and hemodynamic instability necessitating surgical intervention. Whether such complications happen more frequently with group B *streptococcus* compared with other organisms is not clear due to limited experience.

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