Echocardiographic Detection of Bacterial Vegetations in a Child with a Ventricular Septal Defect*  

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A 13-year-old boy with a small ventricular septal defect was admitted with clinical manifestations of acute endocarditis. Coagulase-positive staphylococci were isolated from the blood. Definitive diagnosis was made by detecting bacterial vegetations in the right ventricle on the echocardiogram. Repeated embolization of these vegetations to the pulmonary circulation led to the death of the patient.

Bacterial endocarditis is an important complication which affects children with congenital heart disease. With the advent of antibiotics, the mortality due to bacterial endocarditis has decreased over the last two decades; however, the incidence of bacterial endocarditis seems to have increased, due mainly to the longer survival of children with congenital heart disease, since surgical correction of many of these conditions is now feasible.1

The diagnosis of bacterial endocarditis is most often suspected because of persistent fever in patients with a known congenital cardiac lesion. Other clinical features, such as changes in the cardiac murmur, the appearance of a new murmur due to intracardiac structural damage, or embolization to the systemic or pulmonary circulation, are infrequently present, and even positive blood cultures are not always found.1-3 Echocardiographic studies have been found to be useful in the diagnosis of bacterial endocarditis by detecting and localizing the bacterial vegetations.4 We report the findings in a patient with acute bacterial endocarditis in whom the bacterial vegetations were located in the right ventricle on the echocardiogram.

METHODS

The echocardiogram was obtained using an echocardiograph (Ekoline) with a 2.5-MHz transducer focussed at 5 cm. The aorta and the left ventricle were scanned using the standard position (fourth intercostal space). The pulmonary valve was located by moving a space higher and rotating the transducer anteriorly toward the left shoulder. The tricuspid valve was best seen by angulating the transducer medially and inferioiy from the fourth to fifth intercostal space near the sternum.

CASE REPORT

The patient had been known to have a cardiac murmur since birth. When he was five years old, cardiac catheterization revealed a small ventricular septal defect with a pulmonary-to-systemic blood flow ratio of 1.3 and normal pulmonary arterial pressures. The patient remained asymptomatic until his last illness.

At the age of 13 years, one week following removal of dental braces without antibiotic prophylaxis, the patient developed fever, vomiting, and generalized lethargy. Five days later, he developed tachypnea and pain in the left shoulder and was treated with erythromycin for a clinical diagnosis of bacterial pneumonia.

Deterioration in his general condition prompted admission to the Children's Memorial Hospital ten days after the onset of symptoms. On admission the patient was acutely ill and in a semiconscious state, with periods of extreme irritability. Physical findings included a temperature of 39°C (102.2°F), nuchal rigidity, a sparse petechial skin rash over the feet, arms, and trunk, splinter hemorrhages in the nail beds, and hepatosplenomegaly. Moderately severe respiratory distress led to progressive hypoxia and necessitated artificial ventilation. Bilateral rales were noted in both lung fields, and a chest x-ray film showed densities bilaterally. A grade 3/6 pansystolic murmur was noted, which was essentially unchanged from the previous examination. The second heart sound was closely split.

Laboratory investigations showed the following: hematuria; increased levels of serum creatinine (1.8 mg/100 ml), blood urea nitrogen (78 mg/100 ml), and serum bilirubin (total 5.4 mg/100 ml, with 3.6 mg/100 ml direct); elevated transaminase values; hemoglobin level 9.6 gm/100 ml; hematocrit, 28 percent; and total white blood cell count, 9,000/cu mm, with 73 percent bands. The platelet count was reduced (35,000/cu mm), and the prothrombin time and partial thromboplastin time were both increased. Blood cultures grew coagulase-positive Staphylococcus aureus. Digoxin and dopamine were given for cardiovascular support. Therapy with nafcillin and gentamicin was started to control the generalized bacterial infection.

On the evening of admission, an echocardiogram was obtained. The semilunar and atrioventricular valves were morphologically normal. The left atrium and left ventricle were both free of abnormal echoes; however, in the body of the right ventricle, dense echoes were seen, which became more persistent along the outflow tract of the right ventricle (Fig 1). When the beam was directed toward the right and inferior to the aortic root, these echoes became maximum and

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Echocardiographic detection of bacterial vegetation: The virulent local.

Infective endocarditis is a bacterial disease that affects the heart valves, causing inflammation and abscess formation on the valve. The bacteria responsible for this disease are often Streptococcus viridans, Staphylococcus aureus, and enterococci. The infection is usually caused by the spread of bacteria from a site of infection elsewhere in the body, such as a dental abscess or a skin infection. Once the bacteria reach the heart, they attach to the valve leaflets, causing inflammation and abscess formation.

The most common clinical presentations of infective endocarditis include fever, weight loss, fatigue, and heart failure. Other symptoms may include chest pain, shortness of breath, and a new or worsened heart murmur. Diagnosis is usually made by a combination of clinical symptoms, a physical examination, and diagnostic tests such as echocardiography, blood cultures, and imaging studies.

Treatment of infective endocarditis typically involves a combination of antibiotics and often surgery to repair or replace the damaged valve. Early diagnosis and treatment are crucial in reducing the risk of complications and improving outcomes.

This page features an echocardiogram showing the location of bacterial vegetations on the heart valve. The vegetations are depicted as dark areas on the echocardiogram, indicating the presence of infection. The clinical significance of these findings is discussed in the accompanying text, highlighting the importance of prompt diagnosis and appropriate treatment.
Mucoepidermoid Tumor of Trachea*

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Mucoepidermoid carcinoma of the trachea is rare. Its occurrence in a 14-year-old boy is reported here. This case illustrates the typical course of tracheal tumors with clinical manifestations of cough, wheezing, and hemoptysis, the initially reported normal chest roentgenogram, and the common failure to diagnose tracheal tumor for several months. Early use of tomographic studies and bronchoscopic examination in any person with recent onset of airway obstruction unresponsive to bronchodilator therapy is recommended.

Primary tumors of the trachea are rare but have been reported with increasing frequency in recent years. Symptoms of cough, hemoptysis, dyspnea on exertion, and wheezing are nonspecific. Although manifestations are not recurrent but usually are progressive, patients may be treated for asthma, delaying the proper diagnosis. Because of their location, these tumors are often missed on routine chest roentgenograms. The clinician should alert the radiologist to the atypical clinical features.

Case Report

This 14-year-old white boy was treated for asthma for six months. His initial symptoms were sore throat, nonproductive cough, and wheezing which persisted. Bronchodilator drugs were administered; however, symptoms progressed, and the patient developed intermittent cough, producing yellow-brown mucus. Four reports of hemoptysis, a 2.5 kg (55 lb) weight loss, pain over the left sternal border, and increasing difficulty with inspiration were noted.

The patient's past history and the family history revealed no chronic respiratory problems. Six months after the onset of symptoms, the patient was referred to the Allergy/Clinical Immunology Clinic at the Rush Medical Center. The physical findings were pallor, moderate dyspnea, and mild wheezing. The patient's peak expiratory flow rate was 110 L/min (normal, 400 to 500 L/min), which improved to 180 L/min after administration of epinephrine (Sus-Phrine). This initial chest x-ray film was reported as normal (Fig 1). The patient was admitted to the hospital for further evaluation.

On admission, the patient was normally developed and nourished; however, he appeared pale and in mild respiratory distress. The blood pressure was 122/80 mm Hg, the respiratory rate was 18/min, the pulse was 120 beats per minute, and the temperature was 37°C (98.6°F) orally. Findings from physical examination were within normal limits, except auscultation revealed mild wheezing bilaterally without rales, rhonchi, or stridor. Aside from the tachycardia, the findings

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Figure 3. Postmortem specimen showing pulmonary arterial root (MPA), right ventricular outflow (PB, parietal band; SB, septal band), and inflow tracts. Friable grayish-white vegetations (VEG) are seen covering area of endocardial sclerosis related to jet flow from small ventricular septal defect (VSD). Septal leaflet of tricuspid valve (TV) is free of vegetations. Massive vegetations were present in body and outflow tract, but due to extreme friability, many were dislodged during dissection.