CASE REPORT

Infective Endocarditis Due to Streptococcus Pneumonia

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Abstract:

A young patient with a ventricular septal defect presented with Streptococcus pneumoniae pneumonia complicated by endocarditis. The presentation was acute with high fever, hypotension, leukocytosis, and renal impairment. The organism was penicillin sensitive. Treatment with ampicillin for four weeks produced a successful outcome. Endocarditis due to Streptococcus pneumoniae has become rare in recent years and the association with a ventricular septal defect has not been reported before. Pertinent literature is reviewed.

Key words: Endocarditis, Streptococcus pneumoniae, pneumococcal endocarditis.

Introduction:

Infective endocarditis is an uncommon manifestation of Streptococcus pneumonia infection. In the pre-antibiotic era, Streptococcus pneumoniae caused approximately ten per cent of all cases of native valve endocarditis but the introduction of penicillin and other antibiotics substantially reduced the incidence of pneumococcal endocarditis to less than one per cent. Aronin et al. in a review of the English literature published from 1966 to 1996 found 197 reported cases. There is a strong association with alcohol consumption. The usual portal of entry for infection is the lung. Pneumococcal endocarditis characteristically presents as an acute illness often accompanied by meningitis, rapid destruction of heart valves, congestive heart failure, and is associated with a high mortality. We describe a patient with a ventricular septal defect who developed pneumococcal pneumonia complicated by endocarditis, and the successful outcome.

Case Report:

A 14-year-old male was admitted to Hamad General Hospital in December 2000 with the complaints of fever, shortness of breath, and a productive cough of three days duration. He was a known case of Down's syndrome with ventricular septal defect (VSD) diagnosed when he was four years old. Physical examination on admission revealed an ill-looking patient, with a blood pressure of 80/60 mm Hg, pulse rate 120/minute, respiratory rate 35/minute, temperature 39.2 °C, and bilateral coarse crepitation at both bases although more on the left side. Heart examination revealed a palpable thrill and a pansystolic murmur at the left sternal border. The rest of the physical examination was unremarkable. Laboratory investigations revealed: haemoglobin 13.4 gm/dL, platelets 178,000/mm³, white blood cell count 24,000/mm³, with 93% polymorphonuclear neutrophils, 5% lymphocytes, and 2% monocytes; serum creatinine 163 μmol/L, alanine aminotransferase 31 IU/L, aspartate aminotransferase 77 IU/L, alkaline phosphatase 21 IU/L, lactic acid 5.7 mmol/L; arterial blood gases: pH 7.36, Pco₂ 34 mm Hg, Po₂ 58 mm Hg. Chest radiograph showed a non-homogenous infiltrate at the left base. Two sets of blood culture grew Streptococcus pneumonia colonies sensitive to penicillin (MIC ≤ 0.6 μg/mL). The patient was admitted to the intensive care unit, given intravenous fluids and intravenous ceftriaxone and erythromycin (later changed to ampicillin as penicillin was not available in the hospital at that time). He improved in response to treatment. In view of the presence of VSD and the positive blood
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Discussion:

Our patient fulfilled Dukes’s criteria for definite infective endocarditis (4); he had a predisposing condition for endocarditis (ventricular septal defect), fever (temperature > 38°C), two positive blood cultures growing Streptococcus pneumonia, and an echocardiogram demonstrating vegetations. To our knowledge this is only the second case of infective endocarditis due to S. pneumonia seen in our hospital over the last 18 years. Hamad hospital is the only hospital providing acute medical care for the whole of Qatar. This finding is in accordance with the international literature which indicates that infective endocarditis due to S. pneumonia is rare (1,2,5,6). Aronin et al (2) in a review of the English language literature from 1966 to 1996 found 197 cases of S. pneumonia infective endocarditis. Lefort et al (6) reported 30 cases of S. pneumonia infective endocarditis from France between 1991 and 1998. Infective endocarditis due to S. pneumonia usually has an acute presentation, with fever, sepsicaemia, and rapidly progressive congestive heart failure (1). Subacute presentations have been described occasionally (7). Typically the disease follows pneumonia in the setting of alcoholism (2). Some patients, especially the elderly, have the typical Osler’s triad of pneumonia, endocarditis, and meningitis (2). Less commonly it follows primary infections at other extrapulmonary sites (2). Underlying valvular heart disease is not a prerequisite for pneumococcal endocarditis, and normal valves are frequently affected (1). S. pneumonia infective endocarditis is associated with a high morbidity and a mortality variously reported between 28% and 67% (2,3,6). The poor prognosis has been attributed to both host and microbial factors, including older age, and concomitant diseases as well as the organism’s propensity to infect and rapidly destroy left-sided heart valves (2).

The portal of entry of the organism in our patient was the lung. The presentation was acute with pneumonia and endocarditis but no meningitis. He was not alcoholic and had none of the other conditions known to predispose to pneumococcal sepsis. An interesting feature in our patient is the association of pneumococcal endocarditis and ventricular septal defect which has not been reported before although rupture of the ventricular septum has been reported as a complication of pneumococcal endocarditis (9). Fortunately the S. pneumonia in our patient was sensitive to penicillin although more than 50% of S. pneumonia isolates in our hospital are resistant. Accordingly he was treated with parenteral ampicillin alone for four weeks with a successful outcome. The persistence of vegetations as determined by echocardiography is common after successful medical treatment of infective endocarditis and is not associated with later complications (10). There is no formal recommendation regarding the optimal empiric antibiotic therapy for patients with pneumococcal endocarditis, however one of the third generation cephalosporins such as ceftriaxone with or without vancomycin depending on the prevalence of resistance to penicillin in the area seems to be reasonable (2). This regimen should be modified according to the organism’s final susceptibility pattern. The optimal therapy of pneumococcal endocarditis is unknown but a combined medical-surgical approach employing a prolonged course of parenteral antibiotics plus early surgery appears to result in a lower rate of attributable mortality (2,6).

References: