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Brief Report

Tricuspid valvar endocarditis caused by penicillin resistant pneumococcus

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Abstract A 6-year-old boy presented with endocarditis of the tricuspid valve caused by penicillin resistant pneumococcus. He had no history of congenital cardiac disease. The likely portal of entry for the organism was the middle ear.

Keywords: Endocarditis; penicillin resistant pneumococcus; paediatrics

ENDOCARDITIS OF THE TRICUSPID VALVE IS RARE, especially in a structurally normal heart. It is even more unusual when the causative organism is a penicillin resistant pneumococcus. The emergence of penicillin resistant pneumococcal strains is of increasing concern. We report a case of a child, with no history of congenital cardiac disease, who developed tricuspid valvar endocarditis complicated by valvar regurgitation.

Case report

A 6-year-old boy was admitted to a district general hospital with a 5 week history of swinging pyrexia, arthralgia, weight loss and generalised malaise. It was noted that he had a history of repeated infections of the middle ear, for which he had received multiple courses of antibiotics, including cefaclor and cephalexin when admitted which had been prescribed for an episode of acute otitis media. There had been no previous admissions to hospital, and he was normally fit and well. No murmur had been found on any previous examinations.

On clinical examination he was pyrexic. The pulse was regular, with a rate of 130 beats per minute, and normal in character. Blood pressure was 100/35 mmHg. Auscultation revealed a harsh grade 3 pansystolic murmur, loudest at the lower left sternal edge and radiating widely. Femoral pulses were normal. His liver was palpable 2 cm beyond the costal margin. The remainder of the examination was normal. Two separate blood cultures were taken and they became positive for pneumococcus, resistant to penicillin as assessed by disc diffusion sensitivity testing. A diagnosis of acute endocarditis was made and he was commenced on intravenous cefotaxime, given at a dose of 1 gm thrice daily, combined with flucloxacillin 250 mg 6 hourly, before transfer to the regional cardiology centre for further management.

On arrival, an echocardiogram was performed (Fig. 1). This showed a large mobile vegetation adherent to the antero-superior leaflet of the tricuspid valve. There was moderate-to-severe tricuspid valvar regurgitation (Fig. 2). Cardiac structure was otherwise normal, with normal ventricular function. Following consultation with the microbiologists, the patient was commenced on a six-week regimen of vancomycin given at a dose of 190 mg 6 hourly, and gentamicin 50 mg 8 hourly. On the second day of admission, it was noted that there was an opacity in the left midzone of his chest radiograph. A ventilation/perfusion scan was performed which showed no evidence of pulmonary embolism. Four days after admission, minimum inhibitory concentration estimations became available, which showed that therapeutic levels of the drugs were being achieved, with satisfactory trough levels of gentamycin and vancomycin.

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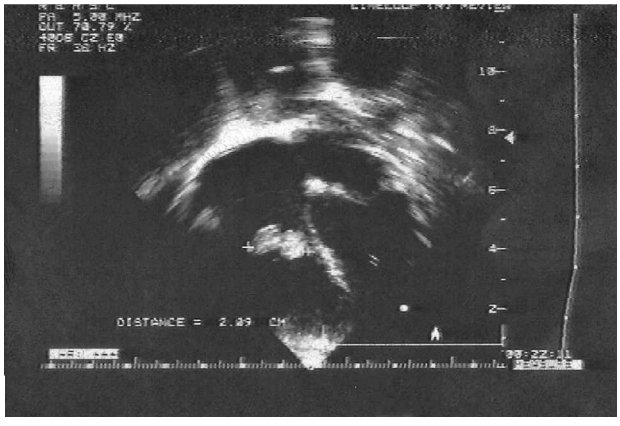


Figure 1.
Four chamber view of heart showing large vegetation attached to antero-superior leaflet of the tricuspid valve.

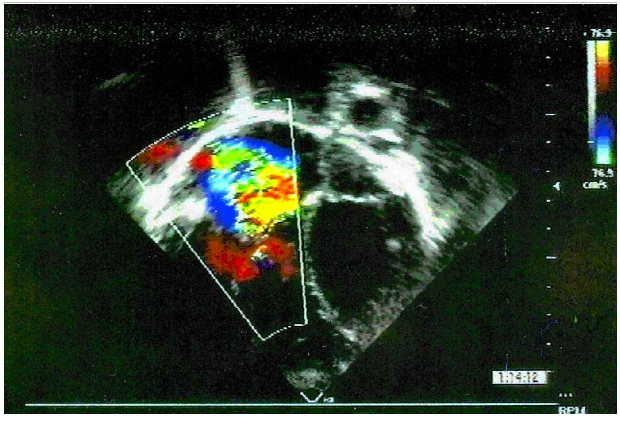


Figure 2.
Colour Doppler imaging demonstrating moderate-to-severe tricuspid valvar regurgitation.

After five days of treatment, repeat blood cultures were negative, although the pyrexia persisted. The antibiotics were therefore changed to Rifampicin, at a dose of 190 mg 12 hourly, and Teicoplanin also at 190 mg 12 hourly for three doses, then 190 mg 24 hourly. After three days of this regime the pyrexia had settled. The antibiotics were continued for a further five weeks. Repeat echocardiograms showed a gradual reduction in the size of the vegetation, and resultant moderate tricuspid valvar regurgitation. The antibiotics were discontinued after the full course, and the patient was discharged home 24 hours after completing the course of treatment. In view of the unusual nature of the cultured organism, extensive immunological investigations were undertaken, all of which proved to be normal.

At subsequent reviews, the tricuspid valve was moderately regurgitant, with prolapse of the antero-superior leaflet. A small remnant of the vegetation remained. The patient required the commencement of diuretic therapy to control symptoms of dyspnoea on exertion. He will remain under cardiology follow-up and may require surgical intervention at a later date.

Discussion

We have described a young boy with endocarditis of the tricuspid valve caused by penicillin resistant pneumococcus. Isolated endocarditis of the tricuspid valve accounts for up to one-tenth of infective endocarditis seen worldwide,[1] although the prevalence in India is closer to one-fifth.[2] This variant of endocarditis has usually been linked with intravenous drug abuse, but there is also an increased incidence in alcoholism, immunodeficiency, in those with indwelling central venous lines, and in the setting of genital sepsis. In our patient, extensive immunological investigations proved normal.

Tricuspid valvar endocarditis affecting a structurally normal heart is rare and, to our knowledge, has only been described previously in a small number of cases with a predisposing cause, for example neonates with indwelling central venous catheters.[3] The organisms usually implicated are *Staphylococcus aureus* and *Streptococcus viridans*. In our patient, a penicillin resistant pneumococcus was isolated, which is rare, and which raises the issues of antimicrobial resistance in pneumococcus and in the treatment of endocarditis.

Penicillin resistant pneumococcus were first reported in Australia, and appeared in the United Kingdom in 1976.[4] The prevalence of intermediate or full resistance to penicillin has increased from 1.5% in 1990 to 3.9% in 1995.[5] There are many different serotypes which have been found to be penicillin resistant. Type 23 is the most common in the United Kingdom.[4] Children have an increased carriage rate of the penicillin resistant pneumococcus, and it is found more commonly in children with repeated infections of the middle ear who have had repeated courses of antibiotics, as is the case in our patient.[6]

New guidelines from the British Society for Antimicrobial Chemotherapy recommend a combination of penicillin and an aminoglycoside, given for not less than 4 weeks when reduced sensitivity to penicillin is identified in streptococcal endocarditis.[7] Vancomycin or teicoplanin for 4 weeks, plus gentamicin for the initial two weeks, is advised for patients allergic to penicillin. There are no specific recommendations for the management of pneumococcal endocarditis. Wilson et al.[8] advise that such management should be informed by sensitivity coupled with the results of minimum inhibitory concentrations, and be coordinated in consultation with a physician

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specialising in infectious diseases. The penicillin resistant pneumococcus is increasingly demonstrating multi-antibiotic resistance, with the third generation cephalosporins and macrolides being particularly affected.

In our patient, the pneumococcus isolated showed intermediate resistance to both penicillin and cefotaxime. The optimal regimen remains unclear and in a large review, Aronin et al. identified only two penicillin resistant infections which detailed the outcome.[9] In both cases, high levels of penicillin and ceftriaxone resistance were encountered. One patient responded to sequential parenteral treatment with imipenem, cefuzonam and ampicillin, while the other responded to a combination of vancomycin and rifampicin. These authors propose empirical therapy with a third generation cephalosporin and vancomycin on the basis that this regimen is established for the treatment of penicillin resistant pneumococcal meningitis.

In conclusion, our experience suggests that penicillin resistant pneumococcal endocarditis should be managed on the basis of estimations of minimum inhibitory concentrations, and a careful review of the clinical response. There are insufficient published series of cases to formulate guidelines.[10] As the minimal values for inhibitory concentrations relative to the penicillin resistant pneumococcus continue to rise, novel regimens may need to be devised.

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