Case reports

Echocardiographic demonstration of pulmonary valve endocarditis associated with congenital ventricular septal defect

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SUMMARY A 38 year old woman previously known to have a heart murmur presented with malaise and bloodstained sputum. Echocardiographic examination showed a subpulmonary ventricular septal defect and probable vegetations on the pulmonary valve, and blood cultures grew Escherichia coli. She made a full recovery after 6 weeks' antibiotic treatment.

The pulmonary valve is rarely affected by subacute bacterial endocarditis and it is the most difficult of the four cardiac valves to visualise by echocardiography. Consequently there are few reports of the echocardiographic demonstration of pulmonary valve vegetations. We present an unusual case in which pulmonary valve vegetations were found in association with a jet lesion from a subpulmonary ventricular septal defect.

Case report

A 38 year old Greek woman with a three month history of general malaise and myalgia and a cough productive of bloodstained sputum who had been told she had a congenital heart lesion travelled to the United Kingdom to seek medical advice.

On examination she was generally well with no stigmata of subacute bacterial endocarditis. Her blood pressure was 120/90 mm Hg with a heart rate of 72 beats/min. There was a loud pansystolic murmur with an associated thrill at the left sternal edge and a soft early diastolic murmur over the pulmonary area that was best heard in inspiration. Her chest x ray showed an enlarged cardiac shadow and pulmonary plethora, but no evidence of pulmonary emboli. The electrocardiogram was within normal limits. A cross sectional echocardiogram showed a ventricular septal defect below the pulmonary valve (Fig. 1), and the presence of a high-velocity jet across this lesion was confirmed with pulsed Doppler examination. The pulmonary valve was thickened and there was a separate echodense mass associated with the valve but which moved independently of it. This was interpreted as a vegetation (Fig. 2). There was Doppler evidence of pulmonary regurgitation. The other valves all appeared to be normal. She had a mild normochromic normocytic anaemia of 11.3 g/dl and a white blood cell count of 10.1 x 10^9/l. Two blood cultures out of six grew Escherichia coli. She was obliged to return immediately to Greece, where she was treated with...
Pulmonary valve endocarditis

Fig. 2 Parasternal short axis section at the level of the pulmonary valve. The pulmonary cusps are abnormally thickened and there is an echodense mass attached to one of them. Ao, aorta; PA, pulmonary artery.

streptomycin and penicillin for 40 days and made a full recovery. Repeat blood cultures were negative.

Discussion

The right heart valves are involved in only about 5% of all cases of endocarditis. It has been suggested that this is due partly to the relatively low incidence of predisposing congenital defects on the right side of the heart and partly to the lower risk of endothelial damage because of lower intracardiac pressures. Thus, infections on normal right sided valves are generally due to virulent organisms, or as a result of heavy inoculations from contaminated intravenous injections, notably in drug addicts.

Staphylococcus aureus is the organism most commonly found in pulmonary valve endocarditis. α haemolytic streptococci, and several less common organisms, such as Pseudomonas, are also found. Escherichia coli, as in this case, is rare but has been reported before.

For reasons that are unexplained, the pulmonary valve is affected in 18–25% of cases of gonococcal endocarditis. With this exception, subacute bacterial endocarditis rarely affects the pulmonary valve even in cases in which there is gross infection of other right sided structures. In Johnson et al’s review of subacute bacterial endocarditis in children, the pulmonary valve was involved in only five of 149 cases.

The risk of endocarditis on the pulmonary valve is increased in the presence of a congenital abnormality or with acquired endothelial damage. The risk of endocarditis in congenital pulmonary stenosis has been estimated to be 0.2 cases per 1000 patient-years. Pulmonary endocarditis has also been reported in Fallot’s tetralogy. Indwelling catheters can cause endothelial damage; in Rowley et al’s study of 55 patients dying after right heart catheterisation, three had vegetations on the pulmonary valve. The endothelium can also be damaged by a high velocity blood jet. In Gersony and Hayes’s series, five patients had subacute bacterial endocarditis associated with a ventricular septal defect, two with vegetations around the margin of the defect and three with vegetations on the aortic valve, one of whom had additional lesions on the tricuspid and pulmonary valves. Bain et al’s study of right sided endocarditis included only one patient with a ventricular septal defect, who had vegetations at the sites of impact of jet lesions on the tricuspid valve and in the right ventricle. Endothelial damage was most probably the disposing lesion in our case, in which the ventricular septal defect was situated just below the pulmonary valve.

The paucity of echocardiographic reports of pulmonary endocarditis results partly from the rarity of the condition and partly from difficulty in visualising the pulmonary valve. M mode recordings have demonstrated a shaggy appearance of the valve in diastole, with or without prominent systolic fluttering. The few cross sectional echocardiographic studies have shown flail leaflets and direct visualisation of vegetative masses. We believe that our case is the first in which an associated ventricular septal defect has been visualised by echocardiography. There is, however, a report of the echocardiographic diagnosis of pulmonary endocarditis where a small ventricular septal defect was subsequently shown at operation.

Subacute bacterial endocarditis must be diagnosed on clinical and bacteriological grounds, but echocardiography is valuable in detecting associated valve lesions. Modern echocardiographic machines can detect vegetations with diameters of >2–3 mm. The addition of Doppler echocardiography provides a highly sensitive method for detecting regurgitant lesions. We found that the technique demonstrated the presence of pulmonary regurgitation and confirmed the site of the ventricular septal defect in a case in which the cross sectional images were only of moderate quality.

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References

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