CASE REPORT

Community acquired staphylococcal pulmonary valve endocarditis in non-drug users: case report and review of the literature

J J Edmond, S J Eykyn, L D R Smith

Abstract

Right sided endocarditis usually involves the tricuspid valve, predominantly in intravenous drug users. It is also occasionally acquired in hospital as a result of contaminated intravascular devices. Isolated infection of the pulmonary valve is rarely seen. A case of community acquired *Staphylococcus aureus* pulmonary valve endocarditis that caused diagnostic confusion is reported. This infection occurred in a patient with no history of intravenous drug abuse and a previously structurally normal heart.

(*Heart* 2001;**86**:e17)

Keywords: *Staphylococcus aureus*; pulmonary valve; endocarditis

Right sided endocarditis usually involves the tricuspid valve and occurs predominantly in intravenous drug users, although occasionally it is acquired in hospital from contaminated intravascular devices. Isolated infection of the pulmonary valve is rarely seen. We describe a patient with community acquired pulmonary valve endocarditis on a structurally normal valve caused by *Staphylococcus aureus.* The patient had no previous history of intravenous drug use and the infection caused diagnostic confusion. We review the previous reported cases of this rare condition.

Case report

A 40 year old previously healthy man was admitted to hospital with one week's history of general lethargy and myalgia with rigors, diarrhoea, and anorexia. His general practitioner had treated him with erythromycin, with little effect on his symptoms. He had never used intravenous drugs, did not smoke, and drank very little alcohol. On examination he looked unwell but was apyrexial. He was tachycardic but had a normal blood pressure. Heart sounds were normal and auscultation of the chest found coarse crepitations at the right base. Abdominal examination was unremarkable. He had a small sore on his cheek, which had been present for five days.

A clinical diagnosis of pneumonia was made, although the initial chest radiograph was unremarkable. Blood tests on admission showed pronounced renal and hepatic dysfunction, C reactive protein (CRP) > 110 mg/l, and raised white blood cell count of 21.6×10^9/l with neutrophilia (92%) and thrombocytopenia (64×10^9/l). He was treated with broad spectrum antibiotics but his condition continued to deteriorate and he was transferred to the intensive care unit for haemofiltration.

When *S aureus* resistant only to penicillin and erythromycin was reported to be growing in both bottles of blood cultures taken on admission, the antibiotic treatment was changed accordingly. An abdominal ultrasound showed only splenomegaly and a transthoracic echocardiogram showed a structurally normal heart. Further chest radiographs showed a septate cavity surrounded by consolidation in the left mid-zone. After 24 days as an inpatient his CRP concentration had returned to normal and he had greatly improved. He was discharged home with oral antibiotics for a further week.

Within 10 days of finishing the oral antibiotics he returned to hospital with rigors, fever, nausea, and vomiting. CRP was 63 mg/l and white blood cell count 8.7×10^9/l; blood cultures were taken and intravenous cefotaxime, flucloxacillin, and gentamicin were given. All bottles of three sets of blood cultures grew *S aureus* sensitive to flucloxacillin; therefore, cefotaxime and gentamicin were stopped. Four days after admission an early diastolic murmur was heard in the left parasternal region and a transthoracic echocardiogram showed large vegetations on the pulmonary valve (fig 1). Intravenous flucloxacillin and oral rifampicin were given for four weeks. The patient felt well and his CRP was normal; he went home only to be readmitted one week later very unwell, febrile, and tachycardic. Blood cultures were taken and he was immediately started on appropriate antibiotics, with systemic improvement. *S aureus* was isolated from all bottles of three sets of blood cultures and he was referred for valve replacement.

At operation the pulmonary valve cusps were found to be destroyed, one with a large vegetation. Gram stain of the excised valve showed a few pus cells and large numbers of Gram positive cocci. A heavy growth of *S aureus* was isolated on culture. He made an

Department of Cardiology, Level 5, Green Lane Hospital, Green Lane West, Auckland, New Zealand
J J Edmond

Department of Microbiology, St Thomas' Hospital, London SE1 7EH, UK
S J Eykyn

Cardiology Department, Royal Devon and Exeter Hospital, Barrack Road, Exeter EX2 5DW, UK
L D R Smith

Accepted 8 August 2001

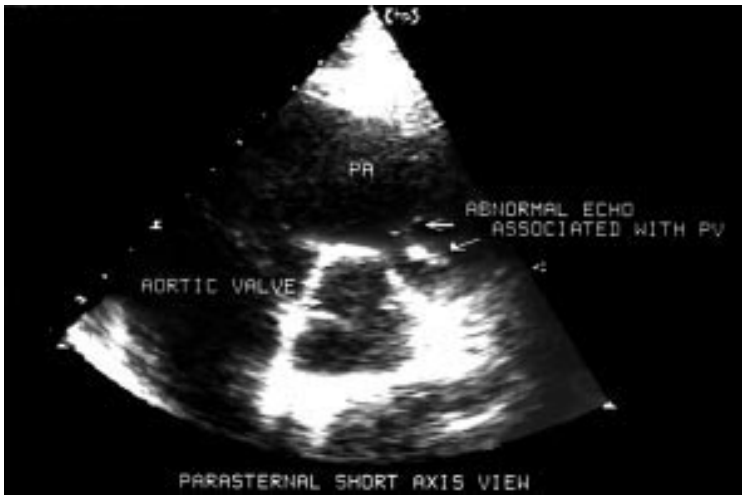

Figure 1 Echocardiograph showing vegetations on the pulmonary valve.

uneventful recovery after a homograft pulmonary valve replacement and four weeks of oral antibiotics.

Discussion

In one year (1999), this and one other case[1] of isolated pulmonary valve endocarditis were seen in the tertiary referral cardiothoracic centre. No other cases had been recognised in the preceding 30 years among 570 prospectively documented cases of endocarditis seen at this hospital. This serves to emphasise how unusual this infection is, particularly when it involves previously normal valves and is not associated with intravenous drug use or intravenous access infection. A review of the literature found only five previously reported cases of *S aureus* isolated pulmonary valve endocarditis in structurally normal hearts in the absence of a history of drug abuse or central venous access (table 1).[1–5] In community acquired *S aureus* endocarditis the source of the staphylococcus is seldom evident but the bacteria must be assumed to have entered the bloodstream through an epithelial breach. In this case, there was a facial sore that was not swabbed but that may have been an infective focus.

Table 1 Previous reported cases of community acquired Staphylococcus aureus pulmonary valve endocarditis in non-drug users

Case report	*Year*	*Patient's age, sex*	*Possible source*	*Pulmonary emboli seen*	*Treatment and clinical outcome*
Levin *et al*[2]	1964	30, male	None	No	Conservative, survived
Cremieux *et al*[3]	1985	31, male	Cutaneous	Yes	Died
Fourestie *et al*[4]	1986	21, female	None	Yes	Died
Calleja *et al*[5]	1992	21, female	Puerperal sepsis	Yes	Surgery, survived
Kelly *et al*[1]	2000	59, male	Unclear	Yes	Conservative, survived

F, female; M, male

Right sided endocarditis presents not with the classical signs of endocarditis but with respiratory symptoms and signs. The patient is usually thought to have pneumonia. Our patient was no exception and was thought to have pneumonia on admission. In four of the five previously reported cases septic emboli were detected, although in one of these patients this was only recognised at a postmortem examination.[4] Community acquired *S aureus* bacteraemia is very much more likely to be associated with endocarditis, or bone or joint infection, than with pneumonia.

Although many organisms have been shown to cause isolated pulmonary valve endocarditis, overall *S aureus* is most commonly involved particularly in cases associated with intravenous access infection and intravenous drug abuse. There are little published data on the need for valve replacement in pulmonary valve infections, but since the tricuspid valve seldom requires replacement in endocarditis by inference this may also be true of the pulmonary valve. In this case report and that of Calleja and colleagues,[5] valve surgery was required as a result of persistent infection rather than haemodynamic instability. Indeed, in the patient reported by Calleja and colleagues the vegetation was removed from the "virtually destroyed" valve, but the valve was not replaced. In reports of other pathogens or *S aureus* infections in intravenous drug abusers, patients have survived with few problems despite seemingly very destructive infections of their pulmonary valves.[6–8]

The most useful clinical message from this case is as a reminder that, although pulmonary valve endocarditis is itself rare, right sided endocarditis must be considered in a patient with community acquired *S aureus* bacteraemia presenting with respiratory symptoms and signs.

1 Kelly J, Thorning G, Ozzard A, *et al.* Back pain and dyspnoea in a middle aged diabetic male. *Postgrad Med J* 2000;76:239–40, 245–8.
2 Levin HS, Runco V, Wooley CF, *et al.* Pulmonic regurgitation following staphylococcal endocarditis. *Circulation* 1964;30:411–6.
3 Cremieux A, Witchitz S, Malergue M, *et al.* Clinical and echocardiographic observations in pulmonary valve endocarditis. *Am J Cardiol* 1985;56:610–3.
4 Fourestie V, Benvenuti C, LeJonc JL, *et al.* Fatal acute circulatory failure in pulmonary valve endocarditis. *Am J Cardiol* 1986;57:895.
5 Calleja F, Eguaras MG, Chacon A, *et al.* Pulmonary valve endocarditis during puerperal sepsis. *J Cardiovasc Surg* 1992;33:292–4.
6 Weiss RJ, LeMire MS, Bajor M, *et al.* Two-dimensional echocardiographic detection of pulmonic valve endocarditis. *J Clin Ultrasound* 1982;10:451–3.
7 Faruqui AMA, Silverman ME. Isolated acqired pulmonary valve regurgitation: report of long-term follow-up. *Br Heart J* 1978;40:198–200.
8 Dzindzio BS, Meyer L, Osterholm R, *et al.* Isolated gonococcal pulmonary valve endocarditis: diagnosis by echocardiography. *Circulation* 1979;59:1319–24.

Heart

Community acquired staphylococcal pulmonary valve endocarditis in non-drug users: case report and review of the literature

J J Edmond, S J Eykyn and L D R Smith

Heart 2001 86: e17
doi: 10.1136/heart.86.6.e17

Notes