Atopic eczema and staphylococcal endocarditis: time to recognize an association?

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Endocarditis caused by *Staphylococcus aureus* is a particularly fulminant form of the disease which carries a mortality rate of up to 47% (Brydie and Clark, 1999). Atopic eczema is a common skin disease affecting 1–3% of the population which is recognized to be associated with *S. aureus* skin colonization in 80–90% of affected individuals (Abeck and Mempol, 1998). Furthermore, *S. aureus* is the commonest organism causing infective exacerbation of eczema (David et al, 1986). Despite this clear link between atopic eczema and *S. aureus* skin infection we could only find one previous report (in a child with a neutrophil chemotactic defect) of acute *S. aureus* endocarditis complicating infected atopic eczema (Pike and Warner, 1989).

We present two cases of community-acquired *S. aureus* endocarditis of the mitral valve in patients with atopic eczema with no history of intravenous drug use or other identifiable skin portal of entry for *S. aureus*.

DISCUSSION

S. aureus infection is associated with increased severity of atopic eczema and topical or systemic antistaphylococcal measures are used in the treatment of exacerbations of the disease. Intact skin and mucous membranes form an important part of the natural host defence against *S. aureus*. In both of our cases atopic eczema had been present for many years and both had evidence of excoriated skin on presentation, thus allowing a breakdown of the natural cutaneous barrier.

The patient in case 1 had worn a dental brace for 2 years, but also had widespread eczematous skin lesions. Although nasal carriage rates of *S. aureus* are higher in individuals with skin diseases such as eczema (Murphy, 1996), skin infection has been shown to be the most common entry route of *S. aureus* in endocarditis with the dental route surprisingly rare (Frimodt-Moller et al, 1983).

The patient in case 2 described an exacerbation of eczema 2 weeks before presentation and was known to have longstanding mitral valve prolapse with moderate mitral regurgitation. Case 1 had severe mitral valve prolapse secondary to infection. Mild prolapse may previously have been present, as left-sided staphylococcal endocarditis is rare in the absence of

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CASE REPORT 1

An 18-year-old man presented with a 3-day history of malaise, pyrexia, confusion and left knee pain. He had a history of atopic eczema since the age of 6 months but was otherwise well. He had worn a dental brace for the past 2 years without complications and had no recent dental intervention. There was no history of intravenous drug abuse.

On examination he was pyrexial at 39.0°C, clinically dehydrated, with a sinus tachycardia of 100 beats per minute and a systemic blood pressure of 130/80 mmHg. He had eczematous lesions on his face, arms and legs. Auscultation revealed no cardiac murmurs and lung fields were clear. There were no stigmata of endocarditis. He was mentally obtunded with a Glasgow Coma Score of 14/15 but had no other neurological signs. The left knee demonstrated a full range of movement and no obvious effusion. Orthopaedic opinion was of a reactive arthritis.

Chest and left knee radiography was unremarkable. C-reactive protein (CRP) was elevated at 251mg/litre, haemoglobin was 12.4g/dl, leukocytes 11.7×10^9/litre (with 89% neutrophils) and platelets 26×10^9/litre. A screen for disseminated intravascular coagulopathy was negative. He was hyponatraemic (sodium 125 mmol/litre) and mildly uraemic (urea 7.8 mmol/litre, creatinine 91μmol/litre).

Blood cultures grew *Staphylococcus aureus* sensitive to flucloxacillin and gentamicin. Computed tomography (CT) of the brain showed generalized cerebral swelling with effacement of the basal cisterns, but no focal abnormality. Treatment was initiated with intravenous flucloxacillin, gentamicin and fluid replacement.

The following day he developed severe pulmonary oedema and haemodynamic compromise necessitating admission to the intensive care unit for inotropic support and ventilation. Urgent transoesophageal echocardiography showed a 2x2 cm vegetation on the anterior mitral valve leaflet (*Figure 1*) with marked prolapse and severe mitral regurgitation. There was systolic flow reversal in the pulmonary veins. Left atrial size was normal and left ventricular function good.

He underwent emergency mitral valve replacement with a St Jude mechanical valve (St Jude Medical Inc, St Paul, Minnesota, USA). At operation there was seen to be almost complete destruction of the anterior mitral valve leaflet. His postoperative recovery was good, completing 6 weeks of antibiotic therapy, and repeat CT showed resolution of the cerebral oedema. During the admission he experienced an exacerbation of his eczema and was treated with topical steroids and emollients by the dermatologists.

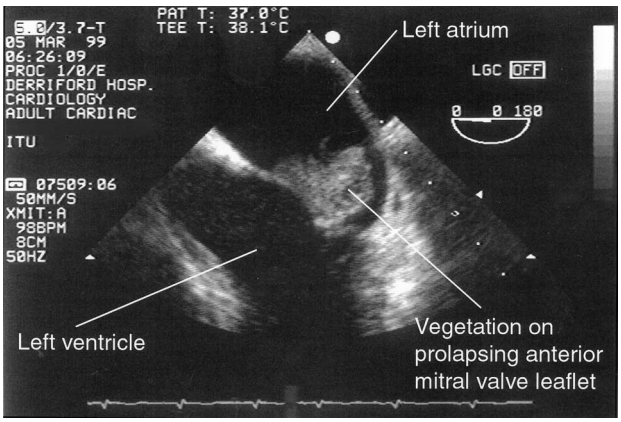

Figure 1. Transoesophageal echocardiogram showing a large vegetation on the anterior leaflet of the mitral valve.

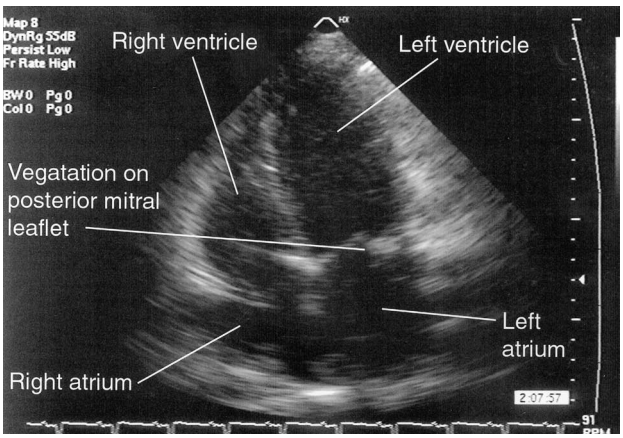

Figure 2. Transthoracic echocardiogram showing a vegetation on the posterior leaflet of the mitral valve.

pre-existing structural or valvular heart disease (Hricak et al, 1998).

Mitral valve prolapse, when associated with thickened leaflets or valvular regurgitation, is recognized as a moderate risk factor for endocarditis with antibiotic prophylaxis recommended during dental and other infective risk procedures (Dajani et al, 1997). However, effective endocarditis prophylaxis should also involve sensible non-chemotherapeutic methods: improved personal hygiene, skin care and gum care, as well as patient education to promote awareness of endocarditis risk, are equally important in patients with known structural cardiac abnormalities predisposing to endocarditis (Dodo and Child, 1996).

CONCLUSION

We have described two patients with acute *S. aureus* endocarditis of the mitral valve in association with excoriated skin lesions of long-standing atopic eczema. The association between atopic eczema, *S. aureus* skin colonization and subsequent infection is well recognized but we are unaware of any previous case reports, in immunocompetent adults, of resultant *S. aureus* endocarditis.

We believe that atopic eczema in patients with valvular or structural heart disease represents a potential risk factor for *S. aureus* endocarditis. Physicians caring for such patients and patients themselves should be aware of this possible association, and closer attention to skin care plus appropriate antistaphylococcal measures may be advisable. HM

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CASE REPORT 2

A 45-year-old man was transferred from a peripheral hospital having been admitted 1 week previously with a 3-day history of rigors, malaise and anorexia. He had a history of asthma, atopic eczema, hypertension and moderate mitral regurgitation secondary to posterior mitral valve leaflet prolapse. He had experienced a worsening of his eczema 2 weeks before admission.

Blood cultures had grown *Staphylococcus aureus* but, despite appropriate intravenous therapy with flucloxacillin and gentamicin, his C-reactive protein (CRP) level remained elevated and he had developed worsening mitral regurgitation. Transthoracic echocardiography at the referring hospital had confirmed a large vegetation on the posterior mitral valve leaflet with severe prolapse and mitral regurgitation.

On arrival at our hospital he was apyrexial but had a sinus tachycardia of 110 beats per minute. There was evidence of severe mitral regurgitation with a gallop rhythm and an apical systolic thrill. Blood pressure was satisfactory at 135/90 mmHg and lung fields were clear. He had numerous peripheral emboli, splinter haemorrhages and extensive eczema an his arms, face and legs with evidence of excoriation.

Routine biochemistry and haematology were satisfactory apart from a mild neutrophilia. CRP was elevated at 85 mg/litre. Transthoracic echocardiography confirmed severe mitral regurgitation with a 1.5x1.5 cm vegetation on a severely prolapsing posterior mitral valve leaflet (*Figure 2*). In view of the severe mitral regurgitation and persistently elevated CRP he underwent emergency mitral valve replacement with a CarboMedics mechanical valve (CarboMedics Inc, Austin, Texas, USA). Intraoperative transoesophageal echocardiography confirmed the transthoracic findings. He made an uncomplicated recovery and completed 6 weeks antibiotic therapy.