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Case Report

CASE REPORT

Tricuspid valve infective endocarditis in a non-IVDU patient with atopic dermatitis

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Abstract

A 29-year-old male, with chronic atopic dermatitis (AD), presented with a 2-week history of fatigue, pyrexia and weight loss. Examination showed eczematous patches with lichenified papules, erosions on the right shin and a new murmur. Blood cultures isolated methicillin-sensitive *Staphylococcus aureus*. Transthoracic echocardiography showed vegetation on the tricuspid valve (TV) that was adherent to the septal leaflet. He was treated for infective endocarditis, attributed to poorly controlled AD, with intravenous Flucloxacillin. Due to ongoing sepsis and pulmonary septic emboli, Clindamycin was added. He underwent TV repair; the septal leaflet was excised, and the remnant two leaflets were brought together with a ring. His patent foramen ovale was closed. His skin was treated with topical steroids and emollients. Right-sided endocarditis of an intact TV is uncommon in a non-intravenous drug user. Therefore, this novel case portrays the importance of aggressively managing AD as it is a risk factor for significant systemic infections.

INTRODUCTION

Atopic dermatitis (AD) affects 1–3% of the population and is seen in all age groups. Antimicrobial, antifungal and antiviral peptides, induced by inflammation, are deficient in patients with atopic dermatitis, and there is skin colonisation by *Staphylococcus aureus* in more than 90% of patients. This increases the risk of invasion of bacteria through the interrupted skin barrier, subsequently promoting bacteraemia and extracutaneous infections including endocarditis, meningitis, encephalitis, bone and joint infections and sepsis [1–5]. We discuss a case of infective endocarditis in a 29-year-old male with AD and no history of recreational drug use.

CASE REPORT

A 29-year-old male presented with a 2-week history of fatigue, headache, fever and weight loss. He had AD since childhood with recurrent exacerbations that were managed topically by his general practitioner. He was not on regular medications and had no known drug allergies, but he had a history of hay fever and family history of atopy. He never smoked, did not drink alcohol and had never used recreational drugs. Prior to admission, he developed an acute AD flare-up that was poorly controlled.

On presentation, he was pyrexial at 38.9°C with a blood pressure of 128/84 mmHg, respiratory rate of 20 breaths per minute and heart rate of 100 beats per minute. His AD affected his upper

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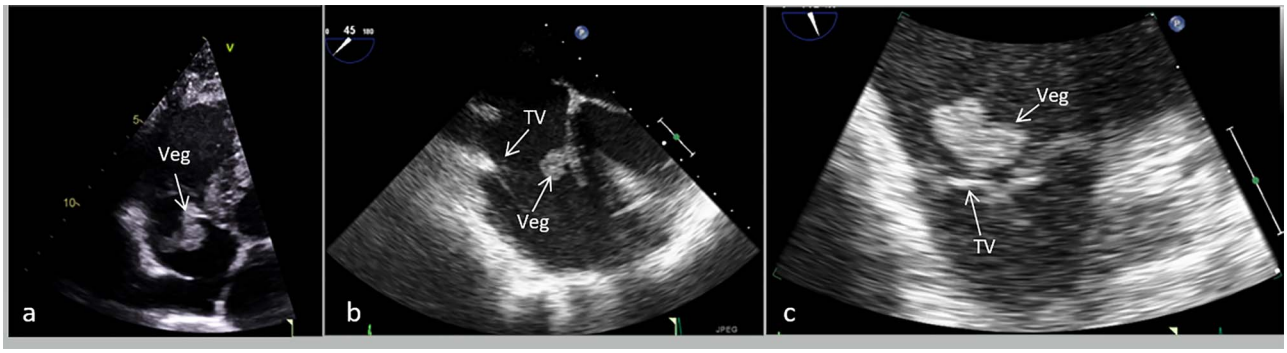

Figure 1: Pre-operative echocardiographic images showing tricuspid valve vegetation (**a**,**b**,**c**).

Modified Duke Criteria for diagnosing infective endocarditis.

Major criteria

1. **Blood culture positive for infective endocarditis (IE)**
   a. Typical microorganisms consistent with IE from two separate blood cultures:
      i. *Viridans streptococci, Streptococcus bovis,* HACEK group, *Staphylococcus aureus*; or
      ii. Community-acquired enterococci, in the absence of a primary focus; or
   b. Microorganisms consistent with IE from persistently positive blood cultures, defined as follows:
      i. At least two positive cultures of blood samples drawn ≥12 hours apart; or
      ii. All of three or a majority of four separate cultures of blood (with first and last sample drawn at least 1 hour apart)
   c. Single positive blood culture for *Coxiella burnetii* or antiphase I IgG antibody titer ≥1:800
2. **Evidence of endocardial involvement**
3. **Echocardiogram positive for IE defined as follows:**
   a. Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation; or
   b. Abscess; or
   c. New partial dehiscence of prosthetic valve
4. **New valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)**

Minor criteria

1. Predisposition: predisposing heart condition, or injection drug use
2. Fever: temperature >38°C
3. Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway lesions
4. Immunologic phenomena: glomerulonephritis, Osler's nodes, Roth's spots, and rheumatoid factor
5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE
6. Echocardiographic minor criteria eliminated

Interpretation

Definite infective endocarditis
- Pathologic criteria:
  - Microorganisms demonstrated by culture or histologic examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen; or
  - Pathologic lesions; vegetation, or intracardiac abscess confirmed by histologic examination showing active endocarditis
- Clinical criteria:
  - Two major criteria; or
  - One major criterion and three minor criteria; or
  - Five minor criteria

Possible infective endocarditis
- Clinical criteria
  - One major criterion and one minor criterion; or
  - Three minor criteria

Diagnosis of infective endocarditis is rejected
- Firm alternate diagnosis explaining evidence of infective endocarditis; or
- Resolution of infective endocarditis syndrome with antibiotic therapy for ≤4 days; or
- No pathologic evidence of infective endocarditis at surgery or autopsy, with antibiotic therapy for ≤4 days; or
- Does not meet criteria for possible infective endocarditis, as above

Figure 2: Modified Duke criteria for diagnosing infective endocarditis [6].

and lower limbs as hyperpigmented eczematous areas with lichenification, excoriation and xerosis with crusted erosion on the right shin. He had no peripheral stigmata of infective endocarditis, but he had a systolic murmur over the tricuspid area.

His electrocardiogram showed sinus tachycardia with a normal PR interval. C-reactive protein level and white cell counts were raised at 248 mg/L and 14.9×10^9 cells/L, respectively, and blood cultures isolated methicillin-sensitive *Staphylococcus aureus* (MSSA). An urgent transthoracic echocardiogram (TTE) showed a large mobile structure (Fig. 1) that appeared to be attached to the tricuspid valve's septal leaflet. This mass prolapsed between the right atrium and right ventricle. There was mild TV regurgitation, a small patent foramen ovale (PFO) and good left and right ventricular systolic functions.

He was diagnosed with definitive infective endocarditis (Fig. 2: meeting all major criteria and minor criteria 2,3) and MSSA bacteraemia, clinically felt to be attributed to his dermatitis (Fig. 3). He was urgently referred to the dermatology team who advised treatment with potent steroid cream, emollients and antimicrobial washes.

He was started on intravenous 2 g Flucloxacillin four times daily (QDS). He suddenly developed dyspnoea, chest pain and haemoptysis. Chest computed tomography (CT) confirmed acute pulmonary emboli, in keeping with septic emboli and infarcts (Fig. 4). Anticoagulation was not advised initially due to the risk of haemorrhagic transformation. Nine days later, Clindamycin 600 mg QDS was added because of ongoing pyrexia. Due to a positive sputum culture, Flucloxacillin was escalated to Meropenem.

Surgery was initially delayed because of chest sepsis; however, because of ongoing pleuritic chest pain and haemoptysis, it was undertaken on day 17. He underwent tricuspid valve repair and annuloplasty with a 28-mm Carpentier Edwards physic annuloplasty ring. The defect in the septal leaflet was closed directly, and the resultant gap was closed by moving the posterior leaflet onto the septum. This left the new valve functionally bicuspid (Fig. 5). The vegetation included part of the papillary muscle that had ruptured. There was a right to left shunt via the small PFO, which spontaneously closed.

Meropenem was de-escalated to Flucloxacillin 3 days postoperatively. Repeat echocardiogram showed good valve function, reasonable left ventricular systolic function, but impaired right systolic function and mild tricuspid regurgitation. Since postoperative CRP levels remained static, CT chest was repeated. A significant increase in embolic burden and infarction was noted, and a clinical decision was made to start anticoagulation.

Repeat blood cultures were negative and he remained apyrexial. An echocardiogram, 1 month later, showed improving right ventricular function. His inpatient stay was complicated by a subsequent hospital acquired pneumonia and Influenza A. Upon completion of 6 weeks of Flucloxacillin and Clindamycin, his CRP level was 17 mg/L. He was discharged on Apixaban for 3 months and emollients with local dermatology follow-up.

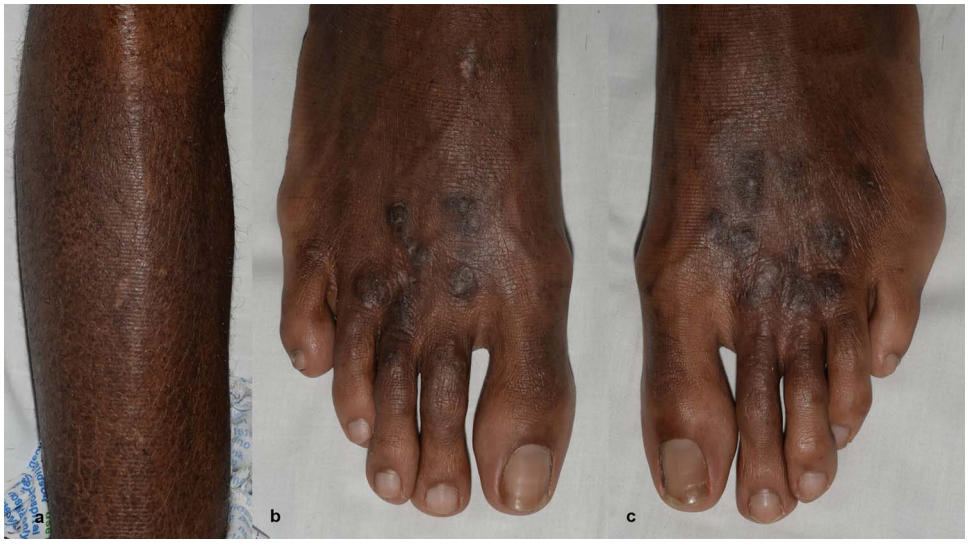

Figure 3: Atopic dermatitis, excoriation and erosion in right shin secondary to xerosis and chronic itching (**a**) and symmetrical lichenification on both feet (**b** and **c**).

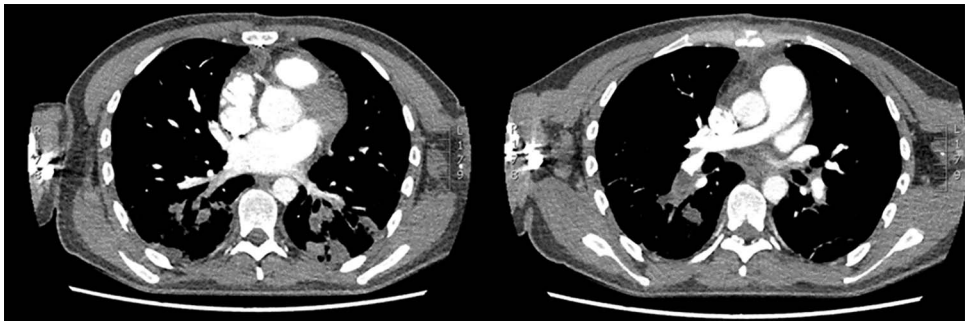

Figure 4: CT scan showing pulmonary emboli in left and right pulmonary arteries (**a**,**b**).

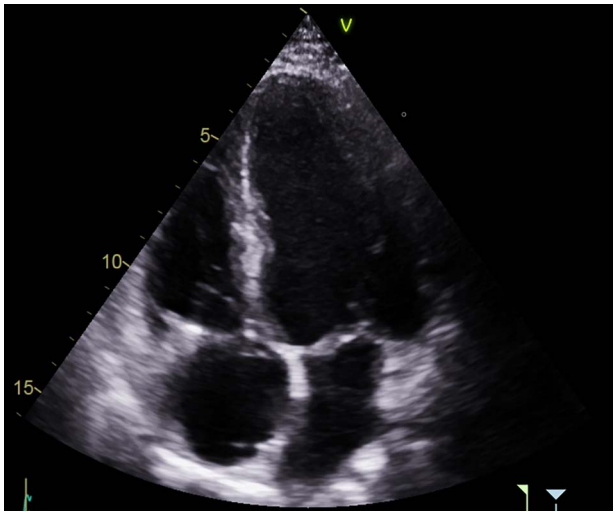

Figure 5: Post-operative echocardiographic images showing repaired tricuspid valve.

DISCUSSION

Infective endocarditis (IE) is associated with high morbidity; its in-hospital mortality rate varies from 15 to 30%. The literature relating atopic dermatitis to IE keeps expanding and yet, patients are not receiving optimal dermatological treatment and appropriate referral [7]. Indeed, Aoyagi et al. [5] identify 24 cases of infective endocarditis in patients with atopic dermatitis; median age was 27.5 years old (similar to our patient), younger compared to the median age of IE in the general population. Micallef et al. [3] report a similar case to ours with the mitral valve being affected, and Tsuboi et al. [8] report a patient with AD having *Staphylococcus aureus* bacteraemia complicated by psoas abscess and IE.

About 5–10% of IE cases are right sided and are most commonly seen in intravenous drug users and immunosuppressed patients. Immunosuppression, the presence of cardiac effects or previous valvuloplasty are recognised risk factors. Our patient was healthy beyond the atopic dermatitis, with no history of recreational drug use. This supports the evidence that AD is an independent risk factor for IE.

Complications of IE include embolic events, which can occur in 20–50% of patients. Aoyagi et al. [5] report that 62.5% of their 24 cases developed embolic events, 50% of which were cerebral. Our patient developed pulmonary emboli with recurrent haemoptysis. The decision to anticoagulate was challenging, but he was started on therapeutic Dalteparin and then Apixaban.

Recognising infective endocarditis and its source is essential for appropriate management. When patients with AD present with prolonged pyrexia, IE should be ruled out including metastatic spread, as per Tsuboi et al. [8]

Therefore, we strongly recommend the early involvement of dermatologists in patients with uncontrolled atopic dermatitis to achieve optimum control and prevent complications. Topical

steroids and oral immunosuppressants are established treatments. Dupilumab is a safe and effective biologic treatment for AD used for refractory cases [9]. Nakanishi et al. [10] support treatment of AD with Dupilumab 2 months before elective cardiac surgery for a case of IE. It is also important to educate patients to seek medical attention when their skin condition flares up. Early use of antibiotics for active infections or prophylactic antimicrobial washes could potentially be considered.

SUPPLEMENTARY MATERIAL

Supplementary material is available at the *Oxford Journal of Medical Case* Reports online.

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ETHICAL APPROVAL

Ethical approval was not required.

CONSENT

Consent was obtained from the patient using the Oxford University Press Consent Form and the identity has been protected.

GUARANTOR

Dr Heerani Woodun. This manuscript has not been published and is not under consideration for publication elsewhere. All the authors have read the manuscript and have approved this submission.

CONFLICT OF INTEREST STATEMENT

We declare no conflict of interest.

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