Recurrent Prosthetic Valve Endocarditis Caused by *Staphylococcus aureus* Colonizing Skin Lesions in Severe Atopic Dermatitis

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Abstract

Infective endocarditis, a serious infection most commonly affecting rheumatic or prosthetic valves, generally occurs after bacteremia. Atopic dermatitis, a very common disease, carries a high prevalence of skin infections, particularly with *Staphylococcus aureus*. While cutaneous colonization by *S. aureus* represents an important source of bacteremia, few cases of infective endocarditis arising from the skin lesions of atopic dermatitis have been reported. We describe a patient with recurrent *S. aureus* prosthetic valve endocarditis developing in this manner.

Key words: atopic dermatitis, *Staphylococcus aureus*, prosthetic valve endocarditis, skin lesion colonization

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Introduction

Infective endocarditis, a potentially devastating infection that most commonly affects rheumatic or prosthetic valves, typically complicates bacteremia associated with dental procedures, long-term indwelling venous catheters, or drug abuse. Atopic dermatitis is a very common disease often associated with infection of skin lesions, particularly by *Staphylococcus aureus*. (1) This cutaneous colonization by *S. aureus* is a potent source of bacteremia. (2) None of the few reports concerning infective endocarditis arising from skin lesions of atopic dermatitis (3-9) have confirmed identity between blood culture and skin isolates, which we recently carried out in a patient with recurrent prosthetic valve endocarditis and atopic dermatitis lesion colonization by *S. aureus*.

Case Presentation

A 27-year-old man who presented in March 2002 with shortness of breath was diagnosed with active infective endocarditis causing severe mitral regurgitation, requiring a

porcine mitral valve replacement. The organism causing endocarditis was *S. aureus*. Three episodes of prosthetic valve endocarditis involving *S. aureus* occurred over the subsequent 2 years, without obvious precipitating causes. In July 2004 pulmonary edema occurred as a manifestation of the fourth episode of prosthetic valve endocarditis, associated with methicillin-resistant *S. aureus* bacteremia; porcine valve replacement was repeated. Shortly after that hospitalization, the patient was admitted with a fifth occurrence of prosthetic valve endocarditis, in October 2004. On admission his temperature was 39.5°C; blood pressure, 130/80 mm Hg; and pulse rate, 124 beats/min. No murmurs, gallops, or friction rubs were audible. The lungs were clear to auscultation. Electrocardiography showed normal sinus rhythm, with no notable abnormality. Chest radiography showed a mildly enlarged cardiac silhouette with no sign of pulmonary congestion; the cardiothoracic ratio was 55.0%. Transthoracic echocardiography showed mobile vegetations on the prosthetic mitral valve (Fig. 1). Blood cultures grew *S. aureus*. Treatment with antimicrobials was begun, using intravenous cefazolin and oral rifampicin. However, vegetations continued to show daily enlargement, and a third mitral valve replacement was performed on the 10th hospital day. Massive vege-

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Table 1. Case Reports of Patients with Infective Endocarditis and Atopic Dermatitis

Case	Author	Age (y) /gender	Blood culture	Valve involved	Surgical treatment
1	Pike ³	3/M	<i>S. aureus</i>	Tricuspid	Vegetectomy, VSD repair
2	Grabczynska ⁴	24/M	MSSA	NA	(-)
3	Kobayashi ⁵	33/M	MSSA	Aortic	Valve replacement
4	Conway ⁶	18/M	MSSA	Mitral	Valve replacement
5	Conway ⁶	45/M	MSSA	Mitral	Valve replacement
6	Onoda ⁷	16/M	MRSA	Aortic	Valve replacement, annulus repair
7	Onoda ⁷	14/M	<i>S. aureus</i>	Mitral	Vegetectomy, commissure repair
8	Satchell ⁸	50/F	<i>S. aureus</i>	Mitral	Valve repair
9	Benenson ⁹	36/F	MSSA	Mitral	Valve replacement
10	Current case	27/M	MSSA	Mitral (PV)	Valve replacement

S. aureus, *Staphylococcus aureus* with antibiotic sensitivity not stated; VSD, ventricular septal defect; MSSA/MRSA, methicillin-sensitive/resistant *Staphylococcus aureus*; NA, information not available; PV, prosthetic valve.

Figure 1. Transthoracic parasternal long-axis image showing the mobile vegetation attached to the mitral prosthesis.

tations were attached to all the leaflets of the mitral bioprosthetic valve. The patient had longstanding atopic dermatitis since childhood, which had shown recent worsening and had not responded to topical steroids. We suspected colonized atopic dermatitis lesions as a source of endocarditis. A culture sample from the skin grew *S. aureus*, showing the same antimicrobial susceptibilities as the blood culture isolates. Pulsed-field gel electrophoresis (PFGE; BML, Saitama, Japan) demonstrated identity of strain between blood and the skin cultures by identical numbers and apparent sizes of bands (Fig. 2). Blood cultures after operation did not grow *S. aureus*. Echocardiography showed no vegetations on the prosthetic valve and the level of C-reactive protein was negative, after a 6-week course of antimicrobial therapy. A dermatologist has maintained careful follow-up for management of atopic dermatitis. The patient remained free of evi-

Figure 2. Pulsed-field gel electrophoresis patterns of chromosomal DNA from our patient. Isolates from blood (1) and skin (2) cultures have indistinguishable band patterns and are considered to represent the same strain. M, lambda ladder markers; C, control strain NCTC8325.

dence of recurrent endocarditis at 18 months after discharge.

Discussion

Here, we report multiple recurrences of prosthetic valve endocarditis caused by *S. aureus* derived from colonized skin lesions in a patient with severe atopic dermatitis. *S. aureus* may colonize lesions in most patients with atopic dermatitis (2); a deficiency in expression of endogenous antimicrobial peptides in these patients' skin, causing localized immunodeficiency, may account for their susceptibility to staphylococcal skin invasion. (10) Yet, only nine cases of infective endocarditis derived from cutaneous infection in atopic dermatitis have been reported in the English language (Table 1). Further, the present case is the first to be confirmed by a molecular method, in this case, PFGE, to involve identical isolates in skin and blood cultures. PFGE is

a promising tool for differentiating various *S. aureus* strains by identifying a genetic pattern unique to each isolate (11). In view of the high bacterial density on atopic dermatitis skin and a disturbed epidermal barrier with multiple breaches, the possibility of artifactual blood culture contamination by skin flora can never be excluded despite thorough skin disinfection prior to venipuncture. However, *S. aureus* with the same antimicrobial susceptibility profile as that of the skin isolate was isolated repeatedly from blood cultures. Benenson et al (9) suspect that atopic dermatitis-associated invasive *S. aureus* infection is probably underreported rather than uncommon. In particular, uncontrolled atopic dermatitis would represent a risk factor for invasive *S. aureus* infections such as endocarditis. We concur that severe atopic dermatitis lesions should be recognized as a likely bacterial source for infective endocarditis.

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