

Seizures in a Patient With Eczema

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Case Report

A 17-year-old female with a past medical history of bronchial asthma, bipolar disorder and severe atopic dermatitis was evaluated in the emergency department (ED) for 1 episode of generalized tonic-clonic seizure (GTC). The seizure episode was preceded by a 2-day history of fatigue and frontal headache. Her initial vital signs were stable except of a temperature of 38°C. Cardiovascular examination revealed normal heart sounds with no evidence of a heart murmur, gallop, or pericardial rub. Laboratory tests showed a normal white cell counts and differentials, hemoglobin of 9.9 g/dL, and platelets of 61 000. Blood urea nitrogen and creatinine were elevated at 34 mg/dL and 1.4 mg/dL, respectively. Lactate dehydrogenase and creatinine kinase were also elevated at 433 and 788. Cerebral spinal fluid analysis was negative. Blood, urine, and cerebrospinal fluid cultures were obtained and the patient was started empirically on ceftriaxone and vancomycin; awaiting the final cultures results.

The initial chest radiograph and head computed tomography (CT) were negative. A transthoracic echocardiogram (TTE) showed normal intracardiac anatomy, normal valve morphology, no intracardiac shunts, normal aortic arch, and normal Doppler across all the valves. There was no pericardial effusion and biventricular function was normal. The patient was admitted to the general pediatric floor where she became hypotensive, tachypneic, and hypoxic despite being on a non-rebreather (NRB) mask. A repeat chest radiograph showed massive pulmonary edema, and she was urgently transferred to the intensive care unit (ICU) where she was intubated and started on mechanical ventilation. On admission to the ICU, she was febrile with a temperature of 38°C, her blood pressure was 97/54 mm Hg, with a pulse rate of 89 beats/min, and with an oxygen saturation of 99% on 60% FiO₂ via mechanical ventilation. On cardiac auscultation there was normal S1 and S2 and a grade 3/6 holosystolic murmur heard at the apex and radiating to the axilla. There was no diastolic murmur, gallop, or pericardial rub. Coarse breath sounds were heard in both sides of lung. Her skin examination revealed extensive dryness and scarring over her entire body, with small purple nodules noted on the soles of her feet and the tips of her fingers.

Laboratory tests obtained on admission showed anemia with hemoglobin of 9.6 mg/dL, thrombocytopenia with platelet count of 55 000. Blood urea nitrogen and creatinine were elevated at 34 mg/dL and 1.1 mg/dL, respectively. Troponin I was elevated at 0.95. Her blood and urine cultures grew methicillin-resistant *Staphylococcus aureus* (MRSA).

An electroencephalogram (EEG) showed evidence of severe neurological dysfunction consisting of slow background activity during the awake state with poorly formed sleep spindles during sleep. Head and neck magnetic resonance imaging/angiogram (MRI/MRA) with contrast showed multiple cortical and subcortical infarcts with the largest measuring 4.4 cm in the right frontoparietal region. A repeat TTE 3 days after the initial study showed multiple vegetations on the anterior and posterior leaflets of the mitral valve. There was moderate mitral regurgitation with mitral valve stenosis. There was no pericardial effusion, and she had normal left ventricular function (Figures 1-5).

The diagnosis of infective endocarditis (IE) was considered “definitive” according to the modified Duke criteria, meeting 2 of the major criteria. Vancomycin was continued and ceftriaxone was switched to cefepime for broad antimicrobial coverage. She was also started on levetiracetam per neurology recommendations. A brain CT scan ruled out intracranial hemorrhage before she was taken to the operative room. The surgery was planned for the infective endocarditis due to the presence of heart failure symptoms refractory to medical management, locally uncontrolled infection, and multiple embolic events.

Her preoperative transesophageal echocardiography showed large vegetations involving mitral valve leaflets with severe mitral valve insufficiency (Figures 6 and 7). Surgical exploration revealed large, mobile vegetations along the free edge of the anterior mitral valve leaflet with a large abscess at the posterolateral commissure and several small abscesses along the posterior leaflet with involvement of the chordae and the distal tips of the

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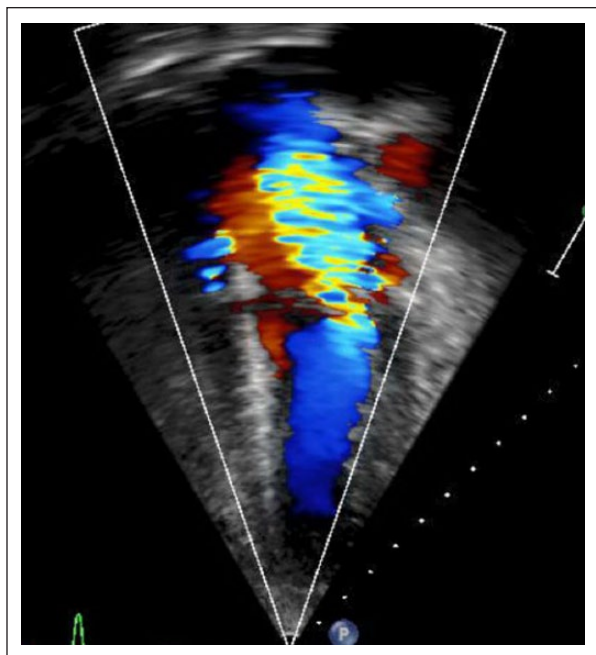


Figure 1. Transthoracic echocardiogram revealing severe mitral regurgitation.

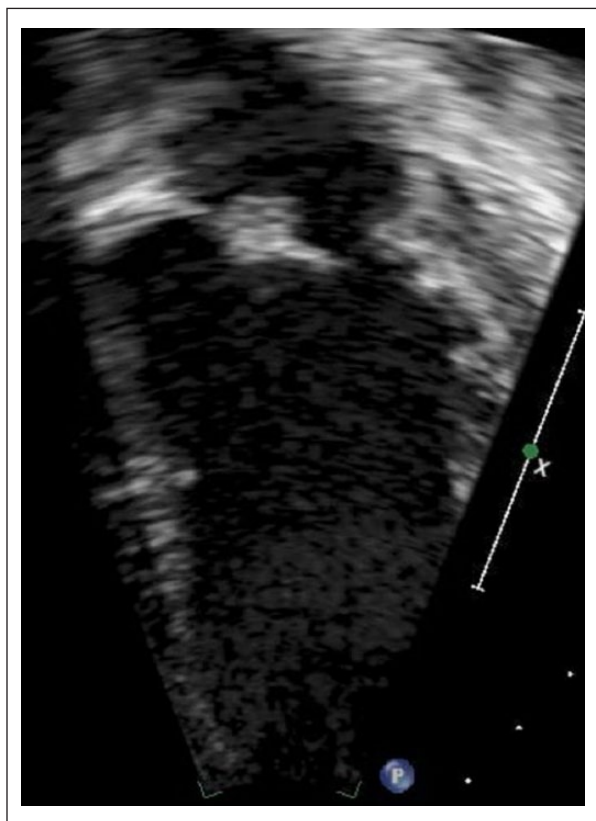


Figure 2. Transthoracic echocardiogram revealing vegetation in the anterior leaflet of the mitral valve.

papillary muscles. There was no involvement of the mitral valve annulus or the conduction area. Surgical repair included debridement of the mitral valve abscesses, and mitral valve replacement with a 31-mm St Jude mechanical valve in the supra-annular position.

Postoperatively, her peripheral blood culture continued to grow staph aureus, therefore her antibiotics were changed to cover a possible MRSA prosthetic valve endocarditis to intravenous cefepim, daptomycin, gentamycin, and levofloxacin. She was extubated 6 days after surgery with gradual recovery of her mental and functional status. She followed an intensive program of physical and respiratory rehabilitation, which resulted in good recovery with some residual left-sided weakness, changes in speech, and difficulties finding words. She was discharged home after 3 weeks of hospitalization. Her discharge echocardiogram showed normal functioning prosthetic mitral valve with no stenosis or regurgitation. She continued to have normal left ventricular function. Three weeks after her discharge she was admitted to an outside hospital with fatigue, stomach, and back pain. While trying to get a CT scan for her head, she arrested and she was resuscitated but never regained consciousness and subsequently passed away.

Final Diagnosis

The final diagnosis was embolic stroke secondary to mitral valve MRSA endocarditis.

Discussion

Atopic dermatitis is one of the most common chronic inflammatory skin diseases, affecting up to 20% of children and 1% to 3% of adults.¹ Atopic dermatitis is a common skin condition in children but very few cases are reported when this condition is associated with acute native valve endocarditis. Colonization of *S aureus* is commonly observed in skin lesions of atopic dermatitis patients, and scratching of the pruritic lesions may lead to reiterative bacteremia and endocarditis (see Figure 8). In native valve endocarditis, *S aureus* accounts for 30% to 35% of cases, whereas in a patient with atopic dermatitis, it is found to be exclusively due to *S aureus*. This could be the result of frequent staphylococcal bacteremia in patients with atopic dermatitis.²

The combination of antimicrobial peptides known as cathelicidins (LL-37) and beta-defensins (HBD-2) is shown to have synergistic antimicrobial activity in the body for effective killing of *S aureus*. Ong et al³ suggested that a deficiency in the expression of those peptides in the atopic skin may account for the susceptibility of patients with atopic dermatitis to skin infection with *S aureus*.

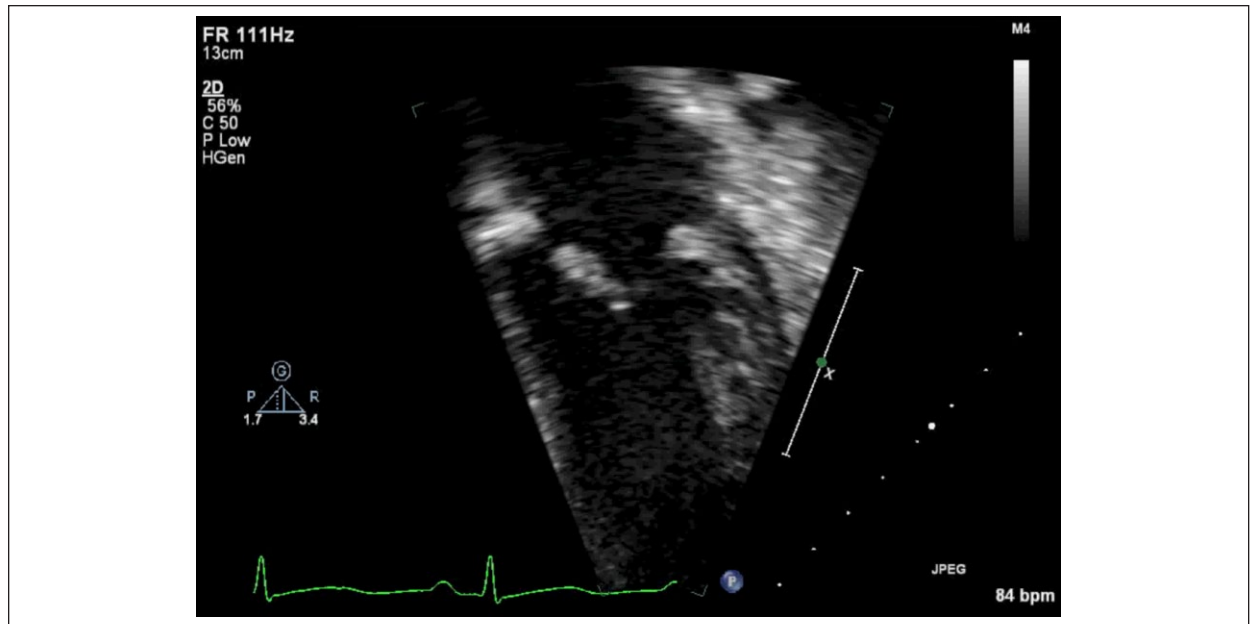


Figure 3. Transthoracic echocardiogram – Apical view revealing vegetation in the anterior and posterior mitral valve leaflet.

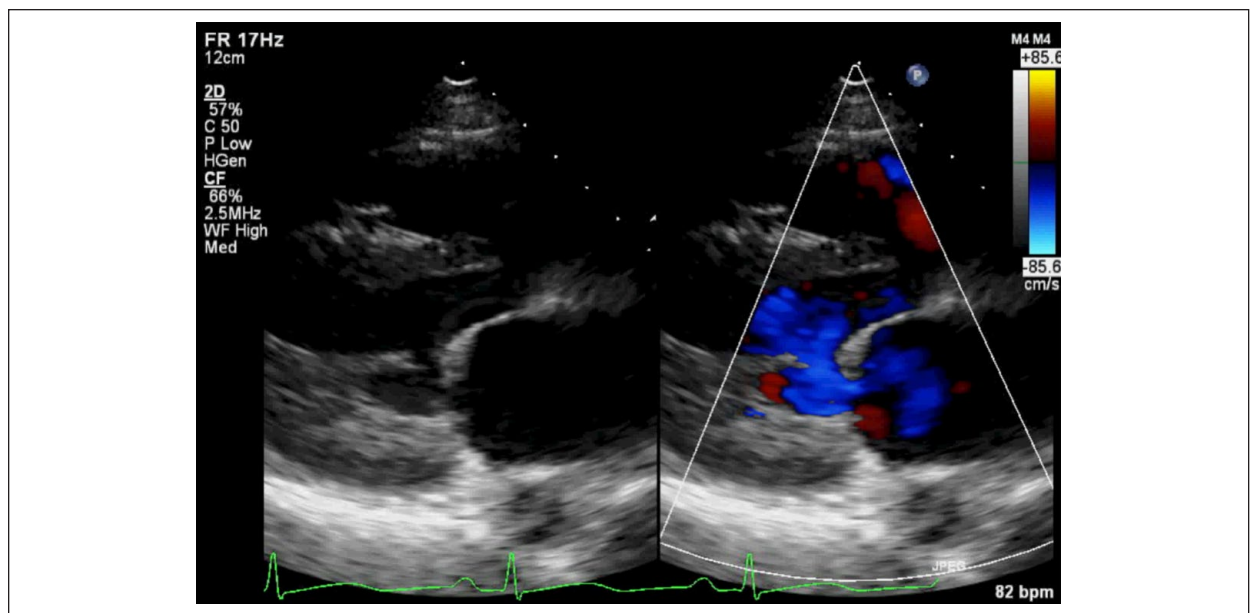


Figure 4. Transthoracic echocardiogram – Parasternal long axis view revealing vegetation in the anterior and posterior mitral valve leaflet.

Recently, an association between allergic disease and epilepsy in childhood was reported. Silverberg et al⁴ found that children with ≥ 1 allergic disease had a higher odds of epilepsy compared with nonallergic children in a US population-based study. They also found that severe atopic dermatitis was associated with even higher risk of epilepsy compared with mild/moderate disease.⁴

Chen et al⁵ also reported an increased incidence of epilepsy in atopic dermatitis cohort when compared to their control in Taiwan.

We performed review of the literature regarding atopic dermatitis and infective endocarditis in children. A total of 4 cases (including ours) were reported (Table 1).

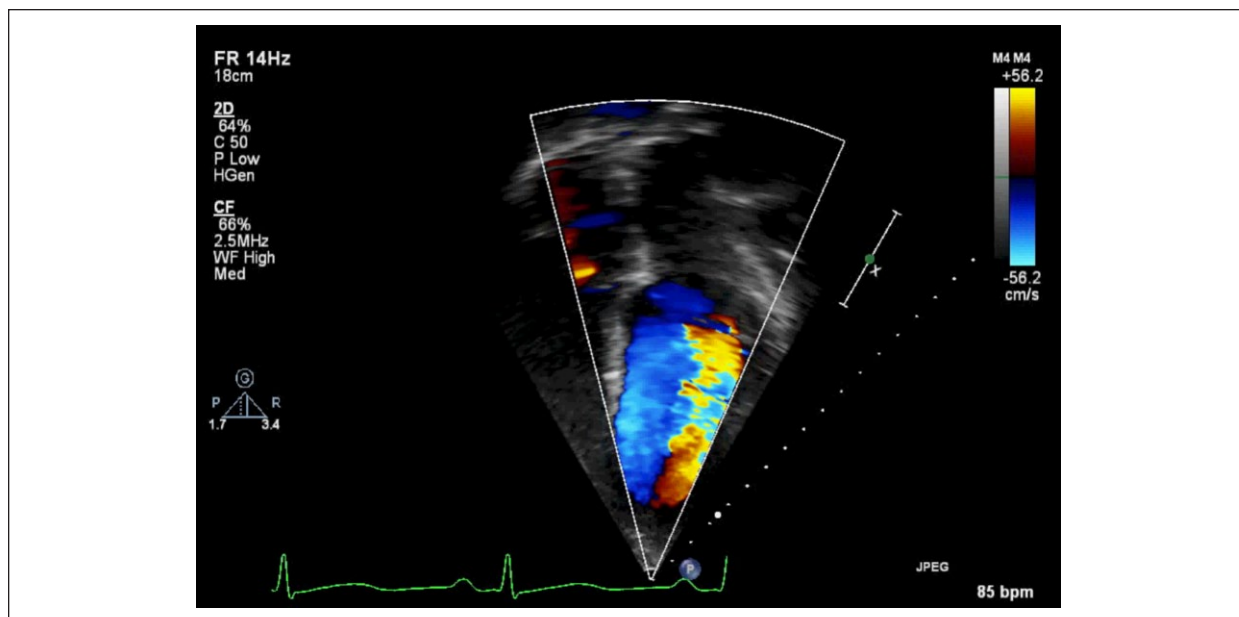


Figure 5. Transhoracic echocardiogram – Apical view revealing flow acceleration across the mitral valve secondary to restricted mobility of the mitral valve leaflets.

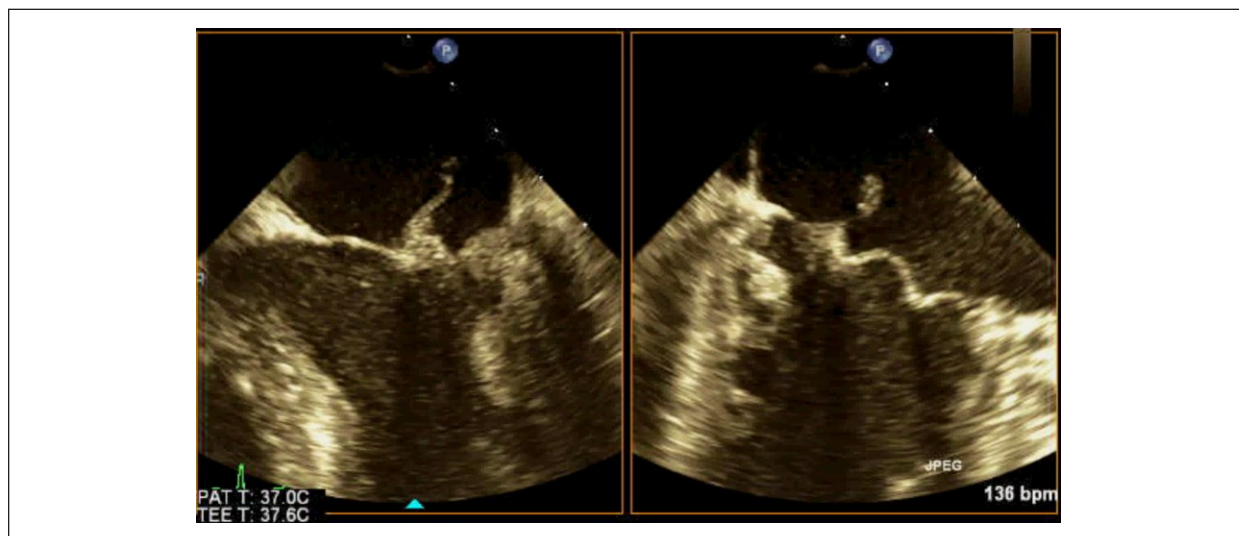


Figure 6. Transhoracic echocardiogram – Apical view revealing flow acceleration across the mitral valve secondary to restricted mobility of the mitral valve leaflets.

The prevalence of endocarditis in patients with *S aureus* bacteremia has been reported as 10% to 20%, and there is increasing agreement that echocardiography should be a routine procedure in these patients.^{8,9} *Staphylococcus aureus* endocarditis course is fulminate when it involves the mitral or aortic valve, with a widespread metastatic infection and death in approximately 25% to 30% of cases.¹⁰ Embolic phenomena occur in

20% to 50% of cases and are frequently the first presentation of infective endocarditis. The occurrence of embolic events confers a worse prognosis, particularly if associated with a neurologic event.^{8,9,11} Approximately one-fifth of patients with native-valve endocarditis have ischemic stroke. Moderate to large infarcts were 5 times more frequent with *Staph aureus* endocarditis than with nonvirulent *Streptococcus* sp. endocarditis.¹²

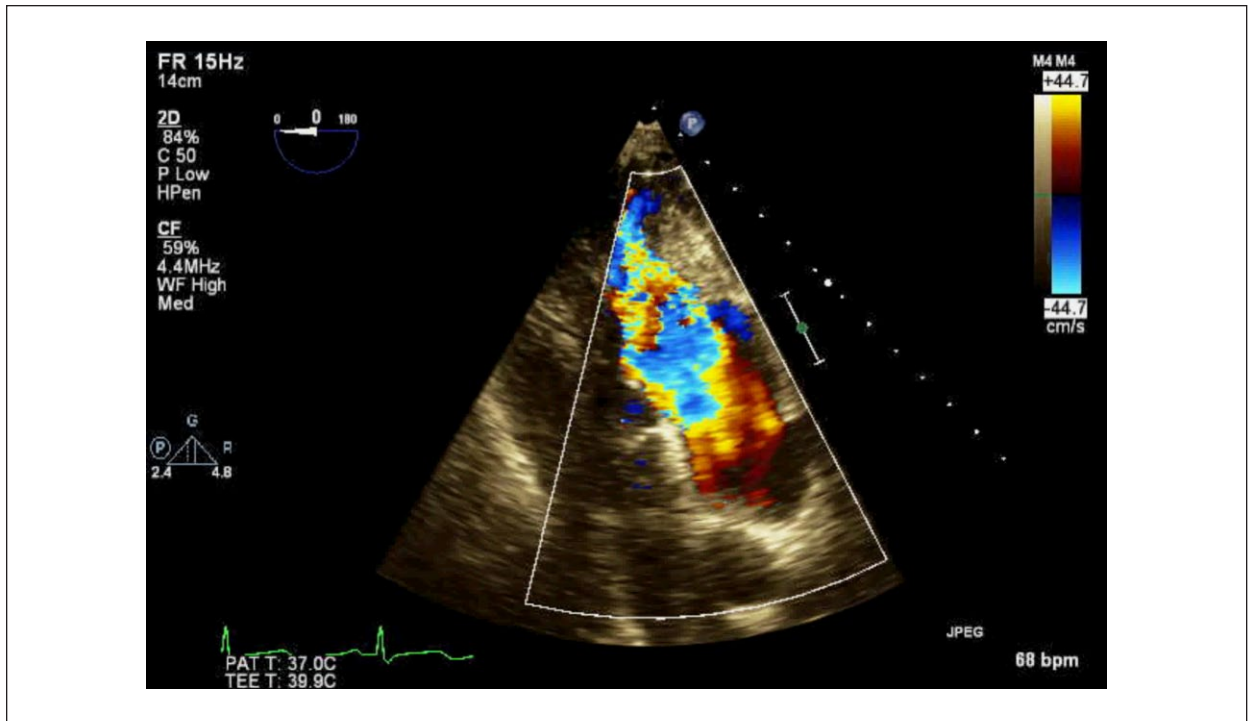


Figure 7. Transesophageal echocardiogram – revealing flow acceleration across the mitral valve secondary to restricted mobility of the mitral valve leaflets.

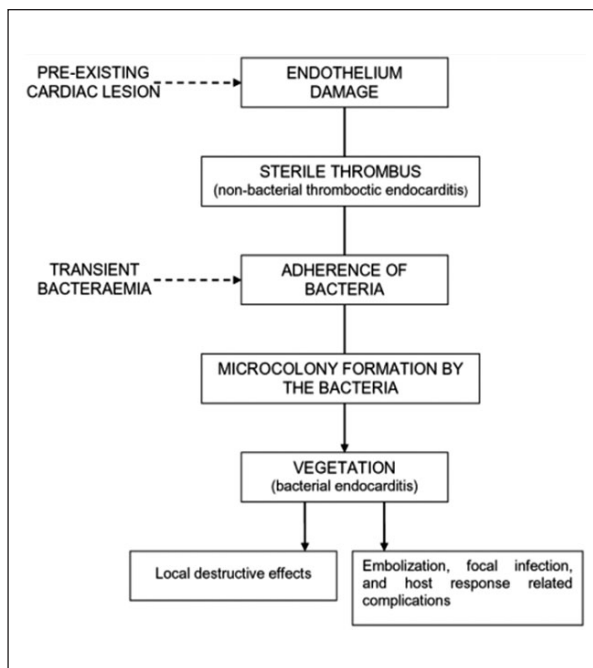


Figure 8. Pathogenesis of endocarditis (From Tomás Carmona I, Diz Dios P, Scully C. Efficacy of antibiotic prophylactic regimens for the prevention of bacterial endocarditis of oral origin. *J Dent Res.* 2007 Dec;86(12):1142-59. Reprinted with permission).

Controversy exists regarding the relationship between corticosteroids and the predisposition to infectious diseases. The detrimental effects of corticosteroids on host defense mechanisms are well known, but it is generally agreed that a short burst leads to few infectious complications, while the risk is greater to patients on long-term corticosteroid therapy.¹³ However, it is believed that some infections are more common and possibly more severe in corticosteroid-treated patients, namely, staphylococcal, tuberculosis, listeria, and gram-negative infections in the bacterial group, as well as certain viral, fungal, and parasitic infections. It seems possible therefore that the interference, however, small, of corticosteroids particularly systemic steroids with the ability of the immune system to check the spread of tissue infection and to clear bacteria from blood, can be sufficient to predispose a patient to develop bacteremia. Hence, when possible patients with eczema should be treated with topical steroids.

In addition to correct surgical treatment, postoperative antibiotic therapy is essential in acute native valve endocarditis as in our patient, and even after negative blood cultures have been established, the use of intravenous antibiotics—followed by oral antibiotics has been recommended.

Conclusion

Atopic eczema in patients with valvular or structural heart disease represents a potential risk factor for *S*

Table 1. Case Reports of Children With Infective Endocarditis and Atopic Dermatitis.

Case	Author	Age (Years)	Gender	Comorbidities	CHD	BCx	Valve involved	Surgical treatment
1	Pike and Warner ⁶	3.5	Male	Asthma, high serum IgE (38 720 µg/L) defective neutrophils migration	VSD	<i>S aureus</i>	Tricuspid	Vegetectomy, VSD repair
2	Onoda et al ⁷	16	Male	High serum IgE (12 100 IU/mL)	None	MRSA	Aortic	Valve replacement, annulus repair
3	Onoda et al ⁷	14	Male	High serum IgE (13 110 IU/mL)	None	<i>S aureus</i>	Mitral	Vegetectomy, commissure repair
4	Current case	17	Female	Eczema, asthma, high serum IgE (> 20 000 IU/mL)	None	MRSA	Mitral	Valve replacement

Abbreviations: BCx, blood culture; CHD, Congenital heart disease; MRSA, methicillin-resistant *Staphylococcus aureus*; VSD, Ventricular septal defect.

aureus endocarditis. Patients with eczema who have a history of native valve endocarditis should be managed more aggressively for eczema by altering dietary patterns, ensuring adequate skin hydration with emollients and use of topical steroids as needed to reduce the frequency of flare-ups as these can be the triggers for bacteremia and endocarditis. Physicians caring for such patients should be aware of this possible association, and closer attention to skin care plus appropriate anti-staphylococcal measures must be considered in their management.¹²

Even with the higher risk of epilepsy in patients with atopic dermatitis, once must consider the possibility of septic emboli and infective endocarditis in those patients.

Author Contributions

SMA proposed the idea of the case report, completed the literature review, formed the first draft of the manuscript and edited the final version of the manuscript. SS completed the literature review and formed the first draft of the manuscript. SA completed the literature review, helped with the formation of the first draft of the manuscript and helped with final edition of the manuscript.

Declaration of Conflicting Interests

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