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Staphylococcus Aureus Infective Endocarditis in an Immuno-competent Patient After Herpes Zoster Skin Infection

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Abstract

Certain skin conditions, like atopic dermatitis, pose a risk for the development of skin infections and, in more advanced stages of bacteremia. Consequently, it is hypothesized that skin diseases could play a role in the onset of Infectious Endocarditis (IE). We present a 21-years-old Japanese female with a history of acne vulgaris. Shingles appeared on the patient's right trunk, thus she consulted a local dermatologist after 9 days, and her condition improved after receiving an antiviral drug. However, 32 days after she developed shingles, she experienced fever and vomiting that did not improve, and presented to a nearby clinic on day 36. She was referred to the hospital on the same day. A medical work-up revealed *Staphylococcus aureus* in two sets of blood cultures taken upon admission. Transesophageal echocardiography unveiled a 1.7 cm vegetation near the septum of the right ventricular outflow tract, leading to her diagnosis of Infectious Endocarditis (IE), attributed to *S. aureus*. Chest imaging displayed multiple nodular opacities within her lung fields, interpreted as a complication associated with a septic pulmonary embolism resulting from IE. Empirical treatment with Ceftriaxone (CTRX) + Sulbactam/Ampicillin (SBT/ABPC) was initiated. CTRX+SBT/ABPC was changed to cefazolin monotherapy after identifying the causative organism, and the patient's condition gradually improved. Our case is a rare as skin disease is not previously regarded as a risk factor that triggers the onset of IE.

Keywords: Infective endocarditis • Shingles of the trunk • *Staphylococcus aureus* • Multiple nodules in the lung • Case report

Introduction

IE results from pathogenic microorganisms adhering to areas of endocardial damage. These areas of damage are often associated with abnormal intracardiac blood flow due to congenital heart disease or valvular heart disease, or caused by intracardiac artificial foreign bodies such as pacemaker leads and artificial heart valves. The disease is believed to develop through the formation of infection foci.

The three most common pathogens in IE are Viridans Group Streptococci (VGS), staphylococci, and enterococci. The distribution of these causative bacteria varies by country, region, patient age, and patient background [1]. Staphylococci have been commonly found in developed countries, but recent trends indicate a rise in *S. aureus* [2,3]. Factors associated with *S. aureus* include medical-related and hospital-acquired conditions (such as hemodialysis, vascular catheterization, surgery, and invasive procedures), artifact-related factors, older age, and intravenous drug addiction. Although there are some reports of drug abuse and piercing, cases of intravenous drug poisoning in Japan are reported to be rare [4].

Studies on how IE-causing bacteria enter the body have identified the route in 74% of cases, with approximately 40% related to skin. However, of these skin-related entries, 40% were attributed to medical device and surgical wound infections. None of the remaining community-acquired infections were ascribed to chronic skin diseases [5]. However, skin diseases are not a major risk for IE, accounting for only 0.8%–1.0% of the underlying etiologies [6,7]. Among skin diseases, atopic dermatitis is often associated with infection of skin lesions by *S. aureus* [8], whereas herpes zoster, and acne vulgaris were rarely reported to cause IE.

We reported a rare case of right-sided cardiac IE associated with skin infections of acne vulgaris and herpes zoster found in septic embolism.

Case Presentation

We report the case of a 21-year-old Japanese female with a history of acne vulgaris, but no history of device insertion, illicit drug use, recent dental care, or surgery. She developed shingles in the right lumbar L2 region and sought treatment from a dermatologist nine days post-onset. She was prescribed amenamevir, and at a follow-up visit a week later, her symptoms had noticeably improved. Over time, the herpes zoster lesions healed without any signs of superimposed infection. However, 32 days post-onset of shingles, she began experiencing persistent fever and vomiting. No new trauma or exacerbation of her acne vulgaris was noted when she visited a medical clinic after 36 days. To identify the cause of her symptoms, a whole-body Chest CT scan was performed, revealing multiple pulmonary nodules. She was subsequently referred to and admitted to our inpatient facility on the same day.

Her initial evaluation revealed a height of 166 cm, weight of 45 kg, blood pressure of 134/84 mmHg; pulse rate of 98 beats/min; blood oxygen level of 98% at room air; body temperature of 38.3 ℃; lymphadenopathy was not palpable; moderate acne vulgaris with 6–20 inflammatory lesions on one side ;no heart murmur; no abnormal lung auscultation; flat/soft abdomen without tenderness; The site of herpes zoster had only walnut-sized pigmentation, and there was no obvious blister or redness.; no Osler and Janeway nodules; no swollen joints.

Table 1 shows the laboratory findings upon admission. Mild hepatic function abnormalities, inflammatory reactions, and increased d-dimer levels were observed.

Whole-body contrast CT revealed multiple nodular shadows in the bilateral lung field (Figure 1). *S. aureus* was detected in two sets of blood cultures collected on admission. Transesophageal echocardiography revealed a lesion of approximately 1.7 cm near the interventricular septum of the right ventricular outflow tract (Figure 2). The aforementioned findings satisfied one major

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Table 1. Laboratory findings.

Marker	Laboratory Values	Reference Range
White blood cell count	10.6	3.3–8.6 × 10^9/L
Red blood cell count	4.83	3.86–4.92 × 10^{12}/L
Hemoglobin	13.9	11.6–14.8 g/dL
Hematocrit	43.2	35.1–44.4%
Mean corpuscular volume	89.4	83.6–98.2 fL
Mean corpuscular hemoglobin	28.8	27.5–38.2 pg
Platelet count	220	158–348 × 10^9/L
Total bilirubin	0.6	0.4–1.5 mg/dL
Alkaline phosphatase(IFCC)	74	38–113 U/L
Aspartate aminotransferase	33	13–30 U/L
Alanine aminotransferase	43	7–23 U/L
Lactate dehydrogenase	290	124–222 U/L
βGlutamyl transpeptidase	42	9–32 U/L
Blood urea nitrogen	9.4	8.0–20.0 mg/dL
Creatinin	0.74	0.46–0.79 mg/dL
C-reactive protein	4.89	0.00–0.14 mg/dL
Procalcitonin	0.89	≤ 0.05 ng/mL
D-dimer	1.15	<1.00μg/mL

L: Liters; g/dL: grams per deciliter; fL: femtoliters; mg/dL: milligrams per deciliter; pg: picogram; U/L: Unit per Liter; ng/mL: nanograms per milliliter; μg/mL: microgram per milliliter

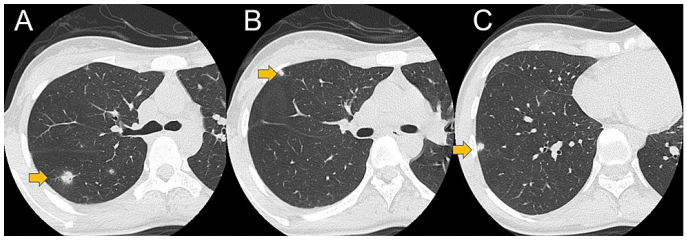

Figure 1. Chest CT image A, B and C show images displaced from cranial to caudal, respectively. Arrows indicate multiple nodules with random distribution in the lung field, representing septic embolism.

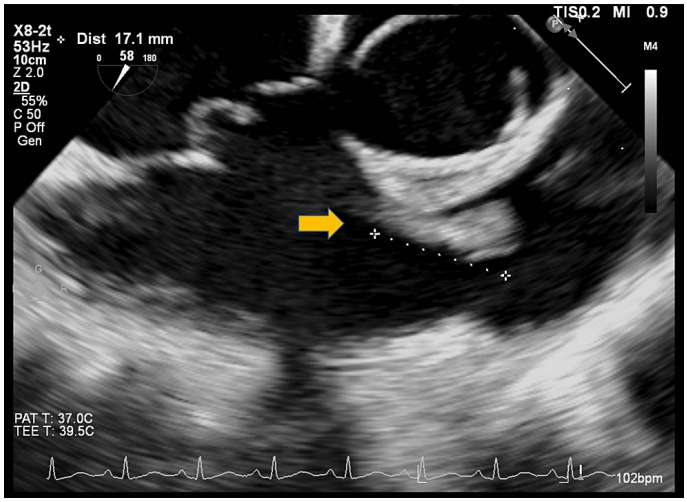

Figure 2. Transesophageal echocardiography.

criterion (endocardial injury findings) and three minor criteria (fever, vascular phenomenon, and microbiological findings) of the modified Duke diagnostic criteria [9], and thus the patient was diagnosed with IE due to *S. aureus*.

A lesion approximately 1.7 cm from the interventricular septum in the right ventricular outflow tract. The arrows in Figure 2 represent vegetation.

Pending the identification and sensitivity of the causative organism, the patient was started on an empirical antibiotic regimen of ceftriaxone and sulbactam/ampicillin. Subsequent blood cultures identified a methicillin-sensitive *S. aureus*, prompting a change from ceftriaxone and sulbactam/ampicillin to cefazolin monotherapy. By the 3rd day of hospitalization, follow-up blood culture tests confirmed the bacteria had been eradicated. Concurrently, her symptoms of nausea subsided and her fever resolved. On the 5th day of hospitalization, she was switched to cefazolin, but on the 14th day, she developed drug-induced pancytopenia, necessitating a switch back to sulbactam/ampicillin. On her 27th day in the hospital, she experienced a drug eruption, prompting a switch to levofloxacin. She was discharged and completed a total of 6 weeks of antibiotic therapy from the day of admission. Her subjective symptoms improved within a few days of hospitalization, and no recurrence has been observed since.

Results and Discussion

Clinical symptoms of IE include heart failure due to preexisting heart disease deterioration or embolization and valvular destruction. Approximately 80% of patients with IE have underlying heart disease (e.g., valvular or congenital heart disease), whereas the remaining 20% do not [10], as in our case. Intravenous drug use is the most common cause of IE in patients without underlying heart disease, followed by bacteremia associated with intravenous catheter placement and medical procedures. In particular, alcoholism, abortion, and immunodeficiency have been reported as risk factors for right-sided IE; however, none of these conditions were present in our patient [11].

In this case, we sought the source of the bacteremia but found no clear cause, aside from moderate acne vulgaris, which was present prior to admission, and nearly healed scars from herpes zoster. Previous studies have shown that *S. aureus* can colonize the skin of patients with atopic dermatitis, suggesting this could potentially be the cause [12].

In patients with atopic dermatitis, antibacterial peptides, such as βdefensin and cathelicidin, are degraded by the influence of inflammatory cytokines, such as interleukin (IL)-4, IL-10, and IL-13, and the skin becomes exposed, making it easier for bacteria to invade and infect [13]. *S. aureus* is detected in only 2%–25% of normal skin [14] but may exceed 90% in patients with atopic dermatitis [15]. Thus, atopic dermatitis is considered a risk factor for IE [12]. However, our current case had no history of atopic dermatitis and had only acne vulgaris and herpes zoster infection before the onset of IE.

However, there are not many reported instances of infective endocarditis originating from skin infections in patients with atopic dermatitis. This lack of reporting may be due to an underrepresentation of the patient population with atopic dermatitis itself, rather than the condition being rare. While there are no reports specifically pertaining to acne vulgaris, it could potentially serve as a gateway for infection given its disruption of the skin barrier.

Moreover, when Herpes Zoster Virus (HZV) infection coincides with a bacterial infection, it has been demonstrated to cause necrotizing fasciitis, indicating that it could potentially enhance the likelihood of a bacterial infection [16]. The complications of varicella-zoster virus infection are diverse and well-known in children, including Reye's syndrome associated with low protein C and S levels, cerebellar ataxia, arthritis, thrombocytopenia, and fulminant purpura. However, secondary bacterial infection is the most common complication [17]. Varicella pneumonia is the most common complication causing hospitalization in adults. Skin barrier mechanism disruption is thought to cause bacterial infection. Additionally, Blank CA, et al. reported a case in which varicella infection impairs the host's immunity, causing a transient granulocyte-killing defect [18]; however, our current case demonstrated no significant immunosuppression. In this case, the skin lesions were quite mild. However, there have been reports of IE developing from microcellulitis due to the subcutaneous injection of illicit drugs, a method known as "skin popping" [19]. Thus, it is considered that herpes zoster and acne vulgaris, skin conditions not originally seen as risk factors for IE, may have contributed to the development of IE in this case.

Conclusion

We encountered a case of IE that appeared to be secondary to acne

vulgaris and herpes zoster, conditions not traditionally viewed as standalone risk factors for IE. Even minor disruptions of the skin barrier or minor skin damage, potentially due to cytokine effects, can lead to bacteremia in the presence of *S. aureus*. The Japanese national guidelines for IE do not advocate for minimally invasive treatment with prophylactic antibiotics, except in cases with a history of IE or atopic dermatitis. Thus, we suggest considering the possibility of IE when dealing with skin diseases that might not initially appear to be risk factors, particularly when other contributing factors or causative bacteria are present.

Further prospective multicenter studies are recommended to assess the association between skin disease and IE.

Acknowledgement

None.

Conflict of Interest

None.

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