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CASE REPORT

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Staphylococcal endocarditis with meningitis and basal ganglia infarcts mimicking meningococcemia

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Abstract

Staphylococcus aureus is one of the common causes of infective endocarditis (IE). IE can present with various neurological complications such as stroke, brain abscess, and meningitis, the mortality rate can be very high in such cases.

KEYWORDS

endocarditis, meningitis, Nepal, Staphylococcus

1 | INTRODUCTION

Staphylococcus aureus bacteremia is the major cause of community-acquired and hospital-acquired bacteremia and usually presents with symptoms such as fever or hypotension. A new sterile site can have established infection because of bacteremia such as infective endocarditis (IE).[1] Incidence of IE due to *S. aureus* bacteremia in a native valve varies, and some studies found it to be grossly between 10 to 20%.[2,3] IE caused by *S. aureus* is more severe and associated with devastating consequences such as severe sepsis, neurological disorders, multiorgan failure, and even higher mortality rates.[4] Neurological complications of IE can manifest in various ways such as embolic stroke, brain abscess, meningitis, and cerebral hemorrhage. The mortality rate associated with neurological complications and IE is high, some studies even report it to be more than 50%. [5,6]

Here, we describe a case of a young female with Staphylococcal endocarditis presenting as isolated meningitis and mimicking meningococcal meningitis with meningococcemia.

2 | CASE PRESENTATION

A 22-year female, previously healthy, presented to our center with fever, headache, altered level of consciousness, and skin rashes. The skin rashes started from palms and soles (Figure 1) which was a petechial type, later progressed to involve both the limbs and trunks some of which were pustular and painful. There were no episodes of loss of consciousness, abnormal movements, and visual disturbance. There was no history of dental and surgical procedures. Other medical history was non-significant. On admission, her BP was 90/68 mm hg, pulse rate of 139 beats per minute, and respiratory rate of 40 per minute. On examination, she was ill-looking, conscious, and drowsy. Pallor and dehydration were present, and she had neck

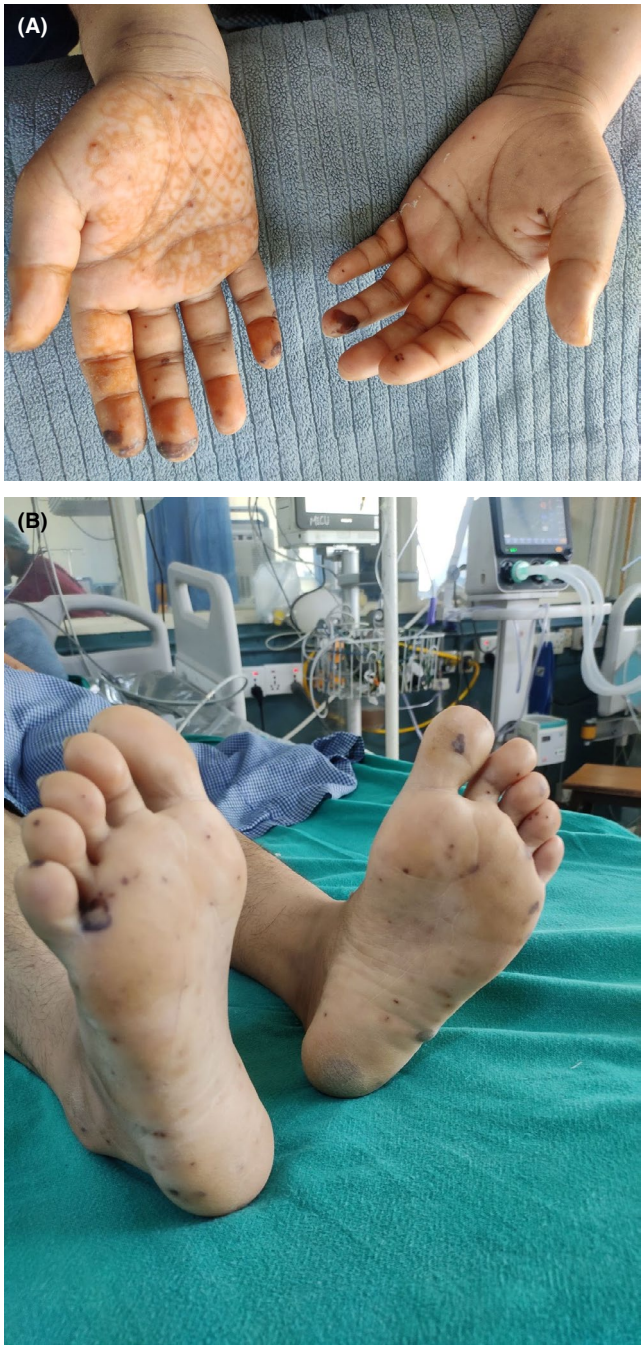

FIGURE 1 (A) and (B): Characteristic Janeway lesion seen in palms and soles of the patient

rigidity, positive Brudzinski, and Kernig signs. The rest of the systemic examinations was normal. Laboratory studies revealed high total leucocyte count (TLC) 16300/cmm with 82% neutrophils and 17% lymphocytes; hemoglobin 11 gm/dl; platelet count 53000/cmm with elevated alanine transaminase, and aspartate transaminase. Sugar, renal function tests, and electrolytes were within the normal range. Prothrombin time, fibrinogen, and FDP d-dimer were all elevated. Lumbar puncture and CSF analysis were done, and the CSF findings were TLC 250/cmm with 80% polymorphs and 20% monomorphs; RBC 2-3/hpf; sugar 1.6mmol/l; and protein 110g/l. But the CSF culture was negative. CT scan showed bilateral thalamic hypodensity (Figure 2) and Japanese Encephalitis was suspected. But the CSF for Japanese Encephalitis came out negative. Our provisional diagnosis was meningococcemia with meningococcal meningitis. The patient was admitted to ICU and managed with ceftriaxone 2 gm IV BD and vancomycin (20 mg/kg/day, BD). The clinical condition of the patient did not improve over the course of the next few days. Scrub typhus, brucella, leptospira, and dengue serology were done and all came out negative. Culture of blood, urine, and sputum was performed. Repeated blood culture grew methicillin-sensitive *Staphylococcus aureus*. Chest X-ray findings were unremarkable. Fundus evaluation was then carried out which revealed exudate with preretinal hemorrhage suggestive of Roth spots. Transthoracic echocardiography (TTE) did not show any endocarditis-related changes but due to strong clinical suspicion transesophageal echocardiography (TEE) was ordered. An echogenic oscillating mass sized 13 * 5mm in anterior mitral leaflet predominantly on the atrial side at the junction of aortomitral continuity suggestive of vegetation (Figure 3) was observed in (TEE). Based on echocardiographic, MRI findings, positive blood culture, and clinical manifestation, diagnosis of *Staphylococcus aureus* bacteremia from foci of infective endocarditis leading to bacterial meningitis was made. IV vancomycin (30 mg/kg/day, BD) was started for 6 weeks. The patient's condition was improved, and she was discharged after negative blood culture and improvement on TEE.

3 | DISCUSSION

Infective endocarditis can present with neurological complications such as meningitis, stroke, and abscess.[7] Isolated bacterial meningitis as the first manifestation of bacterial endocarditis is challenging for any physician to diagnose. In our case, diagnosis was established as the culture of blood revealed the pathogen along with evidence of the vegetation revealed by transesophageal echocardiography. Clinically, this fitted in "Definite IE" according to modified Duke's criteria.

Infective endocarditis can be defined as an infection of the endothelium of the heart and can present either as an acute or subacute form.[8] Its incidence varies in between the countries, in the US alone its incidence is about 15 per 100,000.[9] *Staphylococcus aureus* followed by viridans streptococci is the leading cause of native valve endocarditis.[10] The most common clinical presentation of infective endocarditis is fever (86%–96%), the common clinical signs being the appreciation of a new murmur or worsening of

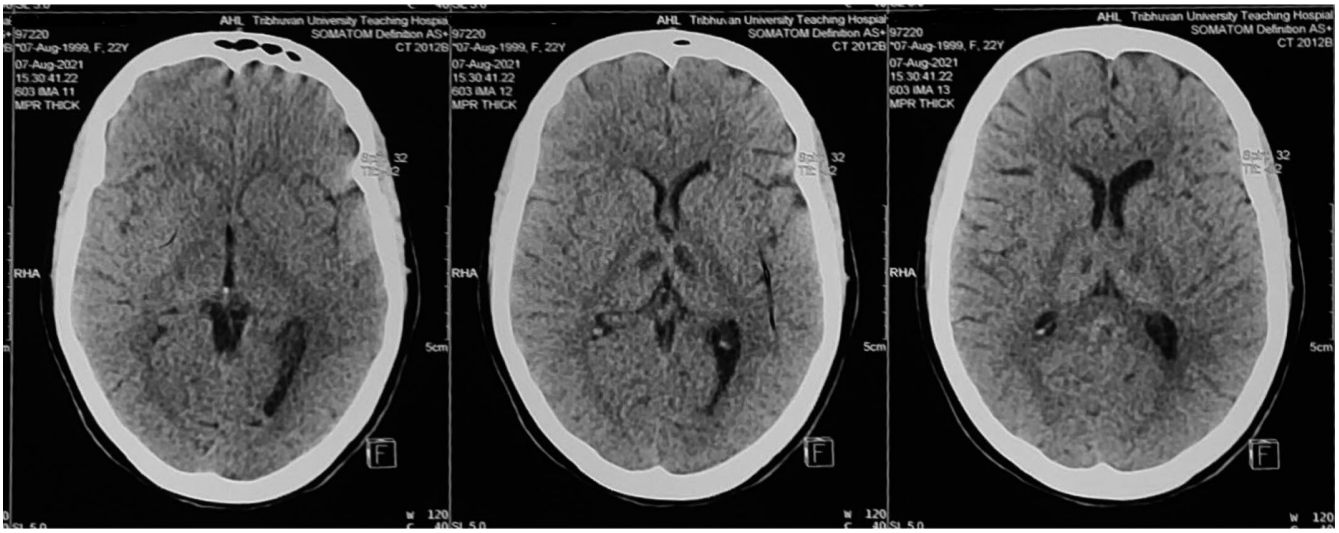

FIGURE 2 Noncontrast CT scan of head showing bilateral thalamic hypodensity

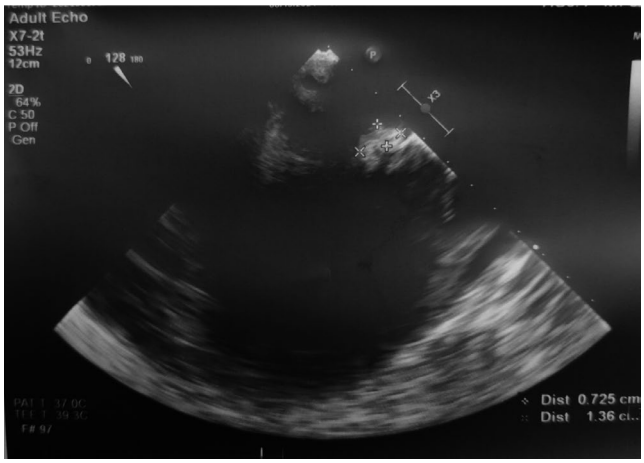

FIGURE 3 TEE showing vegetation in the atrial side in junction of aorto-mitral area

the old murmur.[10] At least, three sets of blood cultures are required for the diagnosis of infective endocarditis, as it detects 96 to 98% of bacteremia.[11] In patients with organisms other than Streptococcus causing IE, the first blood culture was positive in 82% of cases and 100% of cases in the first two cultures.[12]

Staphylococcus aureus was isolated from blood culture but CSF culture is negative in the sample obtained from the patient. CSF culture could be negative due to pre-antibiotic treatment. Timely diagnosis and treatment of IE patients are vital, as they are complicated by dreadful events including neurologic complications. TEE is recommended in patients who have negative or non-diagnostic TTE but still has high clinical suspicion as TEE has more sensitivity than TTE for vegetation in IE. TEE can also be used to diagnose possible IE if Staphylococcal bacteremia is present without a known source.[13] Empirical antibiotics can be started, and then, antibiotics can be tailored as per the blood culture, local resistance patterns, severity of infection, and presence of prosthetic materials.[11]

Staphylococcus aureus is an uncommon cause of bacterial meningitis[14] but it is the most common cause of IE.[15] In a large cohort of 2781 patients with IE, *Staphylococcus aureus* was the most commonly isolated organism (31%).[10] IE due to *Staphylococcus aureus* is more associated with severe complications than other organisms, including severe sepsis (39% versus 6%), major neurological complications (18% versus 8%), and mortality (34% versus 10%).[4] Neurological symptoms can be the initial manifestation of infective endocarditis. Some studies even have predicted the temporal relationship between infective endocarditis and stroke, they found that the risk of stroke starts to increase approximately 4 months before diagnosis, and the highest risk is 1 month after diagnosis of IE.[16] The most common neurological complication in infective endocarditis is ischemic stroke, and 1 to 20% of patients present with meningitis as a complication which indicates that meningitis is a rare one. Meningitis is usually pyogenic but chronic endocarditis can lead to meningeal irritation and CSF pleocytosis. In this case, careful history and examination are required which includes the history of fever before meningeal signs, any risk factors for endocarditis, and a vigilant search for peripheral stigmata of endocarditis.[17] *Staphylococcus aureus* endocarditis-related meningitis may remain unnoticed as 30% of cases do not have a murmur at presentation which is similar to our case.[18] Left-sided vegetations are more common to embolize with clinical sequelae than right-sided vegetations. A review showed that mitral vegetations preferably involving anterior mitral leaflet have the highest risk for embolization.[19] Bilateral thalamic hypodensity is a

classical CT finding in Japanese Encephalitis. Since CSF for the virus was negative, the possible explanation for thalamic hypodensity was thalamic infarct due to septic embolization which was supported by MRI finding too. Septic embolization is more common in middle cerebral artery distribution.[20] Although most patients with acute signs and symptoms of systemic arterial embolization do not have underlying endocarditis, it should be considered as a possible etiology especially in young patients.[21] Silent cerebrovascular complications may occur in up to 80% of patients.[22] The likelihood of detecting cerebral emboli by MRI is markedly higher than that by clinical manifestations.[23] *Staphylococcus aureus* as a causative organism itself increases mortality in patients with IE. The outcome of patients with neurologic complications and *Staphylococcus aureus* IE is variable some reporting it to be up to 74%.[24]

The pustular rashes seen over limbs and trunks were the skin manifestation of *Staphylococcus aureus* bacteremia, whereas the petechial-looking rashes on palms and soles are possibly the Janeway lesions formed due to microabscess in the dermis produced by septic emboli.

Early treatment of bacterial meningitis and endocarditis in our case prevented the development of further neurological complications including stroke. The antibiotic choice for IE-related meningitis is no different from the standard principles of bacterial meningitis management. But the duration of treatment is significantly longer (4–6 weeks) in IE-related meningitis.[7] This might be the reason why the patient did not respond to standard treatment of bacterial meningitis initially as endocarditis was not suspected by then.

4 | CONCLUSION

Staphylococcus aureus is an uncommon isolate among the various organisms causing bacterial meningitis. With the background of poor responsiveness to the standard principles of bacterial meningitis management, additional cardiac workup must be considered as it can be IE-related meningitis. The diagnosis is challenging in our case due to the absence of typical signs and symptoms of IE at the time of presentation and it mimicked meningococcal sepsis. If there is a delay in the diagnosis and treatment, the patient can have severe complications and even death.

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We would like to acknowledge the patient herself and her relatives who allowed us to share their clinical report to the global platform, so that others will be benefitted.

CONFLICT OF INTEREST

The authors would like to declare that they have no competing interests.

AUTHORS CONTRIBUTION

Subarna Giri (SG) and Bhushan Shrestha (BS) involved in the concept of study and study designs. SG, Dharmendra Sapkota (DS), and BS involved in the review of previous literature and preparation of the draft of the manuscript. SG and Aashish Shrestha (AS) involved in the preparation of the final manuscript and editing. Bikram Prasad Gajurel (BG), Niraj Gautam (NG), and AS were the treating physicians of the case. All authors individually did the final proofreading of the manuscript before submission.

ETHICAL APPROVAL

As case reports are exempt from ethical approval in our institution, our article which describes a case report does not require additional permissions from the Ethics committee.

CONSENT

Full written informed consent was obtained from the patient for publication of her case, clinical images, and radiographic images. A copy of written consent can be made available to the editor in chief of this journal upon request.

DATA AVAILABILITY STATEMENT

All the data generated or analyzed during this study are included in the manuscript.

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