

Case Report

A rare case of a novel coagulase negative *Staphylococcus* native valve endocarditis in a 28-year-old male



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ABSTRACT

Coagulase negative staphylococci (CoNS) are an emerging cause of native valve endocarditis in community and healthcare settings. We describe a case of a 28-year-old man with no significant risk factors who presented with *Staphylococcus pettenkoferi* native valve endocarditis. During our patient's initial hospitalization, he was treated for CoNS bacteraemia and subsequently discharged after a protracted hospital course with a transthoracic echocardiogram (TTE) showing no valvular vegetations. However, during the course of his second hospitalization, speciation identified *S. pettenkoferi* and transoesophageal echocardiogram (TEE) showed aortic valve perforations with new regurgitation raising concern for left sided endocarditis. We postulate that our patient may have been infected with the same CoNS species causing aortic valve endocarditis during his initial hospitalization. This case highlights the importance of recognizing CoNS as a possible causative bacterium in NVE, as well as the importance of obtaining a TEE when evaluating a patient for suspected endocarditis.

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Introduction

Coagulase negative staphylococci (CoNS) are a rare cause of native valve endocarditis. However, CoNS are emerging as an important cause of native valve endocarditis (NVE) in both community and healthcare settings [1,2]. *Staphylococcus pettenkoferi* is a novel CoNS that was first isolated from blood cultures in 2002 and has been cited as an emerging pathogen causing systemic infection in humans, including bacteremia [3,4]. We describe a case of a 28-year-old man with no significant risk factors who presented with *Staphylococcus pettenkoferi* native valve endocarditis.

Case presentation

A 28-year-old African American male with a past medical history of multiple sclerosis, diagnosed 6 months ago, was brought into the emergency department (ED) of our hospital by emergency medical services (EMS) after a witnessed generalized seizure. He received 10 mg of midazolam in the field prior to presentation and due to his altered mental status, initial history could not be obtained. On

presentation his temperature was 102.3°F with a heart rate of 103 beats per minute, a blood pressure of 147/74 mmHg, a respiratory rate of 16 and an oxygen saturation of 97% on room air. On physical exam he was lethargic but arousable to verbal stimuli. Cardiopulmonary and abdominal exams were normal with no audible murmurs or bruits. Pulses were 2+ in the upper and lower extremities without peripheral edema or notable skin lesions. Laboratory studies were significant for an elevated white blood cell count of 16 k/uL, an elevated creatinine kinase of 422 U/L, and a lactate of 4.4 mmol/L. Chest radiograph showed no evidence of acute infiltrate or consolidation. A computerized tomography (CT) scan of his head showed numerous scattered hypodensities throughout the bilateral cortex and in the right posterior limb of the internal capsule predominantly centered at the gray-white junction concerning for septic emboli (Fig. 1). Blood and urine cultures were drawn in the ED and he was subsequently started on broad spectrum antibiotics with vancomycin and piperacillin/tazobactam after receiving 1 g of levetiracetam and 650 mg of acetaminophen. The medical intensive care and neurology teams were consulted, and the patient was admitted to medical telemetry with a plan to perform transthoracic echocardiogram (TTE), continue empiric antibiotics and start continuous video electroencephalography (vEEG) monitoring.

On the second day of hospitalization, aerobic blood cultures grew gram positive cocci in clusters that later speciated as *Staphylococcus*

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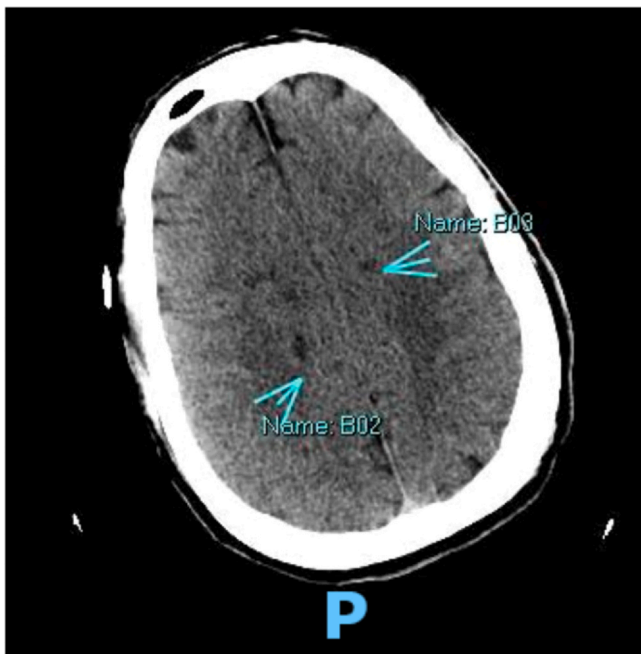


Fig. 1. Computerized Tomography (CT) imaging of the head on presentation illustrating scattered hypodensities throughout the bilateral cortex.



Fig. 2. Magnetic Resonance Imaging (MRI) of the head showing numerous white matter lesions of both cerebral hemispheres.

pettenkoferi on the third day of admission. TTE showed no evidence of valvular vegetations or regurgitation and vEEG showed multiple nonconvulsive focal seizures. He was continued on levetiracetum 500 mg twice a day for the seizures and the antibiotic regimen was de-escalated to vancomycin and ceftriaxone after the infectious disease team was consulted. The patient continued to spike fevers while on antibiotics despite subsequent blood cultures drawn on the 2nd and 3rd day of his hospitalization showing no growth to date. A transoesophageal echocardiogram (TEE) performed on the fourth day of his hospitalization did not show evidence of valvular vegetations but revealed micro-perforations in the left coronary leaflet with trace to mild aortic regurgitation. The structural heart team was consulted and advised that no surgical intervention was indicated

for the echocardiogram findings. On the fifth day of his admission magnetic resonance imaging (MRI) of the brain showed numerous white matter lesions of both cerebral hemispheres, suggestive of demyelinating disease (Fig. 2). After a discussion with the infectious disease and neurology team, the decision was made to clinically monitor the patient off antibiotics after ruling out the possibility of septic emboli on imaging and the lack of vegetations on the echocardiogram. His antiepileptic regimen was titrated to levetiracetum 1500 mg BID with the addition of lacosamide 100 mg BID to control the seizures.

Collateral history obtained from his previous hospitalization at another institution a few months prior to his presentation revealed that during that admission, the patient had been brought in by EMS after being found down with dilated pupils and urinary incontinence. Urine toxicology was negative at that time, however the patient later admitted to using synthetic marijuana. He was admitted for septic shock secondary to CoNS bacteremia isolated in 4 blood culture bottles that were unable to be speciated at that institution. He was treated empirically with broad spectrum antibiotics and underwent a TTE that was negative for valvular vegetations, as well as a lumbar puncture that showed no infectious etiology. He was discharged from this institution after a protracted hospital stay with a course of steroids as part of the treatment of a multiple sclerosis flare after MRI showed demyelinating lesions. During both hospitalizations, his antibiotic regimen was discontinued after negative TTE results and subsequent blood cultures clearing after several days of broad-spectrum antibiotics. He was discharged from our institution with a plan for primary care and neurology follow up as an outpatient.

Discussion

The variability in clinical presentation of infective endocarditis (IE) and the importance of early accurate diagnosis requires a diagnostic strategy that is both sensitive for disease detection and specific for its exclusion across all forms of the disease [5]. In 1994, Durack and colleagues from the Duke University Medical Center proposed a diagnostic schema that stratified patients with suspected IE into 3 categories: definite, possible, and rejected cases [6]. As per the modified Duke's criteria, our patient was classified as a "possible endocarditis" based on his echocardiogram findings of new valvular regurgitation with perforations in the setting of fevers and CoNS bacteremia. *S. pettenkoferi* is a relatively novel species that has been cited as causing bacteremia in humans. However, to our knowledge there are no reports of this bacteria causing endocarditis [3,7].

Although CoNS are a frequent cause of prosthetic valve endocarditis, they are traditionally regarded as a rare cause of native valve endocarditis (NVE) [2]. CoNS are usually skin flora with the tendency to colonize foreign materials in the body, hence the propensity of causing prosthetic valve endocarditis. It is unusual for CoNS to cause NVE, especially in a young patient without the cardinal risk factors for endocarditis, such as intravenous drug abuse, poor dental health or long term intravenous indwelling catheters [8]. During our patient's initial hospitalization, he was treated for CoNS bacteremia and subsequently discharged after a protracted hospital course with TTE showing no valvular vegetations or regurgitation. However, during the course of his second hospitalization, he underwent a TEE that showed aortic valve perforations with mild aortic regurgitation. This finding raised concern for previous left-sided endocarditis.

Aortic cusp perforations secondary to endocarditis with moderate to severe regurgitation associated with the development of congestive heart failure (CHF) is an indication for surgical intervention [9]. Our patient had only mild aortic regurgitation and no signs and symptoms of CHF. Of note, our patient only grew *S.*

pettenkoferi in one of his blood cultures bottles, however, all subsequent blood cultures were drawn while our patient was on broad-spectrum antibiotics. We postulate that our patient may have been infected with the same CoNS species causing aortic valve endocarditis during his initial hospitalization where TEE was not performed. He was subsequently discharged after clinical improving with several days of intravenous antibiotic treatment, and did not receive a prolonged antibiotic course as all subsequent culture data was negative.

Conclusion

Although native-valve endocarditis is typically caused by *S. aureus*, viridans streptococci, and enterococci, awareness of disease caused by emerging organisms is important when clinical suspicion of endocarditis is present [10]. In younger individuals without valvular hardware, CoNS endocarditis is extremely rare. In the initial hospitalization, where only limited TTE was performed, valves and vegetations could not be identified and the patient was treated only for CoNS bacteremia rather than endocarditis. However, because TEE performed in the second hospitalization was able to identify aortic valve perforations with regurgitation, endocarditis could be appropriately identified and treated. This case highlights the importance of recognizing CoNS as a possible causative bacterium in NVE, as well as highlighting TEE as a more sensitive imaging modality for the diagnosis of endocarditis [5].

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Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Ethical approval

Not applicable

CRediT authorship contribution statement

Caleb V. Wutawun Ashe: Writing – original draft, Writing – review & editing. **Rosaline Ma:** Writing – original draft. **Rohan Goyal:** Writing – review & editing. **Zachary Morrow:** Writing – review & editing. **Gin Den William Chang:** Writing – review & editing. **Nazish Ilyas:** Supervision.

Disclosures

None of the authors have anything to disclose.

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