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### Case Report

## Leuconostoc species endocarditis in an intravenous drug user

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#### ABSTRACT

Infective endocarditis is a potentially lethal infection, which predominantly affects the atrioventricular valves. Rapid identification and management is critical to reduce morbidity and mortality in this patient population. Herein, we present a case of a 24-year-old man with *Leuconostoc* species infective endocarditis of the aortic valve. Disease course was complicated by several septic emboli to the brain, central retinal artery, and spleen. This case serves to remind clinicians that *Leuconostoc* species, which are typically not pathogenic to human species, can cause infective endocarditis in individuals with a history of intravenous drug use.

**<Learning objective:** It is crucial that clinicians maintain a high index of suspicion in high-risk patients for infective endocarditis with *Leuconostoc* species, especially in the setting of positive blood cultures with group viridans streptococcus resistant to penicillin. Although cases of penicillin resistant group viridans streptococci have been reported, it is not common and merits further review. *Leuconostoc* is a Gram-positive ovoid cocci that is intrinsically vancomycin-resistant and is typically non-pathogenic to the human species.>

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### Introduction

Infective endocarditis (IE) is most commonly caused by the hematogenous spread of various microorganisms that target the endocardium of heart chambers and valves, with staphylococci and streptococci accounting for the majority of cases. Due to the multitude of bacterial and even fungal pathogens, treatment options should be targeted specifically to eradicate microorganisms that are isolated in blood cultures. If empiric therapy is warranted, first-line treatment usually targets methicillin susceptible and resistant staphylococci, streptococci, and enterococci. Although, there is an important caveat clinicians must consider when using vancomycin antibiotic therapy. When dealing with IE caused by *Leuconostoc*, vancomycin is not a suitable option for treatment [1]. *Leuconostoc* is a genus of Gram positive, catalase and oxidase negative, cocci placed within the family of leuconostocaceae [1,2]. It was previously believed that this bacterial species was non-pathogenic in nature until recently published reports revealed the infectious potential of the *Leuconostoc* species in many patient

populations [2]. Although there have been previously reported sensitivity panels for this organism, it has a high level of intrinsic vancomycin resistance and therefore merits a higher index of suspicion when discussing endocarditis [1]. The case presented here serves to remind clinicians of the devastating potential of untreated infective endocarditis and to raise awareness for the possibility of infection with the typically non-pathogenic *Leuconostoc* species.

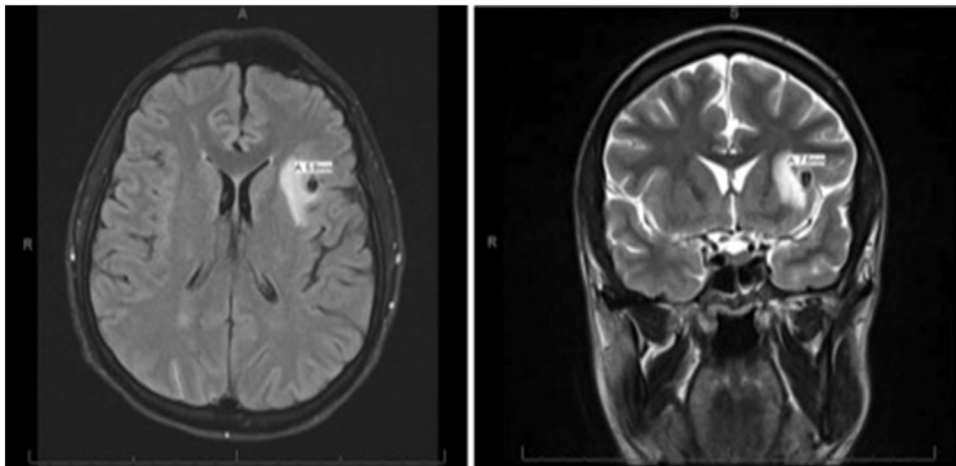
### Case report

#### Presentation

The patient was a 24-year-old male tobacco smoker with a past medical history of intravenous drug use (IVDU), bicuspid aortic valve, and untreated hepatitis C who presented with two weeks duration of worsening bilateral temporal headaches and intermittent fevers. He described the headaches as bilateral, temporal, throbbing, pressure-like, non-radiating, and intermittent. The patient had been taking high-dose ibuprofen for two weeks without any relief of symptoms. He also reported having intermittent vision loss precipitated by lateral movement of his head. Other associated symptoms included tinnitus in his right ear and photosensitivity in the right eye. The patient also had a

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**Fig. 1.** Magnetic resonance imaging (MRI) without contrast of the brain showing a focal area of hypo-attenuation seen on T2, fluid attenuated inversion recovery, and diffusion weighted imaging sequences within the subcortical left frontal lobe measuring 6 mm. The focus is hyper-intense on T1 non-contrast MRI.

persistent petechial rash in the lower extremities bilaterally. On presentation, the patient's blood pressure was 99/64 mmHg, heart rate was 103/min, temperature was 36.7 °C, respiratory rate was 18/min, and oxygen saturation was 100% on room air. Physical examination was remarkable for dilated pupils (diameter 7 mm), a grade IV/VI systolic ejection murmur radiating to the carotid artery bilaterally, Janeway lesions, Osler nodes, and a bilateral non-blanching petechial rash below the knees bilaterally. Pertinent laboratory studies included: white blood cell count (WBC) 18,000/ $\mu$ l, C-reactive protein (CRP) 104 mg/dl, erythrocyte sedimentation rate (ESR) 68 mm/h, creatinine (Cr) 4.6 mg/dl (baseline Cr 0.9 mg/dl), complement component 3 (C3) 56.6 mg/dl, complement component 4 (C4) 25.2 mg/dl, fractional excretion of sodium (FENa) 3.5%. The patient also had a troponin I elevation to 1.55 ng/ml without any electrocardiogram (ECG) changes or anginal symptoms.

#### Imaging studies

The initial chest X-ray (CXR) demonstrated mild cardiomegaly without any pulmonary or osseous abnormalities. Initial non-contrast computed tomography (CT) scan of the head showed a 2.5 cm region of hypo-attenuation in the left frontal lobe. Non-contrast magnetic resonance imaging (MRI) of the brain (Fig. 1) showed a focal area of hypo-attenuation seen on T2, fluid attenuated inversion recovery (FLAIR), and diffusion weighted imaging (DWI) sequences within the subcortical left frontal lobe measuring 6 mm. Transthoracic echocardiography (TTE) (Fig. 2) demonstrated a bicuspid aortic valve with calcified leaflets and reduced cusp separation, severe aortic regurgitation, moderate aortic stenosis with a valve area of 1.22 cm<sup>2</sup>, and vegetations on both leaflets of the aortic valve. There was also grade 1 diastolic dysfunction and a moderate pericardial effusion. The left ventricular size was within normal limits and the left ventricular ejection fraction was 55–60%. Transesophageal echocardiography (TEE) showed a 2.6 cm aortic root with no definitive evidence of abscess and confirmed the TTE findings of aortic valve vegetations. Final measurements of the aortic valve vegetations were never determined due to the extent of vegetation involvement and fibrosis within native myocardial tissue. The largest vegetation was approximated to be at least 1.2 cm at its largest length. Neuroangiography (Fig. 3) revealed a 6.9  $\times$  5.3 mm mycotic aneurysm of the left distal M2 branch of the middle cerebral

artery (MCA) with subtle subarachnoid hemorrhage and left central retinal artery occlusion. CT without contrast of the abdomen was remarkable for splenomegaly and multiple areas of low density in the spleen, consistent with splenic infarction. Renal ultrasound showed increased echogenicity and edematous parenchyma. Non-contrast CT of the chest was remarkable for multifocal alveolar infiltrates throughout both upper and lower lobes and patchy consolidations in lower lobes.

#### Infectious disease consultation

Considering the results of imaging studies and clinical findings, a diagnosis of embolic phenomenon in the setting of left-sided endocarditis was established. The patient was started on vancomycin 1000 mg intravenous (IV) daily and ceftriaxone 1 g IV daily, which eventually was deescalated to ceftriaxone alone for viridans streptococcus group bacteremia on initial blood cultures. Aerobic and fungal cultures were negative. Interestingly, on blood culture sensitivity panel, the viridans streptococcus was not sensitive to penicillin. Considering the penicillin resistance, repeat cultures were sent to an outside laboratory for further analysis,



**Fig. 2.** Transthoracic echocardiography showing a bicuspid aortic valve with moderate valve stenosis and bacterial vegetation.

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