Native valve *Neisseria meningitidis* endocarditis with embolic infarcts

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ABSTRACT

Infective endocarditis caused by *Neisseria meningitidis* (NM) is a very rare manifestation of invasive meningococcal disease with only a few cases described in the literature. We report a case of a native mitral valve endocarditis caused by NM in a 61-year-old female without meningitis.

Our case highlights the importance of considering NM as one of the causative organisms of infective endocarditis, even in the absence of meningitis. Even though the cases of NM endocarditis have drastically decreased with the advent of antibacterials, NM should always be considered in the differential diagnosis in certain high-risk groups, like diabetics, polysubstance abusers, and elderly individuals. The prognosis is good with early appropriate antibiotic treatment with or without surgery.
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Introduction

The most common pathogens that cause bacterial infective endocarditis (IE) include staphylococci, streptococci, and enterococci. *Neisseria meningitidis* (NM) is a Gram-negative diplococcus that commonly causes bacterial meningitis in young adults and children. It is the second most common cause of community-acquired bacterial meningitis in young adults. Cardiac involvement of NM is infrequent in the era of antimicrobials. IE from NM in the absence of bacterial meningitis can pose a significant diagnostic challenge. We report a case of a 61-year-old female who presented with an embolic phenomenon secondary to NM endocarditis without meningeal involvement.

Case report

A 61-year-old female was brought to our emergency room with vomiting and altered mental status. The patient's medical history included poorly controlled type-2 diabetes on insulin, marijuana use, and alcohol use disorder. Upon presentation, she was afebrile with a heart rate of 110 bpm, blood pressure 121/77 mm Hg, oxygen saturation 90 % on room air, and tachypneic. The neurological exam was notable for an altered level of consciousness with a Glasgow coma scale of 7 and bilateral horizontal nystagmus.

Her pupils were equal in size and reactive to light. Meningeal signs were absent. Cardiac auscultation revealed a systolic murmur

at the apex, and the respiratory exam was notable for bilateral rales. She was intubated for airway protection. Initial laboratory studies were significant for leukocytosis of 22,000/uL (reference 5–10), a glucose level 1500 mg/dL, plasma osmolarity 465 mOsm/L, and no metabolic acidosis. BUN and creatinine levels were also elevated at 102 mg/dL and 3.3 mg/dL, respectively.

A chest X-ray showed pulmonary vascular congestion. A head CT scan showed diffuse right temporal, parietal, and left parietal subarachnoid hemorrhage and right frontal subdural hematoma measuring up to 35 mm in thickness without any mass effect. CT angiography of the head and neck did not show any intra- or extracranial large vessel stenosis. In addition, the brain MRI revealed multiple foci of embolic infarcts in bilateral occipital, parietal, and frontal lobes in the gray-white matter junction, basal ganglia, cerebellum, and left thalamus. Subarachnoid hemorrhage in the right and left parietal lobes was also observed (Fig. 1A and B). The cerebrospinal fluid (CSF) analysis showed evidence of probable subarachnoid hemorrhage with an RBC count of 2080/uL. The CSF was also significant for a mildly elevated protein level of 77 mg/dL, as well as 6 and 3 WBCs in the first and second CSF bottles, respectively. Gram stain, PCR for HSV, and culture of the CSF fluid were negative for meningitis. Initial blood cultures showed no growth (i.e in 24 h). Further workup included a transesophageal echocardiography, which revealed normal left ventricular function with a large vegetation on the posterior leaflet of the mitral valve measuring 1.96cm × 1.29cm with posterior leaflet perforation and moderate to severe anteriorly directed mitral regurgitation (Fig. 2A–C, Movies 1A-D). There were no other valve abnormalities or evidence of an interatrial shunt. An abdominal CT scan revealed embolic splenic infarct (Fig. 1C). Subsequent blood cultures on Day 2 of admission grew Gram-negative cocci, which were later

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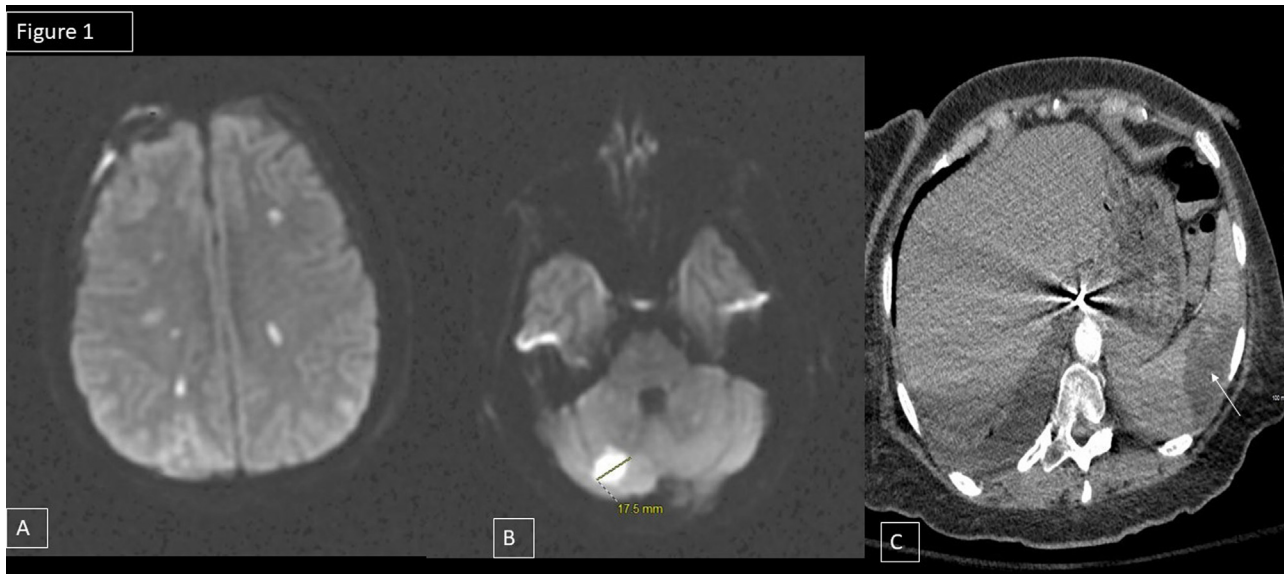*E-mail address:* shegde49@siumed.edu (S. Hegde).

Fig. 1. MRI Brain, T2 flair images showing multiple Scattered T2 hyperintensities in frontal and parietal regions [A] and a large hyperintensity in the pontocerebellar [B] region representing embolic phenomenon with hemorrhagic transformation. Axial image of CT abdomen with IV contrast showing a large wedge shaped hypodensity in the spleen representing a splenic infarct [C].
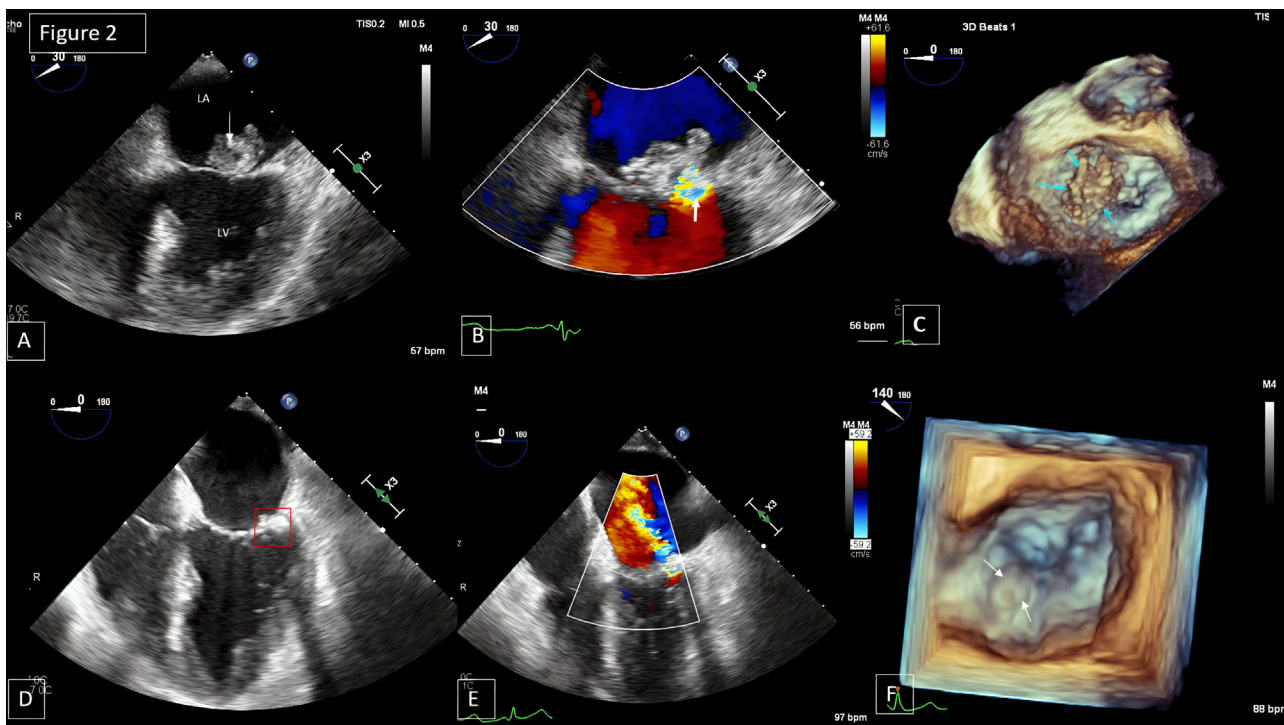

Fig. 2. Trans-esophageal echocardiography at mid-esophgeal showing left atrium (LA), Left ventricle (LV) and a large vegetation on the mitral valve [A]. [B] TEE with color doppler showing mitral leaflet perforation. [C] 3D TEE image of the vegetation as seen from the surgeons view. [D] TEE showing small calcified vegetation on the mitral valve post antimicrobials.[E] TEE with Doppler showing perforation of the mitral valve. [F] 3D TEE image of the vegetation post antimicrobials.

identified as *Neisseria meningitidis*. The initial empirical antibiotic coverage of the patient's septic shock included vancomycin and piperacillin-tazobactam. Following a positive blood culture result, antimicrobial therapy was switched to 2 g of ceftriaxone twice a day for adequate coverage of the meningococcal infection. She was appropriately treated with insulin and hydration for her diabetic hyperosmolar state. The patient's mental status gradually improved, and she was extubated on day 10 of her hospital stay. She was discharged to a rehabilitation facility to complete her antibiotic course. After the completion of six weeks of antimicrobials, a repeat TEE showed a small calcified vegetation on the posterior leaflet of the mitral valve along with leaflet perforation (Fig. 2D–F, Movies 1E-G).

The patient underwent a successful mitral valve replacement with a 25-mm Edwards Life Science Magna Ease Pericardial Valve (Movie 1 H) along with a coronary artery bypass graft x 1 (SVG-

PDA). Her postoperative recovery was uneventful, and she was discharged back to the rehabilitation facility. Follow-up one-month post-discharge, the patient was recovering well from surgery. However, she had incomplete neurological recovery with residual right extremity weakness and dysphagia.

Discussion

NM is a Gram-negative diplococcus. It was initially described in 1805 and isolated in 1882 from the CSF of an infected person. Humans are a unique reservoir of this bacterium. It is estimated that about 10 % of people are asymptomatic carriers in their nasopharynx. Systemic infections caused by NM vary in their severity, ranging from transient bacteremia to fulminant infection with a high mortality rate. The most common forms of systemic meningococcal infection include meningitis and meningococcemia with or without concomitant meningitis [1].

Among *Neisseria* species, *N. gonorrhoeae* and *N. elongata* are known to affect cardiac valves. Cardiac valve involvement is uncommon in generalized meningococcal infection. However, NM is known for its meningitis and, in rare cases, septicemia. There are a few reports of myocardial dysfunction with meningococcemia secondary to a massive release of proinflammatory cytokines [2]. NM as a cause of endocarditis without meningitis is a rare clinical occurrence. Cases of meningococcal endocarditis have been described in the literature before the era of antimicrobials. However, with the advent of antimicrobials, the cases have drastically decreased, with only 13 cases reported since 1960 [4]. Nevertheless, when it involves the heart, it is known to affect the left side more often than the right side of the heart and furthermore, native valves are more commonly affected than prosthetic valves.

There is a known correlation between complement deficiency and risk of developing fulminant meningococcal infection [3]. Other risk factors include diabetes, alcohol and polysubstance use, and age. Our patient was 61 years of age and had poorly controlled diabetes, in addition to alcohol and marijuana abuse, which may have predisposed her to meningococcal endocarditis. Complement levels were not checked in our patient. Systemic meningococcal infection can be treated with intravenous penicillin G or a third-generation cephalosporin. Our patient required valve replacement in addition to antimicrobials for severe mitral regurgitation secondary to valve destruction [5].

This case highlights the importance of considering NM as one of the causative organisms of infective endocarditis, even in the absence of meningitis. Even though the cases of NM endocarditis have drastically decreased with the advent of antimicrobials, it should always be considered in the differential diagnosis in certain high-risk groups like diabetics, polysubstance abusers, and elderly individuals. The prognosis is good with early appropriate antibiotic treatment with or without surgery.

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Declaration of Competing Interest

The authors have no disclosures to make.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.idcr.2020.e00918.

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