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CASE REPORT

Endocarditis due to *Gemella haemolysans* in a newly diagnosed multiple myeloma patient

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An 87-year-old Caucasian woman with hypertension, diabetes mellitus type 2, and COPD was admitted with 1-week duration of back pain and weight gain. The physical examination revealed jugular venous distention, rales in the left lower lung field, and severe pitting edema over lower extremities. As workup for leukocytosis, blood cultures grew *Gemella haemolysans*. Subsequently, a transthoracic echocardiogram revealed vegetation on the non-coronary aortic leaflet and mild aortic stenosis. She was treated with ampicillin and gentamicin. After further investigation, the patient was diagnosed with plasma cell myeloma, the monoclonal lambda type. This is the first reported case of *G. haemolysans* endocarditis in a multiple myeloma patient.

Keywords: Gemella haemolysans; *endocarditis*; *multiple myeloma*; Gemella; *antimicrobial therapy*

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Infective endocarditis (IE) is mostly caused by Gram-positive bacteria including Staphylococcus and Streptococcus. *Gemella haemolysans* is a Gram-positive coccoid, catalase-negative, facultative anaerobic microorganism of the mucus membranes in humans. However, *G. haemolysans* is able to cause severe and generalized infection as opportunistic pathogens, and it has become an emerging bacterial etiology in IE. Generally, Gemella endocarditis is associated with previous valvular damage or a poor dental state. Gemella endocarditis without preexistent valvular pathology has also been reported 1–3. Here, we describe the first case of bacterial endocarditis caused by *G. haemolysans* in a newly diagnosed multiple myeloma patient.

Case report

An 87-year-old Caucasian female with a history of hypertension, atrial fibrillation (non-valvular) on Coumadin anticoagulation, diabetes mellitus type 2, and COPD was admitted to the hospital with severe back pain, right hip pain, and weight gain over 20 pounds over the past week. Patient also reported constipation due to the use of opiate. She denies fever and chills.

On admission, she was afebrile and the physical examination revealed jugular venous distention, few rales in left lower lung field, no wheezing, grade 2 systolic murmur at the aortic area radiating upward, and severe pitting edema over bilateral lower extremities. Laboratory studies disclosed the following data: WBC 8,690/mm^3 (10% bands, repeated WBC 11,730), Hb 10.5 g/dL, hematocrit 32.5%, platelet 162,000/mm^3, MCV 98, RDW 17.0, sodium 128 mEq/L, Cr 1.5 mg/dL, BUN 39 mg/dL, bicarbonate 21.7 mmol/L, pro BNP 4,841, total protein 10.6 g/dL, and albumin 3.2 g/dL. Chest radiography revealed pulmonary vascular congestion. ABG revealed pH 7.34, pO2 70, pCO_2 55, and SaO_2 92%. The initial treatment was targeted for heart failure and COPD exacerbation. We treated her with furosemide, short period of prednisone, and tiotropium bromide inhalation. Her hypoxia and edema improved and the Cr improved to 0.6 mg/dL.

As workup for leukocytosis, peripheral blood culture grew *Gemella haemolysans* in two cultures. Subsequently, transthoracic echocardiogram revealed LVEF 60–65%, mild aortic stenosis, and one small vegetation on the non-coronary aortic leaflet (Fig. 1); pulmonary artery pressure was 65 mmHg. We diagnosed infective endocarditis (IE), and we started ampicillin (2 g every 6 h) and gentamicin (100 mg daily, renal dosing) according to the sensitivities. Patient's blood cultures were negative twice before discharge. We continued both ampicillin and gentamicin for 4 weeks from the date of first negative blood culture.

We performed further investigations for source of infections. MRI of lumbar spine with contrast does not

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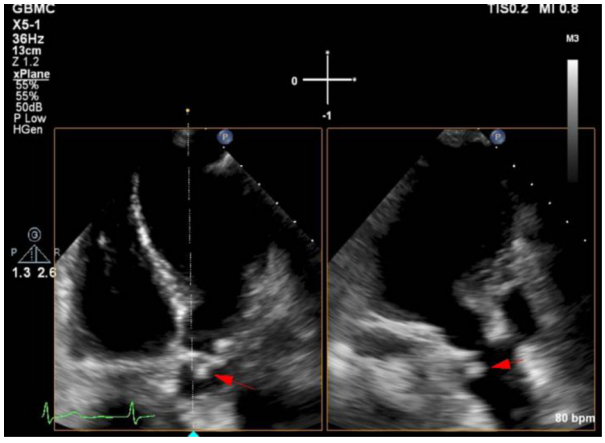

Fig. 1. TTE: a small, mobile 4–5 mm echodensity at the tip of the non-coronary leaflet.

show any evidence of discitis, vertebral osteomyelitis, and epidural abscess, but mild T12 compression fracture. Abdominal and pelvic CT showed colon fecal impaction. Given the patient's age, anemia, back pain, and elevated gamma gap, we have the suspicion of plasma cell disorder. Patient's immunofixation electrophoresis showed an IgG lambda monoclonal protein comprising 39.9% of the total protein, equivalent to 3.8 mg/dL; therefore, we performed bone marrow aspiration and biopsy, which revealed plasma cell myeloma, monoclonal lambda type (35% of the cellular elements). Further skeletal survey did not find any abnormalities except the T12 compression fracture. Head CT showed cortical atrophy, without any bony changes. Patient was discharged with dexamethasone 20 mg weekly and antibiotics. Patient died unexpectedly 1 month later, no autopsy was performed.

Discussion

The members of *Gemella* species have been classified as *G. morbillorum*, *G. haemolysans*, *G. bergeri*, *G. sanguinis*, *G. palaticanis*, and *G. cuniculi* on the basis of DNA hybridization and comparative 16S rRNA gene sequencing (2). Infections due to *G. haemolysans* have been reported infrequently.

For the first time, Thjotta and Boe described *G. haemolysans* as *Neisseria haemolysans* in 1938 (4). Reyn et al. (5) showed that the structure of the cell wall of *G. haemolysans* was typical of Gram-positive cocci by electron microscopy. The comparative thinness (10–20 nm) of its cell wall accounts for easy decolorization during Gram staining. *G. haemolysans* may therefore appear as Gram-variable or even Gram-negative. It can be misidentified as a viridians streptococcus or remain unidentified.

G. haemolysans often colonizes in the upper respiratory, gastrointestinal, and genitourinary tract as a commensal organism and occasionally causes localized and/or disseminated infections. It is a rare cause of endocarditis, with 19 cases reported in the literature and only four cases associated with prosthetic valve endocarditis (1, 2, 3, 6). Our patient is the second case reported in the United States (7). Most documented cases of *G. haemolysans* endocarditis occur in patients with underlying mitral or aortic valve disease (including prosthetic valves) and/or poor dentition or dental manipulation, which was not the situation with this patient. Infected valves were aortic and mitral, both of them with the same percentage (50%) (1–3). There are also case reports about meningitis (8) and brain abscess (9).

For diagnosing IE, there is generally good agreement between transthoracic echocardiography (TTE) and pathologic findings from autopsy or surgery. However, there are limitations in ultrasound measurements of small vegetations (<3 mm), which might be undetectable by TTE (10). Since our patient's vegetation size was 4–5 mm, and she was fragile, we defer the transesophageal echocardiography.

Antimicrobial therapy for *G. haemolysans* IE would typically include penicillin or ampicillin in combination with gentamicin. In all published cases, patients were successfully treated with antibiotics alone or in combination with cardiac valvular replacement, one patient with prosthetic valve endocarditis requiring implantation of a total artificial heart as a bridge to heart transplantation (6). The results of antimicrobial susceptibility studies by Buu-Hoï A (11) demonstrate that all strains of *G. haemolysans* were highly sensitive to penicillin G and ampicillin. Their results suggest that penicillin G combined with an aminoglycoside can be recommended for the treatment of endocarditis caused by *G. haemolysans.*

Patients with multiple myeloma are more susceptible to infections, which are the major causes of morbidity and mortality. Despite the common occurrence of bacteremia, IE is seldom reported. By searching PubMed, we can only find scattered case reports without statistic data about IE in multiple myeloma patients.

Conclusion

G. haemolysans is a rare but important cause of IE. The association of *G. haemolysans* and multiple myeloma has not been reported to date. Patients with multiple myeloma are susceptible to infection due to compromised immunoglobulin synthesis and function. In summary, because of the lack of other risk factors, we felt that multiple myeloma maybe a under recognized risk factor for endocarditis due to *G. haemolysans.*

Disclosure

None of the authors have any financial or personal bias that would inappropriately compromise the publication of this work.

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