A Rare Case of Prosthetic Valve Endocarditis Caused by Extended-spectrum β-Lactamase Producing *Escherichia coli*

Sir,

Prosthetic heart valve endocarditis accounts for 20% of cases of infective endocarditis.[1] Endocarditis due to non HACEK (Hemophilus species, *Actinobacillus actinomycetemcomitan, Cardiobacterium hominis, Eikenella corrodens*, Kingella species) Gram negative bacteria is uncommon. Among the Gram negative bacteria, extended-spectrum *β* –lactamase (ESBL) - producing *Escherichia* species causing prosthetic valve endocarditis (PVE) is extremely uncommon.

Our patient was a 62-years-old African American female with history of mitral regurgitation s/p bioprosthetic mitral valve replacement four years ago, left sided hemiplegia

after right basal ganglia infarct and severe penicillin allergy presented to hospital for respiratory distress. Patient was tachypneic, tachycardic, normotensive and febrile with temperature of 39.6°C. On examination, there was newly found pansystolic murmur grade 2/6 at apex of the heart, diffuse expiratory rales and left sided hemiplegia. There were no peripheral stigmata of endocarditis. Her leukocytes count was 13,000/mm^3 with 81% neutrophils, 14% lymphocytes and 5% monocytes. Urinalysis was negative for leukocyte esterase, 1-5 leucocytes/high power field and no bacteria. Roentogenogram showed mild pulmonary congestion. Initial arterial blood gas analysis depicted respiratory acidosis. CT scan of head without contrast showed chronic infarct in right basal ganglia. Patient was admitted to intensive care unit (ICU), supported with non invasive ventilation and treated with intravenous vancomycin and aztreonam for health care associated pneumonia. The Gram-stained smear showed Gram-negative bacilli. On day three, transesophageal echocardiogram (TEE) revealed 5-mm mobile echogenic structure on bioprosthetic mitral valve and mild mitral regurgitation [Figure 1]. The isolate was identified as *E. coli* and the sensitivity results depicted ESBL producer susceptible to carbapenems only. Antibiotics were switched to imipenem-cilastin that improved her clinical condition. Patient was transferred to nursing home with intravenous antibiotic therapy for six weeks. Surgical treatment was not considered because of clinical response to the antibiotic therapy, mild regurgitation of mitral valve and high risk for surgery because of co-morbidities.

E. coli is a common organism to cause bacteremia in hospitalized patients, but the endocarditis due to *E. coli* is rare. The epidemiology and microbiological etiology of PVE have changed and there is a marked increase in PVE caused by Enterococcus.[2] The major risk factors for infections with ESBL-producing organisms are advanced age, female sex, diabetes mellitus, underlying urinary tract infection, prolonged hospital stay, duration of ICU stay and prior exposures to cephalosporins, quinolones and three or more courses of antibiotic therapy within the preceding year.[3] *E. coli* endocarditis is associated with higher rate of intracardiac abscess, sepsis and in-hospital mortality.[4] Modified Duke's criteria have lower sensitivity in diagnosis of PVE.[1] Blood culture is the best identification method providing live bacteria for susceptibility testing. TEE has higher sensitivity and specificity in detecting vegetations, valve abscesses, fistulae, perivalvular regurgitation and prosthetic valve dehiscence.

The carbapenems are commonly used as the drug of choice for severe infections due to ESBL-producing *Enterobacteriaceae*. The presence of ESBL confers resistance to third and fourth-generation cephalosporins and monobactams and co-resistance to fluoroquinolones, tetracyclines, and aminoglycosides.[5] Surgical treatment for PVE is associated with high mortality but the prognosis is better with the early surgery, radical debridement of infected tissue and in presence of complications like heart failure and valve abscesses.[6]

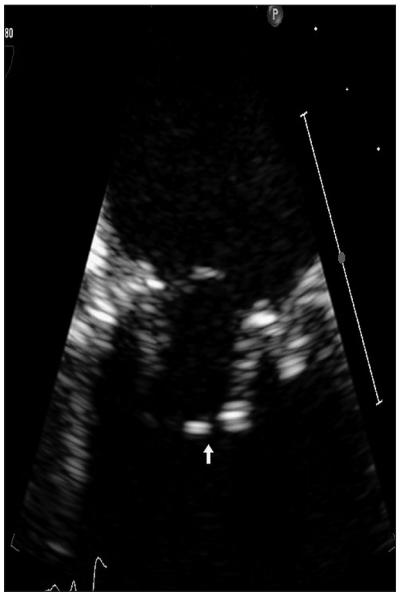

Figure 1: Transesophageal echocardiogram – bioprosthetic mitral valve area showing 5 mm size, mobile, echogenic structure attached to the peripheral rim

ACKNOWLEDGMENT

We sincerely thank Dr. Rajesh Sehgal, Division of Cardiology, Advocate Christ Medical Center for the interpretation of echocardiography images.

Harshit H Modi, Snesha H Modi, Bazgha Rehan Siddiqui, John M Andreoni[1]

Department of Internal Medicine, [1]Division of Infectious Diseases, Advocate Christ Medical Center/ University of Illinois, Chicago

Address for correspondence:
Dr. Harshit H Modi, E-mail: modiharshit@hotmail.com

REFERENCES

1. Authors/Task Force Members, Galiè N, Hoeper MM, Humbert M, Torbicki A, Vachiery JL, *et al.* Guidelines for the diagnosis and treatment of pulmonary hypertension: The Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS), endorsed by the International Society of Heart and Lung Transplantation (ISHLT). Eur Heart J 2009;30:2493-537.

2. Fredlund H, Bjoreman M, Kjellander J, Sjöberg L, Bjorne L, Ohlin AL. A 10-year survey of clinically significant blood culture isolates and antibiotics susceptibilities from adult patients with hematological diseases at a major Swedish hospital. Scand J Infect Dis 1990;22:381-91.
3. Morepth S, Murdoch D, Cabell CH, Karchmer AW, Pappas P, Levine D, *et al*. Non-HACEK gram negative *bacillus* endocarditis. Ann Intern Med 2007;147:829-35.
4. Branger S, Casalta JP, Habib G, Collard F, Raoult D. *Escherichia coli* endocarditis: Seven new cases in adults and review of the literature. Eur J Clin Mirobiol Infect Dis 2005;24:537-41.
5. Livermore DM, Woodford N. The β-lactamase threat in *Enterobacteriaceae*, *Pseudomonas* and *Acinetobacter*. Trends Microbiol 2006;14:413-20.
6. Gutierrez-Martin MA, Galvez-Aceval J, Araji OA. Indications for surgery and operative techniques in infective endocarditis in the present day. Infect Disord Drug Targets 2010;10:32-46.

Access this article online

Quick Response Code:

Website:
www.jgid.org

DOI:
10.4103/0974-777X.77310