Elmer Press

Case Report

J Med Cases. 2015;6(2):51-54

Aortic Insufficiency Secondary to Enterococcus faecalis Endocarditis in an HIV-Positive Patient

Ilana M. Schlam-Camhi[a, c], George Layton[b], Javier Cisneros-Cortes[a], Benjamin A. Goldberg-Dalma[a], Moises Bucay[a]

Abstract

Enterococcus faecalis is the third most common cause of endocarditis. HIV infection is an independent risk factor for the development of endocarditis when the cell count is below 200 cells/mL and without retroviral therapy. A 49-year-old Caucasian male, with HIV infection in triple therapy, no detectable viral counts and CD4 cell count over 1,000 cells/mL, is admitted to the hospital with dyspnea and an acute bacterial infection. On the physical exam, he presented tachycardia, systolic murmur I/IV, bilateral basilar rales, hypotension and fever. The echocardiogram showed an aortic valve vegetation, with prolapse of one of the leaflets, severe aortic insufficiency, mild mitral regurgitation and severe pulmonary hypertension. CXR with bilateral infiltrations and CT scan showed in addition mediastinal lymphadenopathy. He underwent an emergency cardiac surgery replacing his aortic valve for a prosthetic, and the culture of the vegetation was positive for *E. faecalis*. Treatment with vancomicin and gentamicin was initiated. Infective endocarditis by *E. faecalis* in this patient had probably a non-HIV-related etiology because of the adequate antiretroviral therapy that he was having. Most probably the rectal abscess could have been the source of the sepsis.

Keywords: Infective endocarditis; *Enterococcus faecalis*; HIV; Antiretroviral therapy

Introduction

Enterococcus faecalis is the third most common cause of infective endocarditis. HIV infection is an independent risk factor for the development of infective endocarditis when the cell count is below 200 cells/mL and no retroviral therapy. We present a 49-year-old patient with history of HIV infection, in triple therapy with no detectable count and CD4 over 1,000 cells/mL, admitted to the hospital with dyspnea, fever, rapid clinical deterioration, an active rectal abscess, positive bacteremia and echocardiographic evidence of acute aortic endocarditis with severe insufficiency secondary to *E. faecalis* endocarditis. The aggressive medical and surgical treatment is emphasized and the role of underlying HIV infection is discussed.

Case Report

A 49-year-old Caucasian male with history of HIV infection (on triple therapy with no detectable counts and CD4 over 1,000 cell/mL), deep venous thrombosis, trigeminal neuralgia associated to *Herpes zoster*, rectal abscess and hepatitis C, was admitted to the hospital with dyspnea and an acute bacterial infection. He denied alcohol consumption and intravenous drug use, however, admitted to receptive anal intercourse.

On examination, the patient was awake, alert, oriented, no jugular vein distention, tachycardic, a systolic murmur I/VI was present and no hearable diastolic murmur, bilateral basilar rales auscultated and the rest of the exam was normal. His vitals were 89/52 mm Hg, heart rate 104 beats per minute, respiratory rate 20 breaths per minute, temperature 38.2 °C and oxygen saturation 95% on room air.

White count was 13.3 × 10^3/µL, hemoglobin was 10.2 g/dL, MCV was 83.3 fL, platelets was 187,000/µL, pH was 7.38, PCO_2 was 35 mm Hg, PO_2 was 55 mm Hg, sodium was 136 Meq/L, potassium was 3.8 Meq/L, chloride was 101 Meq/L, CO_2 was 24 mmol/L, calcium was 8.2 mg/dL, and albumin was 2.6 g/dL.

An echocardiogram was performed showing a mass on the aortic valve apparently consistent with vegetation with prolapsing of one of the leaflets of the severe aortic insufficiency, mild mitral regurgitation and severe pulmonary hypertension (77 mm Hg). Bilateral infiltrations were noted on chest X-ray. The chest CT scan revealed same bilateral pulmonary infiltrates in the lower lobes and in addition mediastinal lymphadenopathy.

All the findings were consistent with endocarditis with severe aortic valve insufficiency and heart failure; therefore, aortic valve was replaced with a mechanical valve (20 mm

Manuscript accepted for publication September 12, 2014

[a]Cardiology Clinic of San Antonio, Methodist Healthcare System, San Antonio, TX, USA
[b]South Texas Posion Center, Division of Emergency Medicine, University of Texas Health and Science Center, San Antonio TX, USA
[c]Corresponding Author: Ilana Schlam-Camhi, Javier Barros Sierra 245-502 C Santa Fe, 01210, Alvaro Obregon, Mexico City, Mexico.
Email: schlamilana@gmail.com

doi: http://dx.doi.org/10.14740/jmc1946w

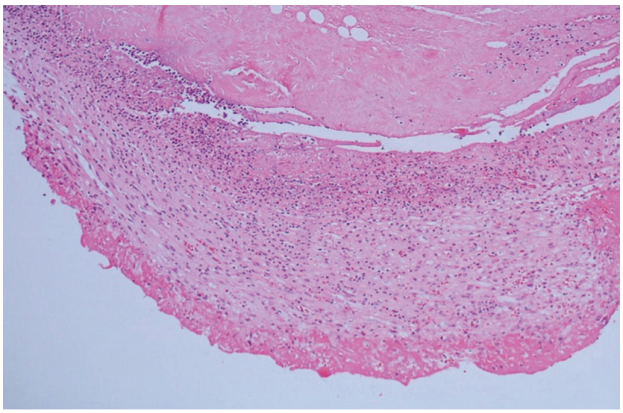

Figure 1. The valve shows inflammatory cells mixed with bacterial colonies (black areas on hematoxylin-eosin stain).

Medtronic Bileaflet). The main surgical findings were a trileaflet aortic valve with a destroyed right coronary cusp and a large prolapsing vegetation (Fig. 1); another vegetation on the non-coronary cusp was noted without evidence of abscesses.

An aortic vegetation culture was done and it showed *E. feacalis*.

Postoperative transesophageal echocardiogram revealed good prosthetic valve function with no paravalvular leaks, even though large bilateral pleural effusions persisted.

The patient's hemodynamic status and symptomatology improved after the surgery and antibiotic treatment with vancomicin and gentamicin.

Discussion

Infective endocarditis is caused by microbial infection of the endothelial lining of intracardiac structures causing persistent bacteremia, usually on one or more heart valve leaflets, but may involve mural endocardium, chordal structures, myocardium, and pericardium. Bacteria that have entered the bloodstream must be able to adhere to the damaged endothelium, exposed extracellular matrix or areas of fibrin deposition. Early diagnosis and aggressive treatment are the critical components of a successful management strategy [1-3].

In the past, HIV infection was associated with immunosuppression; however, in modern times, for patients with the appropriate retroviral treatment, HIV appears to be an independent risk factor for the development of endocarditis when the CD4 count is below 200 cells/mL. Our patient had over 1,000 cells/mL and no detectable viral counts, for that reason, this kind of infection would not be considered and the first possible diagnosis was community acquired pneumonia [1].

A wide range of microorganisms is associated with infec-

Table 1. Duke's Criteria

Major criteria
Two positive blood cultures for a typical microorganism of infective endocarditis
Positive echocardiography (vegetation, myocardial abscess or new partial dehiscence of a prosthetic valve)
New regurgitant murmur
Minor criteria
Presence of a predisposing condition (fever >38 °C)
Embolic disease
Immunological phenomena
Osler nodes
Roth spots
Glomerulonephritis
Rheumatoid factor
Positive blood cultures not meeting the major criteria or serologic evidence of active infection with an organism that causes endocarditis
A definite diagnosis of endocarditis (80% accuracy) is made with:
2 major criteria
1 major criterion and 3 minor criteria
5 minor criteria
Possible endocarditis:
1 major and 1 minor criterion
3 minor criteria

If these criteria thresholds are not met or either an alternative explanation for illness is identified or the patient has defervesce within 4 days, endocarditis is highly unlikely.

Table 2. Enterococcal Endocarditis Medical Treatment

Penicillin	+	Streptomycin or gentamycin (aminoglycoside of choice due to less resistance)
Ampicillin 2 g IV every 4 h	+	Gentamycin 1 mg/kg IV every 8 h
Penicillin-G 3 - 4 × 10^6 units every 4 h	+	Gentamycin 1 mg/kg IV every 8 h
If patient is allergic to penicillin: vancomycin 15 mg/kg IV every 12 h	+	Gentamycin 1 mg/kg IV every 8 h

The recommended duration of combined therapy should be 4 - 6 weeks.

tious endocarditis. The most common are *Streptococcal* species (40%), *Staphylococcus aureus* (28%), and *Enterococci* species (9%). Enterococcal bacteremia is a severe infection with 23-46% of mortality rate, and the most common complication is endocarditis (5-12%), representing the third cause of infectious endocarditis [2, 3].

The *Enterococcus* species formerly classified as group D *Streptococci* is now defined as a distinct genus. These organisms are normal inhabitants of the GI and genitourinary tracts and may enter the bloodstream after manipulation of the colon, urethra, or bladder [1].

The clinical presentation of infective endocarditis is mainly fever (85-99%), with a duration that ranges from a few days to 2 weeks. In 18.9% of patients, there are peripheral lesions such as petechiae, subungueal hemorrages, Osler nodes, Janeway lesions and Roth spots but none of these findings were reported in the patient history [4].

The diagnosis of endocarditis can be done by the Duke's criteria (Table 1). The patient had three major criteria and one minor, therefore granting the diagnosis of endocarditis [5, 6].

The transthoracic echocardiography is 40-63% sensitive. The transesophageal echocardiography is 90-100% sensitive, especially for detecting vegetations, identifying valve ring abscesses and pulmonary and prosthetic valve endocarditis. The electrocardiogram is not helpful for diagnosis but can show conductive abnormalities that suggest formation of a myocardial abscess [5].

In the case of the enterococcal endocarditis, medical treatment, according to the Infectious Diseases Society of America, should be given with cell wall-active agents that raise the permeability of the enterococcal cell wall and act synergically as to permit the aminoglycoside to have a bactericidal effect at a minor dose, thus reducing toxicity for the patient (Table 2). This patient received treatment with vancomicin and gentamicin due to bacterial sensitivities [5, 6].

There are specific indications for surgical treatment; if a valvular regurgitation led to acute heart failure (especially aortic valve involvement), the valve must be replaced, not responding to 7 - 10 days of medical treatment and sinus of Valsalva or septal abscesses [6]. The patient was treated surgically because he developed acute aortic insufficiency and he was hemodynamically unstable.

Conclusion

Infective endocarditis by *E. faecalis* in this patient had probably a non-HIV-related etiology because of the adequate antiretroviral therapy that he was having. Most probably the rectal abscess could have been the source of the sepsis. Hepatitis C infection could have acted as an independent risk factor. Despite HIV infection, other risk factors must be considered with a sepsis diagnosis.

Acknowledgement

We want to thank Dr Thomas Casey (Methodist Healthcare System, San Antonio, TX, USA) for helping with the images.

References

1. O'Gara PT, Haldar SM. Chapter 86. Infective Endocarditis. In: Fuster V, Walsh RA, Harrington RA, eds. Hurst's The Heart. 13th ed. New York: McGraw-Hill; 2011. http://www.accessmedicine.com/content.aspx?aID=7830705. Accessed August 29, 2012.
2. Karchmer AW. Chapter 124. Infective Endocarditis. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J, eds. Harrison's Principles of Internal Medicine. 18th ed. New York: McGraw-Hill; 2012. http://www.accessmedicine.com/content.aspx?aID=9119283. Accessed August 29, 2012.
3. Martinez Odriozola P, Munoz Sanchez J, Arriola Martinez P, Lizarralde Palacios E, Santamaria Jauregui JM, Zuazo Meabe J, Cisterna Cancer R, et al. [Enterococcal infective endocarditis: description of 12 cases]. An Med Interna. 2007;24(11):539-542.
4. Durante-Mangoni E, Bradley S, Selton-Suty C, Tripodi MF, Barsic B, Bouza E, Cabell CH, et al. Current features of infective endocarditis in elderly patients: results of the International Collaboration on Endocarditis Prospective Cohort Study. Arch Intern Med. 2008;168(19):2095-2103.
5. Habib G, Hoen B, Tornos P, Thuny F, Prendergast B, Vilacosta I, Moreillon P, et al. Guidelines on the prevention, diagnosis, and treatment of infective endocarditis (new version 2009): the Task Force on the Prevention, Diagnosis, and Treatment of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and the International Society of Chemotherapy (ISC) for Infection and Cancer. Eur Heart J. 2009;30(19):2369-2413.
6. Baddour LM, Wilson WR, Bayer AS, Fowler VG, Jr.,

Bolger AF, Levison ME, Ferrieri P, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. Circulation. 2005;111(23):e394-434.