Fall 2014

Enterococcus Faecalis Endocarditis: A Case Study

Torri Curtis
Otterbein University, torri.curtis@otterbein.edu

Enterococcus Faecalis Endocarditis: A Case Study

Torri Curtis, BSN, RN, OCN

Otterbein University, Westerville, Ohio

Introduction

Infectious endocarditis is an uncommon disease of the heart stemming from a bacterial or fungal infection on the endocardial surface of the heart or heart valve (Pierce, Calkins, & Thornton, 2012). From review of the literature, infective endocarditis is fairly uncommon and may be difficult to initially diagnose. Per Sabe, Shrestha, and Menon, occurrence of infective endocarditis (IE) is nearly 0.9-6.2 per 100,000 persons per year (2013). Several preexisting conditions such as aging, heart valve disease, implantable cardiac devices, a history of heart disease such as rheumatic fever, and invasive procedures can put a patient at higher risk for IE (Leone et al., 2012). As the occurrence of IE appears to be rare, it is important to emphasize that the complications of this infection are abundant even when treated acutely and appropriately. Per Mantan, Sethi, and Batra, the infection-related complications of IE can include myocardial abscess, meningitis, osteomyelitis, renal abscess, renal infarcts, glomerulonephritis, and purulent pericarditis (2013, p. 368). However, this list of complications is not all inclusive. The risks of thrombolytic events are increased with IE and one can experience a stroke, pulmonary embolism, and even blindness (Mantan et al., 2013). Treatment for infectious endocarditis must be expediential and persistent. Long term intravenous antibiotics are the treatment of choice and surgery may be indicated especially if a patient has a prosthetic valve endocarditis (Sabe et al., 2013).

As infective endocarditis is an uncommon disease, the pathophysiologic impact on the body is significant. Proper diagnosis, management, and long term follow up could be improved through a better understanding of the disease process and systemic effects on the body. As a result, a case study involving a patient diagnosed with Enterococcus faecalis infective endocarditis was chosen for review.

Presentation of Case

Signs and Symptoms

A 54 year old male with a prior medical history of hypertension, pyelonephritis, nephrolithiasis, and acute prostatitis presented to the emergency room in July of 2014 with symptoms of rigors, chills, fever, diplopia, and lower extremity edema and weakness. Upon presentation, temperature was 102.0 F, pulse rate was 98, respirations were 24, blood pressure was 100/58 and oxygen saturation on room air was 90%. Pain level was 7. Skin was diaphoretic and warm to touch. Pupils equal, round, and react to light. Bilateral lower extremity edema present with erythema. Positive Homan's sign. Lab results revealed hemoglobin of 9.3. Blood cultures positive for Enterococcus faecalis. CT scan of abdomen negative. CT scan of brain negative. Chest x-ray normal. EKG was normal. Venous Doppler study negative. Renal ultra sound showed stable nephrolithiasis. Prior to admission, patient had been treated with IV antibiotics of Ampicillin 2 grams IV every 4 hours and Rocefin 1 gram every day for 2 weeks for treatment of positive blood cultures of gram positive cocci. Patient was admitted for fever of unknown origin. IV antibiotics were initiated. Consultations were performed by the infectious disease and cardiology teams. A transthoracic echocardiogram was performed which revealed mitral valve vegetation, minimal mitral valve regurgitation, and a normal ejection fraction of 55-60%. With positive blood cultures for Enterococcus faecalis and mitral valve vegetation by transthoraic echocardiogram, a diagnosis of Enterococcus faecalis endocarditis was made. IV antibiotics of Ampicillin 2 grams every 4 hours and Gentamycin 1 gram every 8 hours were initiated. After blood pressure and fever stabilized and negative blood cultures were obtained, patient was discharged to home on IV Ampicillin 2 grams every 4 hours and IV Gentamycin 1 gram every 8 hours via PICC line for 4 weeks. After completion of antibiotic therapy, patient had follow up with infectious disease physician and cardiologist. A repeat transthoracic echocardiogram was completed which showed anterior mitral valve leaflet thickening with no enlargement of the echo density seen on prior exam and borderline concentric left ventricular hypertrophy with mass not prolapsing into left atrium. Ejection fraction was 55-60%. Patient will have continued follow up exams with a cardiologist and infectious disease physician to monitor and screen for any complications related to infective endocarditis such as recurrent infection, congestive heart failure, valvular dysfunction, or organ dysfunctions. Patient was also instructed to report any fever greater than 100.0 F immediately. Patient was also instructed to have antibiotic therapy prophylactically prior to dental appointments or invasive procedures.

Underlying Pathophysiology

An infection caused by bacteria that enters the bloodstream and settles in the heart lining, valve, or blood vessel is endocarditis. As bacteria is the most common cause of IE, viruses, fungi, and parasites can also cause IE (McCance, Huether, Brashers, & Rote, 2014). Blood cultures commonly isolated for IE are Staphylococcus aureus, viridans Streptococcus, and Enterococcus faecalis (Pierce et al., 2012). Enterococcus faecalis is the third most common cause of infective endocarditis and the mortality and morbidity is high (Miro, Pericas, & Del Rio, 2014). Although infective endocarditis refers specifically to infection in the lining of the heart, IE also affects the valves and muscles of the heart ("American Heart Association," 2014).

The pathophysiology of IE includes three elements of endocardial damage, blood-borne microorganism adherence to the endocardial surface that is damaged, and the formation of infective endocardial vegetations (McCance et al., 2014). Endocardial damage can include trauma, heart disease, prosthetic valves and valvular heart disease (McCance et al., 2014). IE also requires the presence of a microorganism in the blood and the attachment to the intracardiac surface (Pierce et al., 2012). This can occur by mode of trauma, dental procedures, cardiac surgery, and central line infections. The infective endocardial vegetations infiltrate the thrombi in the cardiac tissue and then accelerate fibrin formation by activating the clotting cascade as the bacterial colonies are rooted in the fibrin clots; making them inaccessible to host defenses (McCance et al., 2014). After the microorganism settles on the cardiac surface, the infection can disseminate to other tissues in the body causing a systemic infection ("National Institutes of Health," 2010).

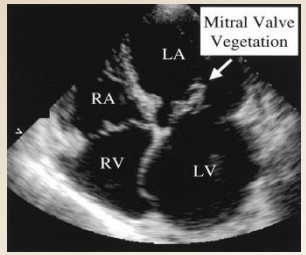

Figure 1: Infectious Endocarditis of Mitral Valve (Cabell, Abrutyn, & Karchmer, 2003, figure 1).

Significance of Pathophysiology

Infective endocarditis is uncommon, but the effects from the disease process can involve several organ systems including cardiac, neurological, musculoskeletal and renal systems and can be fatal if not treated in an expedited fashion. Infection related complications of IE can include embolism, heart valve destruction and heart failure, osteomyelitis, pericarditis, glomerulonephritis, and blindness (Mantan et al., 2013). Embolic risk occurs in 22%-50% of patients with IE and involve complication of stroke and pulmonary embolism (Anavekar et al., 2011). Emboli from IE can often involve the lungs, coronary arteries, spleen, GI system, extremities, and central nervous system (Chung, Chen, Tai, Huang, & Chen, 2014). Embolic events related to IE can cause devastating neurologic damage and organ failure (Sabe et al., 2013). Cardiac complications are serious and include formations of heart murmurs, heart valve regurgitation, and congestive heart failure (Mocchegian & Nataloni, 2009). Additional complications include damage to the conduction system of the heart leading to bundle branch block, intra-cardiac fistulas, and pericarditis (Sabe et al., 2013). Heart valve replacement may need to be a consideration if patient has extensive damage to a heart valve. Renal complications of IE occurs in approximately 25% of the patients and the most common diagnosis are renal infarcts, renal abscesses, glomerulonephritis and interstitial nephritis (Mantan et al., 2013).

Implications for Nursing Care

The clinical manifestations and multi-organ involvement of IE are many. Diagnosis of IE is based on clinical, microbiologic, and echocardiographic findings (Chung et al., 2014). Evaluation and work up for diagnosis of IE should include cardiac assessment for heart murmur or heart failure which would include a baseline electrocardiogram, an echocardiogram, blood cultures prior to initiation of antibiotic therapy, evaluation for petechiae of mucous membranes and extremities, complete blood count, serum electrolytes, and urinalysis for hematuria, proteinuria, or pyuria (Pierce et al., 2012). The principles of therapy for IE is initial hemodynamic stabilization, obtaining blood cultures for microbial identification and treatment (Sabe et al., 2013). Echocardiography is also one diagnostic modality in patients with suspected IE. Evaluation of IE has improved dramatically after the introduction of echocardiography which is the primary technique for the detection of vegetations on the heart wall and valves and IE related complications (Cecchi et al., 2013). Clinical judgment is crucial in the first stages of care for a patient with IE. Systematic evaluation and assessment is essential and nurses evaluating patients can be advocates in the initiation of appropriate tests and evaluations. Although uncommon, knowing predictive factors for IE is useful in identifying patients with IE. These factors include: being male, prior valvular heart damage, fever, stroke, emboli, splenomegaly, leukocytosis, and thrombocytopenia (Chung et al., 2014). The latest American Heart Association guidelines recommend penicillin or ampicillin plus gentamicin for 4 to 6 weeks as the combination of choice for Enterococcus faecalis infective endocarditis (Miro et al., 2014). It is also imperative to have an infectious disease consultation for the patient in addition to a cardiology consult. ID consultation has been associated with improved outcomes including improved survival (Yamamoto et al., 2012). Follow up and patient education is important. Intravenous catheters should be removed immediately following completion of antibiotic therapy and transthoracic echocardiography should be performed to establish a new baseline result for the patient (Pierce et al., 2012). Patients should also receive education regarding the importance of regular follow up visits to their specialists including infectious disease and cardiology. (Pierce et al., 2012). In addition, patients need to know that they may require antibiotic therapy prior to invasive procedures. The American Heart Association recommends antibiotic prophylaxis prior to dental procedure for patients with a history of IE ("American Heart Association," 2014).

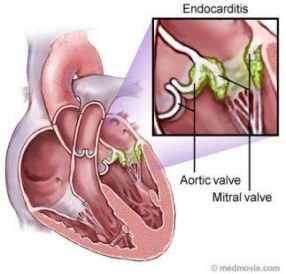

Figure 2: Infectious Endocarditis of Mitral Valve (American Heart Association website, 2008).

Conclusions

Infective endocarditis occurs at a low rate, but can be associated with high morbidity and mortality, with mortality occurring at a rate of 15-40%, in light of antibiotic and surgical advancements (Sabe et al., 2013). Early diagnosis of IE is crucial for successful clinical outcomes and prevention of long term complications. Treatment of IE should be targeted toward the causative microorganism, based on the cardiac valve involved, and the clinical status of the patient (Sabe et al., 2013). A multimodality approach in care and treatment is essential and should include a collaborative effort between the infectious disease specialist, primary care physician, cardiologist, cardiothoracic surgeons, and nursing team. Through enhanced awareness of IE, improved outcomes from this disease can be realized.

References

American Heart Association website. (2008). http://hearthealth.aha-krames.com/RelatedItems/77,AHADC7

Anavekar, N. S., Schultz, J. C., Correa De Sa, D. D., Thomas, J. M., Lahr, D., Tleyjeh, I. M., ... Baddour, L. M. (2011, November). Modifiers of symptomatic embolic risk in infective endocarditis. *Mayo Clinic Proceedings*, *86*, 1068-1074.

Cabell, C. H., Abrutyn, E., & Karchmer, A. W. (2003). Bacterial Endocarditis the disease, treatment, and prevention. Retrieved from http://circ.ahajournals.org/content/107/20/e185.figures-only

Cecchi, E., Chirillo, F., Faggiano, P., Imazio, M., Cecconi, M., Morio, A., ... Belli, R. (2013). The diagnostic utility of transthoracic echocardiography for the diagnosis of infective endocarditis in the real world of the italian registry on infective endocarditis. *Echocardiography*, *30*, 871-879.

Chung, J., Chen, J., Tai, H., Huang, P., & Chen, W. (2014, February). Lower leg weakness as the presentation of infective endocarditis with septic emboli. American Journal of Emergency Medicine

Heart valves and infective endocarditis. (2014). Retrieved from http://www.heart.org/HEARTORG/Conditions/More/HeartValveProblemsandDisease/Heart-Valves-and-Infective-Endocarditis_UCM_450448_Article.jsp

Leone, S., Ravasio, V., Durante-Mangoni, W., Crapis, M., Carosi, G., Scotton, P. G., ... Suter, F. (2012). Epidemiology, characteristics, and outcome of infective endocarditis in Italy: The italian study on endocarditis. *Infection*, *40*, 527-535.

Mantan, M., Sethi, G. R., & Batra, V. V. (2013, September). Post-infectious glomerulonephritis following infective endocarditis: Amenable to immunosuppression. *Indian Journal of Nephrology*, *23*, 368-370.

McCance, K. L., Huether, S. E., Brashers, V. L., & Rote, N. S. (2014). *Pathophysiology the biologic basis for disease in adults and children* (7th ed.). St. Louis, Missouri: Elsevier Mosby.

Miro, J. M., Pericas, J. M., & Del Rio, A. (2014, September). A new era for treating enterococcus faecalis endocarditis Ampicillin plus short-course Gentamicin or Ampicillin plus Ceftriaxone: That is the question! *Circulation*, 1763-1766.

Mocchegian, R., & Nataloni, M. (2009, December). Complications of infective endocarditis. *Cardiovascular & Hematological Disorders-Drug Targets*, *9*, 240-248.

Pierce, D., Calkins, B. C., & Thornton, K. (2012, May 15). Infectious endocarditis: Diagnosis and treatment. *American Family Physician*, *85*, 981-986.

Sabe, M. A., Shrestha, N. K., & Menon, V. (2013). Contemporary drug treatment of infective endocarditis. *American Journal of Cardiovascular Drugs*, *13*, 251-258.

What is endocarditis? (2010). Retrieved from http://www.nhlbi.nih.gov/health/health-topics/topics/endo/

Yamamoto, S., Hosokawa, N., Sogi, M., Inakaku, M., Imoto, K., OhjP, G., ... Iwata, K. (2012). Impact of infectious diseases service consultation on diagnosis of infective endocarditis. *Scandinavian Journal of Infectious Diseases*, *44*, 270-275.

OTTERBEIN UNIVERSITY