Pathophysiology of Infective Endocarditis

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What Is Infective Endocarditis?

I have selected IE because I currently work in the Cardio Thoracic Intensive Care Unit (CTICU) and we have frequent open heart surgery patients that develop infective endocarditis for different reasons ranging from poor oral hygiene to unwary use of intravenous drugs. The pathology behind the infection as well as the multitude of issues that occur has always interested me. As I continue my career in nursing anesthesia I will have to perform sedation for these patients that require surgery as a treatment option and understanding the pathological process behind their infection and potential heart valve failure will be beneficial.

Introduction

Pathogen(s) can gain access to the bloodstream via intravenous catheter, injection drug use, or a dental infection for example (Holland et al., 2016). The original site adheres to an area of abnormal cardiac valve surface; the infected vegetation is created by the proliferation of the pathogen within a protective matrix or serous membranes after 2 months (Holland et al., 2016). The variability in clinical signs and symptoms includes chest pain, non-specific fever, lymphadenopathy, malaise, fatigue, weight loss, and anemia (Holland et al., 2016). Analysis of the infecter determines the appropriate empirical antibiotic therapy and to identify patients at high risk for complications who may be best managed by early surgery (Baddour et al., 2015). The signs of infection can be managed by the host in an effort to control the ongoing infection (Holland et al., 2016).

Signs and Symptoms

Classic history and clinical ophthalmologic manifestations: sustained bacteremia or fungemia, evidence of active valvulitis, peripheral embolism, and immunological vascular phenomena (Baddour et al., 2015). Septic embolism includes digital, subcutaneous, and intravenous. Embolism is due to the formation of microemboli that travel to the lungs, and extremities, and promotes the inflammatory endothelial cells to release tissue factor and cytokines, triggering a coagulation cascade (Holland et al., 2016). The identification of the presence of microemboli is suggestive of IE. The immune cells that express integrins that bind to the endothelial cells become activated causing the coagulum and colonizing, the presence of microemboli (Holland et al., 2016). According to Holland et al. (2016), "equivalent damage to the valvular surface may result from a variety of factors, including turbulent blood flow related to primary valvular damage from specific systemic diseases (such as rheumatic carditis), mechanical injury by catheterization, radiation, or trauma, or from repeated injections of solid particles in IDU. This endothelial damage prompts the formation of fibrin-platelet deposits overlaying interstitial edema" (Holland et al., 2016). The formation of fibrin-platelet deposits overlaying interstitial edema (p. 3). Pathogenesis of IE includes valvular regurgitation, infection of the heart valves, and the presence of microemboli that travel to the lungs, and extremities, and promotes the inflammatory endothelial cells to release tissue factor and cytokines, triggering a coagulation cascade (Holland et al., 2016). The identification of the presence of microemboli is suggestive of IE. The immune cells that express integrins that bind to the endothelial cells become activated causing the coagulum and colonizing, the presence of microemboli. The infection of the cardiac valves is a result of the continuous bacteremia that typically characterizes IE. Opsonic antibodies, engulfing antibodies, complement- binding antibodies, cryoglobulins and antiplatelet antibodies, and cryoglobulins directed against bacterial heart-attack proteins and membrane-attack complex-processed products produced by the host in an effort to control the primary infection (Holland et al., 2016).

Pathophysiological Processes of Infective Endocarditis

I. Underlying Pathophysiology

IE results in stimulation of both humoral and cellular immunity, as manifested by hypergammaglobulinaemia, splenomegaly and the presence of microemboli in the peripheral blood (Holland et al., 2016). The variability in clinical signs and symptoms includes chest pain, non-specific fever, lymphadenopathy, malaise, fatigue, weight loss, and anemia (Holland et al., 2016). The variability in clinical signs and symptoms includes chest pain, non-specific fever, lymphadenopathy, malaise, fatigue, weight loss, and anemia (Holland et al., 2016). According to Holland et al. (2016), "equivalent damage to the valvular surface may result from a variety of factors, including turbulent blood flow related to primary valvular damage from specific systemic diseases (such as rheumatic carditis), mechanical injury by catheterization, radiation, or trauma, or from repeated injections of solid particles in IDU. This endothelial damage prompts the formation of fibrin-platelet deposits overlaying interstitial edema" (Holland et al., 2016). The formation of fibrin-platelet deposits overlaying interstitial edema (p. 3). Pathogenesis of IE includes valvular regurgitation, infection of the heart valves, and the presence of microemboli that travel to the lungs, and extremities, and promotes the inflammatory endothelial cells to release tissue factor and cytokines, triggering a coagulation cascade (Holland et al., 2016). The identification of the presence of microemboli is suggestive of IE. The immune cells that express integrins that bind to the endothelial cells become activated causing the coagulum and colonizing, the presence of microemboli. The infection of the cardiac valves is a result of the continuous bacteremia that typically characterizes IE. Opsonic antibodies, engulfing antibodies, complement- binding antibodies, cryoglobulins and antiplatelet antibodies, and cryoglobulins directed against bacterial heart-attack proteins and membrane-attack complex-processed products produced by the host in an effort to control the primary infection (Holland et al., 2016).

II. Implications For Nursing Care

The variability in clinical presentation of IE and the importance of early accurate diagnosis require a diagnostic strategy that is both sensitive for disease detection and specific for IE. "An epidemiological profile of infective endocarditis" (Baddour et al., 2015). The variability in clinical signs and symptoms includes chest pain, non-specific fever, lymphadenopathy, malaise, fatigue, weight loss, and anemia (Holland et al., 2016). The variability in clinical signs and symptoms includes chest pain, non-specific fever, lymphadenopathy, malaise, fatigue, weight loss, and anemia (Holland et al., 2016). According to Holland et al. (2016), "equivalent damage to the valvular surface may result from a variety of factors, including turbulent blood flow related to primary valvular damage from specific systemic diseases (such as rheumatic carditis), mechanical injury by catheterization, radiation, or trauma, or from repeated injections of solid particles in IDU. This endothelial damage prompts the formation of fibrin-platelet deposits overlaying interstitial edema" (Holland et al., 2016). The formation of fibrin-platelet deposits overlaying interstitial edema (p. 3). Pathogenesis of IE includes valvular regurgitation, infection of the heart valves, and the presence of microemboli that travel to the lungs, and extremities, and promotes the inflammatory endothelial cells to release tissue factor and cytokines, triggering a coagulation cascade (Holland et al., 2016). The identification of the presence of microemboli is suggestive of IE. The immune cells that express integrins that bind to the endothelial cells become activated causing the coagulum and colonizing, the presence of microemboli. The infection of the cardiac valves is a result of the continuous bacteremia that typically characterizes IE. Opsonic antibodies, engulfing antibodies, complement- binding antibodies, cryoglobulins and antiplatelet antibodies, and cryoglobulins directed against bacterial heart-attack proteins and membrane-attack complex-processed products produced by the host in an effort to control the primary infection (Holland et al., 2016).

Significance of Pathophysiology

Determine the source and characteristics of the infection is vital in the effective treatment of IE. According to Cama et al. (2018), “the epidemiological profile of IE in Western countries has changed dramatically over the last years because of the increased scarcity of rheumatic fever, the ageing of the population, and the emergence of new risk groups” (p. 4). Ensuring that the interdisciplinary team is properly educated on the management and recognition of IE is vital to positive outcomes of the patients. It can affect a diverse group of patients, spanning from intravenous drug user to an elderly man after open heart surgery. Understanding the pathophysiology of IE helps healthcare providers in providing the appropriate treatment plan.

Gross

Antibiotic

Dosage 

Weeks 

Prophylaxis

Streptococcus viridans susceptible to penicillin

Penicillin G or amoxyillin

30-60 U/kg/d, or clindamycin

6 weeks 

No

Streptococcus viridans resistant to penicillin

Clindamycin or amoxyillin

30-60 U/kg/d, or clindamycin

6 weeks 

No

Staphylococcus aureus

Methicillin-resistant

Vancomycin or oxacillin

20-30 mg/kg/d 

2-4 weeks 

No

Vancomycin or oxacillin

20-30 mg/kg/d 

No

Endocarditis, due to streptococcal infections, or -nonbacterial thrombotic endocardritis (NBT)

Vancomycin or oxacillin

20-30 mg/kg/d 

4 weeks 

Yes

Endocarditis, due to streptococcal infections, or -nonbacterial thrombotic endocardritis (NBT)

Vancomycin or oxacillin

20-30 mg/kg/d 

6 weeks 

Yes

Allograft is in bicuspid or mitral mitral valve

Vancomycin or oxacillin

20-30 mg/kg/d 

6 weeks 

Yes

Infective Endocarditis Antibiotic Treatments (Cobo Molinos, 2018)

Figure 1. Infective Endocarditis (Liewman, Pritik, Mokoveni, & Patel, 2018)

Figure 3. Pathophysiological processes of infective endocarditis.