Pathophysiology of Infective Endocarditis

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**What Is Infective Endocarditis?**

According to Cobo Molinos (2018), “Infective endocarditis (IE) is a disease caused by a microbial infection of the cardiac endocardium. The infection, with involvement of the heart valves and endocardium, are characteristic” (p. 1). IE generally develops in patients with a previous injury of the cardiac endocardium. It is a rare disease that is difficult to identify and diagnose, which requires the collection of the characteristics of different specialists such as intensive care specialists, infectious disease specialists, microbiologists, cardiologists, and surgery specialists (Cobo Molinos, 2018). It can also present in patients with no history of heart disease due to the ability of the bacteria to come from different sources such as a wound, injury to the heart valve, or surgical to name a few (Baddour et al., 2015). According to Cobo Molinos (2018), “the following four categories should be included according to the location of the infection and the presence or absence of endocardial foreign material. IE can affect native valve, IE on left prosthetic valve, right IE, and related IE with the device. In some of the type of presentation of the disease, the following situations can be identified: IE acquired in the community is associated with healthcare (nosocomial and non- nosocomial) IE. IE is caused by drug addicts” (p. 2). With the multitude of causes and outcomes of IE, it is difficult to diagnose, treat, and prevent.

**Introduction**

I have selected IE because I currently work in the Cardiac Thoracic Intensive Care Unit (CTU) and I can frequently open-heart surgery patients that develop infective endocarditis for different reasons ranging from poor oral hygiene to susceptibility of intravenous drugs. The pathology behind the infection as well as the multitude of isues that occur has always interested me. As I continue my career in nurse anesthesia I will have to perform sedation for these patients that are required surgery as a treatment option and understanding the pathological process behind their infection and potential heart valve failure will be beneficial.

**Signs and Symptoms**

- **Classic history and clinical manifestations:** sustained bacteremia or fungemia, evidence of active valvular, peripheral emboli, and immunological vascular phenomena (Baddour et al., 2015).
- **Septic emboli include:** digital gangrene and pulmonary emboli (Holland et al., 2016).
- **Laminated aneurysm:** formation (Holland et al., 2016).
- **Pectechiae, splinter hemorrhage, intracranial bleeding, and conjunctival hemorrhage:** (Cobo Molinos, 2018).
- **Ooze’s nodes:** can present which are painful raised lesions on pads of fingers and toes (Holland et al., 2016).
- **Cough, fever, and chest pain:** can present in patients with IE (Baddour et al., 2015).

**Pathophysiological Processes of Infective Endocarditis**

Pathogens can gain access to the bloodstream via intravenous catheter, injection drug use, or a dental infection for example (Holland et al., 2016). These pathogens then adhere to an area of abnormal cardiac valve surface. The infected vegetation is created by burying of the proliferating organism within a protective matrix or serum molecules (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 disease states (such as rheumatic cardicardia), mechanical injury by catheters or other foreign bodies, or arising from repeated injections of solid particles in IDU. This endothelial damage prompts the formation of fibron-platelet deposits overlaying interstitial edema” (p. 3). Holland et al. (2016) elaborates, “the development of IE requires the simultaneous occurrence of several independent factors: alteration of the cardiac valve surface to produce a suitable site for bacterial attachment and colonization; bacteria with an organism capable of attaching to and colonizing valve tissue, and creation of the infected mass or ‘vegetation’ by ‘burying’ of the proliferating organism within a protective matrix of serum molecules (for example, fibrin) and platelets” (p. 3). The bacterial vegetation that develops on a damaged cardiac valve surface can detach and cause ischemic damage to various areas or organs including the brain, lungs, and extremities (Holland et al., 2016).

**Underlying Pathophysiology**

- **IE results in stimulation of both humoral and cellular immunity, as manifested by hypergammaglobulinemia, splenomegaly and the presence of macrophages in the peripheral blood (Holland et al., 2016).**
- Several classes of circulating antibodies are produced in response to the continuous bacteremia that typically characterize IE. Opaque antibodies, agglutinating antibodies, complement-binding antibodies, complement-fixing antibodies and antiblood produce antibodies directed against bacterial heart-streptococcal and monoclonal, may be produced by the host in an effort to control the ongoing infection (Holland et al., 2016).

**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 localization of the source of the disease (Baddour et al., 2015).**
- **The diagnostic of IE must be made as soon as possible to initiate appropriate empirical antibiotic therapy and to identify at patients with high risk for complications who may be best managed by early surgery (Baddour et al., 2015).**
- **Input of an interdisciplinary team is efficiently by nursing to help prevent and manage IE should be used to assure the mandatory for the patient to return for recurrent IE (Baddour et al., 2015).**
- **Identify and manage the various signs and symptoms associated with IE in the unpredictable nature of septic embolism (Holland et al., 2016).**
- **Educating those at increased risk for developing IE with appropriate prophylactic heart valve valve and other invasive surgeries.**

**Conclusion**

IE is a serious condition that needs immediate attention. According to Camma et al. (2018), “the epidemiological profile of IE in Western countries has changed dramatically over the last 50 years because of the marked scarcity of rheumatic fever, the aging of the population, and the emergence of new risk groups” (p. 4). Ensuring that the interdisciplinary team is properly trained and educated on the management and recognition of IE is vital to positive outcomes of the patients. It can affect a diverse group ranging from intravenous drug users to an elderly man after open heart surgery. Exploring the pathophysiology of IE helps healthcare providers in providing the appropriate treatment plan.

**References**


**Pathogen Table**

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**Figures**

Figure 3. Mechanisms of Infective Endocarditis (Holland et al., 2016).  
- Valve colonization due to mechanical injury involves binding to the valve’s surface. Antibodies that are monoclonal release tissue factor and cytokines, as a result more platelets are attract and become activated causing the vegetation to grow (Holland et al., 2016).  
- Valve colonization as a consequence of an inflammatory endothelial lesion involves activated endothelial cells that express integrins that promote the local deposition of fibrinogen (Holland et al., 2016).  
- Bacterial infection and endothelial cells release tissue factor and cytokines, causing blood clotting and promoting the extension of inflammation and vegetation formation (Holland et al., 2016).

**Figures**

Figure 4. Mechanisms of Infective Endocarditis (Holland et al., 2016).  
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