7-25-2018

Pathophysiology of Infective Endocarditis

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Infective Endocarditis

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What Is Infective Endocarditis?

According to Cobo Molinos (2018), "Infectious endocarditis (IE) is a disease caused by a microbial infection of the cardiac endothelium. The vegetation, with involvement of the heart valves and endocardium, are characteristic" (p. 1) IE generally develops in patients with a previous injury of the cardiac endothelium. IE is a rare disease that is difficult to identify and diagnose, which requires the collaborative efforts of different specialist such as intensive care specialists, infectious disease specialists, microbiologists, cardiologists, and surgery specialists (Cobo Molinos, 2018). IE 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 oral, wound, or surgical to name a few (Baddour et al., 2015). According to Cobo Molinos (2018), "the following four IE categories should be separated according to the location of the infection and the presence or absence of intracardiac foreign material: IE on left native valve ,IE on left prosthetic valve, right IE, and related IE with the devices. In relation to the type of acquisition of the disease, the following situations can be identified: IE acquired in the community, IE associated with healthcare (nosocomial and non-nosocomial), and IE in injecting drug addicts" (p. 2). With the multitude of categories and causes IE is a difficult illness to diagnose, treat, and prevent.

Introduction

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

Signs and Symptoms

- Consistent history and classic oslerian manifestations: sustained bacteremia or fungemia, evidence of active valvulitis, peripheral emboli, and immunological vascular phenomena (Baddour et al., 2015).
- Septic emboli including digital gangrene and pulmonary emboli (Holland et al., 2016).
- Mycotic aneurysms (Holland et al., 2016)
- Petechiae, splinter hemorrhage, intracranial bleeding, and conjunctival hemorrhage (Cobo Molinos, 2018).
- Osler's nodes can be 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)

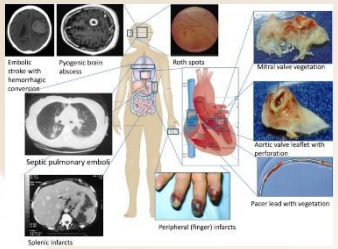

Figure 4. Mechanisms of Infective Endocarditis (Holland et al., 2016)

Germ	Antibiotic	Dosage	Weeks	Prosthetics
Streptococci (susceptible to penicillin)	Penicillin G or ceftriaxone	50,000 U/kg/6h 100 mg/kg/24h	4 weeks	No
Streptococci (relatively resistant to penicillin)	Penicillin G or ceftriaxone plus gentamicin	50,000 U/kg/4h 100 mg/kg/24h 1 mg/kg/8h	2 to 4 weeks	No
Enterococci, Strep. viridans, Abiotrophia sp., Streptococci (resistant to penicillin)	Penicillin G plus gentamicin	50,000 U/kg/4h 1 mg/kg/8h	4 to 6 weeks	No
Streptococci, Enterococci, or Abiotrophia	Vancomycin or vancomycin plus gentamicin	20 mg/kg/12h 20 mg/kg/12h 1 mg/kg/8h	4 to 6 weeks	Yes
Enterococci Streptococci, or Abiotrophia	Vancomycin plus gentamicin	20 mg/kg/12h 1 mg/kg/8h	6 weeks	Yes
Allergic to beta-lactam or resistant to methicillin	Vancomycin	20 mg/kg/12h	6 weeks	Yes

Infective Endocarditis Antibiotic Treatments (Cobo Molinos, 2018)

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 matric of 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 carditis), mechanical injury by catheters or electrodes, or injury arising from repeated injections of solid particles in IDU. This endothelial damage prompts the formation of fibrin-platelet deposits overlying 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; bacteremia 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 the 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)

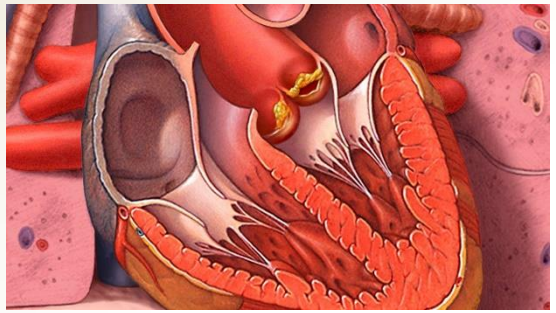

Figure 1. Infective Endocarditis (Liesman, Pritt, Maleszewski, & Patel, 2018)

Significance of Pathophysiology

Determining the source and characteristics of the infection is vital in the effectiveness of the treatment. Baddour et al. (2014) states, "at least 3 sets of blood cultures obtained from different venipuncture sites should be obtained, with the first and last samples drawn at least 1 hour apart (p. 1439). It is important in this time that effective treatment of sepsis or other signs and symptoms related to IE are being appropriately managed by the interdisciplinary team.

Underlying Pathophysiology

- IE results in stimulation of both humoral and cellular immunity, as manifested by hypergammaglobulinaemia, 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 characterizes IE. Opsonic antibodies, agglutinating antibodies, complement-fixing antibodies, cryoglobulins and antibodies directed against bacterial heat-shock proteins and macroglobulin's are produced by the host in an effort to control the ongoing infection (Holland et al., 2016).

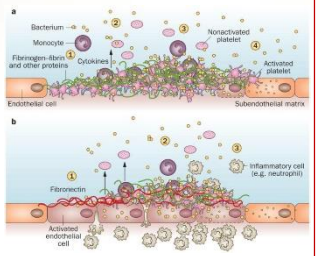

Figure 3. Mechanisms of Infective Endocarditis (Holland et al., 2016)

- Valve colonization due to mechanical injury involves bacteria binding to the coagulum and colonizing, the adhered monocytes release tissue factor and cytokines, as a results 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 fibronectin (Holland et al., 2016). Bacteria are internalized and endothelial cells release tissue factor and cytokines, causing blood clotting and promoting the extension of inflammation and vegetation formation (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 its exclusion across all forms of the disease (Baddour et al., 2015)
- The diagnosis of IE must be made as soon as possible to initiate 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)
- Inpatients and outpatients with IE should be educated by nursing to help prevent and manage oral diseases that predispose to bacteremia and may therefore contribute to the risk for recurrent IE (Baddour et al., 2015).
- Identify and manage the various signs and symptoms associated with IE including the unpredictable nature of septic emboli (Holland et al., 2016).
- Educating those at increased risk for developing IE including post-operative prosthetic heart valve and other invasive surgeries.

Conclusion

IE is a serious condition that is on the rise. According to Camou 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 aging 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. IE can affect a diverse group of patients ranging from intravenous drug user to an elderly man after open heart surgery. Exploring the pathophysiology of IE helps healthcare members in providing the appropriate treatment plan.

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