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Pathophysiology of Infective Endocarditis (IE) Associated with Intravenous Drug Use

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Introduction

- Intravenous drug use (IVDU) poses many risks to those who use them. One of the risks being infective endocarditis.
 - "Infective endocarditis (IE) is an infectious microbial disease of the heart valvular endothelium.
- The characteristic lesion consists of the presence of vegetations that can settle in the valvular veils, tendon cords or mural endocardium" (Molinos, 2018, p. 1).
- Complications of IE include: congestive heart failure, embolization of septic emboli to other organs, periannular extension of infection, metastatic foci of infection, mycotic aneurysms, and anticoagulation issues (Baddour, et. al., 2015, pp. 32-37).
- Contemporary multicentre assessments of outcomes in IE have estimated inpatient mortality between 14% and 18% (Tung et. al, 2015, p.412).

Signs & Symptoms

Most Common

- High fever (usually above 100.4°F)
- Chills
- Night sweats
- Headaches
- Shortness of breath
- Cough
- Cardiac murmurs
- Fatigue
- Muscle and joint pain

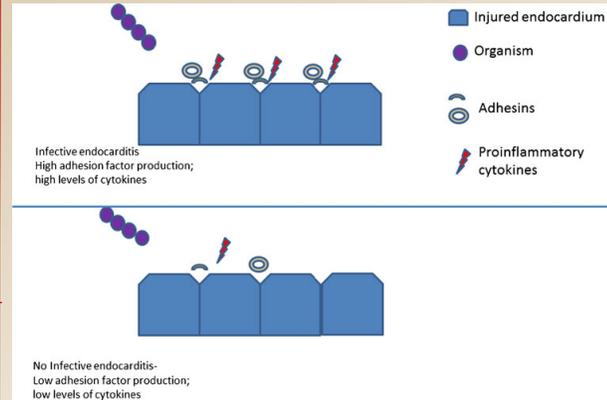
Other Symptoms



Image retrieved from: https://thoracickey.com/wp-content/uploads/2016/07/B978145574860000025_6_025-002-9781455748600.jpg

Underlying Pathophysiology

Underlying Lesion: Degenerative processes such as fibrosis, calcification, congenital heart diseases, mechanical lesions secondary to material implantation, and turbulent blood flows (IVDU) causes endocardial damage. This then exposes the extracellular matrix and results in apoptosis of endocardial cells which then produces tissue factor and a non-bacterial thrombus formation. This thrombotic formation is known as a "vegetation." (Thuny, 2016, p.1)



Upper panel: Infective endocarditis producing organism, increased production of adhesion factors, leading to increased aggregation and proinflammatory cytokine production with propagation of vegetation.

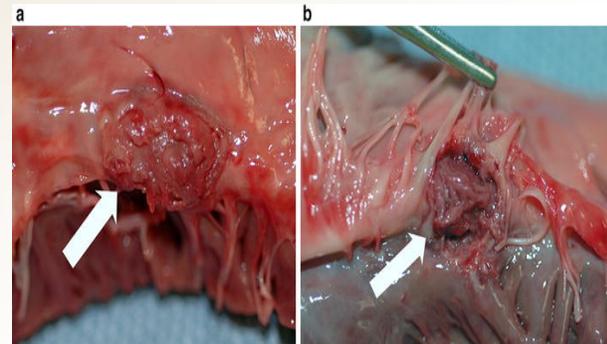
Lower panel: Organism possessing decreased adhesion capacity, leading to lower levels of inflammation and no endocarditis.

Image retrieved from: Keynan, Y., & Rubinstein, E. (2013)

Host-Pathogen Interaction: Pathogens adhere to the exposed extracellular matrix proteins, referred to as Microbial Surface Component Reacting with Adhesive Matric Molecules, released by the non-bacterial thrombotic vegetation. After the pathogen adheres to the vegetation, colonization and invasion of the myocardium occurs and maintains the inflammatory and coagulation processes. The vegetation will continue to grow (neoangiogenesis) and destroy the valve tissue. (Thuny, 2016, p. 1-2)

A destructive lesion (arrow) of the mitral valve of the heart and b viewed from its undersurface

Image retrieved from: Lapkus, M., & Prahlow, J. A. (2015)



- In the instance of IVDU, the pathogen is introduced into the host via bacteria located on the skin, the needle, or the substance being injected. The key factor required for IVDU to cause IE is myocardial tissue damage that has produced a vegetation. Research shows that chronic IVDU can cause non-bacterial vegetations on heart valves due to the repetitive damage from injected particulate matter (UpToDate, 2018)
- According to Lapkus and Prahlow (2015), "the risk of IE in IVDU is estimated at 2%-5% per year (p. 610)
- "Staphylococcus aureus accounts for more than one-half of cases of IE among IVDUs. Streptococci and enterococci are the next most common pathogens. Less commonly, fungi and gram-negative bacilli can also cause IE in IVDUs" (UpToDate, 2018).

Significance of Pathophysiology

IE is a significant diagnosis because of its ability to affect almost every vital body system.

- Heart Failure** - Heart failure is the most common complication from IE and can often lead to surgical intervention (Habib, et. al, 2015, p. 26). Myocardial tissue death from a bacterial vegetation causes valvular insufficiency and regurgitation, leading to heart failure.
- Uncontrolled Infection** - IE can lead to a persisting infection (positive blood cultures for greater than 7-10 days) and a perivalvular extension of the original infection. Some pathogens in IE can be resistant to antibiotics and be even more difficult to treat. According to Habib, et. al., (2015), "Perivalvular extension of IE is the most frequent cause of uncontrolled infection and is associated with a poor prognosis and high likelihood of the need for surgery" (p. 26).
- Embolism** - Embolic events are very frequent (up to 25% have already occurred upon diagnosis of IE) and can be fatal. Splenic and brain emboli are the most common site with left-sided IE. Pulmonary embolism and pacer lead embolism are the most common type with right-sided IE. The risk of embolism falls from 20-50% to 6-12% after IV antibiotic initiation (Habib et. al., 2015, p. 28). Therefore, early diagnosis and initiation of IV antibiotics in IE is imperative.
- Neurological complications** - According to Habib et al. (2015), Transient ischemic attack, intracerebral or subarachnoid haemorrhage, brain abscess, meningitis, infectious (mycotic) aneurysm and toxic encephalopathy are also seen, and firm evidence supports that additional clinically silent cerebral embolisms occur in 35-60% of IE patients" (p. 28).
- Splenic complications** - When a piece of the vegetation embolizes and lodges in the spleen and infarct occurs and is often asymptomatic. Although not as common, splenic abscesses also may occur and require surgical treatment.
- Cardiac Disturbances** - Myocarditis and pericarditis may occur from abscess formation from IE. This may lead to ventricular arrhythmias that often prove fatal. Pericardial effusions and aneurysms involving the pericardium may also occur. Conduction abnormalities such as heart blocks and atrial fibrillation may occur due to the spread of infection beyond the endocardium, through the valves and into conduction pathways. Myocardial ischemia or myocardial infarction may also occur if an embolus travels to the coronary arteries.
- Renal Failure** - Acute renal dysfunction (ARD) occurs in about 6-30% of IE patients and may worsen the prognosis of IE as ARD is independently associated with increased hospital mortality (Habib et. al., 2015, p. 30) This complication of IE may change the dosage of IV antibiotics to allow for renal considerations.

Implications for Nursing Care

- Assess patients for signs and symptoms of IE and report findings timely to the physician.
- Obtain a thorough health history, specifically regarding cardiac and IVDU history.
- If a patient has a confirmed IE (especially left sided), monitor the patient for complications such as embolization of the vegetation to other organs (decreased urine output, confusion, abdominal/back pain, shortness of breath, increasing chest pain).
- Nurses must also know the correct procedure for drawing blood cultures in order to avoid contamination of the specimen.
- The nurse must also be able to properly educate the patient on IVDU cessation and the instance of reoccurrence of IE in IVDU.

Conclusion

- Infective Endocarditis is a serious condition associated that if not treated, can lead to death.
- IE has an affect on many other vital body systems.
- IVDU puts the patient at risk for developing IE.
- Key pathophysiology points:
 - Damage to myocardial tissue makes tissue prone to bacterial attachment.
 - IVDU allows an easy route for bacteria to enter the bloodstream and attach to the valve (vegetation.)
 - Vegetation growth damages the valves to dysfunction and can lead to other organ damage (as earlier discussed.)
- Nurses must monitor patients with IE closely for complications.
- Educating patients with IE on IVDU cessation is important to prevent reoccurrence.

References



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