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Prosthetic Valve Endocarditis and Hypertension

Dejan Nesic*

Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Italy

Abstract

In contrast to Native Valve Endocarditis (NVE), prosthetic valve endocarditis (PVE) is a rare and serious complication of valve replacement that is associated with a high rate of morbidity and mortality. There are two main problems: figuring out what PVE is and how to treat it The diagnosis of PVE is challenging and frequently requires a variety of imaging methods in addition to standard microbiological tests. Although Computed Tomography (CT) and 18F-fluodeoxyglucose positron emission tomography/CT are frequently required, Transesophageal Echocardiography (TEE) remains the most common imaging tool for PVE diagnosis.

Keyword: Endocarditis • Morbidity • Treatment of hypertension • Diagnosis

Introduction

Stable fever, embolic complications, valve dehiscence, intracardial abscess, heart failure, and staphylococcal and fungal PVE all require surgical treatment to avoid fatal outcomes. Diagnostic and therapeutic approaches for PVE patients have been significantly complicated by transcatheter valve implantations and devices. The most effective treatment for PVE is still up for debate, despite advancements in our knowledge of the disease's pathogenesis and management. More research is required [2,3] to develop therapy methods for this potentially fatal outcome.

Literature Review

Prosthetic Valve Endocarditis (PVE) is a serious, potentially fatal side effect of replacing a valve. It occurs in 0.3–1.2 percent of patients annually and accounts for 10–30 percent of all cases of infectious endocarditis (IE). Those with prosthetic heart valves are more likely to develop IE. It has been discovered that the incidence and survival rates of PVE on biological and mechanical prosthetic valves differ. Thanks to advances in diagnosis and treatment, the mortality rate of PVE has decreased significantly over time, from 56 to 60 percent in the 1970s to 22.8% at the turn of the century. However, mortality continues to be high, and one possible explanation for this may be that complications are more prevalent due to distinctive pathophysiology, particularly in early PVE. The infection can cause damage to bioprosthetic as well as mechanical valves, and at five years, the frequency is the same (5.7 percent). Mechanical valves appear to be more susceptible to infection in the first three months following surgery. The cutoff point for defining early and late PVE is generally agreed to be 12 months following surgical intervention, despite the significant differences in causative microorganisms between PVE that occurs within a year of surgery and PVE that occurs later. Early PVE, which lasts for a year, is usually found in the first two months after a valve replacement. It can be brought on by hematogenic spread in the first days or months or by microorganisms invading the prosthesis during the procedure [1].

***Address for Correspondence:** Dejan Nesic, Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Italy, Email: Dejan.n19@usi.it*

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Perivalvular tissue is disrupted when microorganisms enter the prosthetic ring, increasing the likelihood of valvular dehiscence, pseudoaneurysm, or abscess formation. The thrombotic risk is increased by the absence of mechanical prosthesis endothelization in the early postoperative phase. Candida-related PVE is a rare but fatal condition, with documented case study fatality rates ranging from 37 to 62.5%. Nosocomial or healthcare-associated infections account for the majority of the causes of this PVE [2].

PVE does not have any one particular clinical presenting feature. The majority of patients have a fever and lose appetite when they arrive. After surgery, these symptoms are frequently ignored because they are so prevalent. A new heart murmur, a left bundle brunch block, heart failure, or embolic events are all signs of PVE. These patients rarely present with Osler's nodes, Janeway's lesions, or Roth's patches. Coronary artery embolization may result in a myocardial infarction. Frequently, the most common cause of sudden death is a ruptured valve[3]. Positive blood cultures and echocardiographic evidence of vegetation, paravalvular abscess, fistula, or valve dehiscence (NVE) are used to diagnose PVE, as is the case with native valve endocarditis. On the other hand, previous antibiotic use often results in sterile blood cultures, especially in the early stages of PVE. When antibiotics have not been administered previously, blood cultures are positive in ninety percent of PVE patients. A DNA test using pulsed-field gel electrophoresis to rule out PVE may be necessary for a single blood culture with isolated coagulase-negative Staphylococcus and sterile samples. As a direct result of direct contamination of the operating environment, PVE may have developed a polyclonal infection [4].

Consistently positive blood cultures, septic embolism, heart failure, and death are among the side effects of PVE. In patients with prosthetic valve endocarditis, the most significant predictors of in-hospital mortality are persistent infection and heart failure. While PVE issues are comparable to those experienced by NVE patients, they are more challenging to treat. Because the length of the bacteremia is determined by the bacterium, it is difficult to determine the precise definition of persistent bacteremia or relapse. Bacteremia-positive blood cultures in methicillin-resistant S. aureus (MRSA) infections can be observed even after seven days of treatment, whereas S. viridans infections can become sterile after 48 hours. When PVE is used, septic embolism increases mortality and morbidity. Similar to NVE, it may present as a brain, splenic, or renal abscess. In a sample of 111 PVE patients, the risk of stroke was found to be 23%. In addition, a hemorrhagic transformation occurred in 42% of PVE patients, most likely as a result of anticoagulant medication, which is frequently utilized in these settings [5].

Discussion

There is no one specific clinical presenting feature for PVE. When they arrive, the majority of patients have a fever and lose appetite. Because they are so common after surgery, these symptoms are often ignored. PVE is

characterized by new heart murmurs, left bundle brunch blocks, heart failure, and embolic events. Rarely do these patients present with Roth's patches, Janeway's lesions, or Osler's nodes. Myocardial infarction can occur after coronary artery embolization. A ruptured valve is frequently the leading cause of sudden death. As with native valve endocarditis, positive blood cultures and echocardiographic evidence of vegetation, paravalvular abscess, fistula, or valve dehiscence (NVE) are used to diagnose PVE. On the other hand, particularly in the early stages of PVE, previous antibiotic use frequently results in sterile blood cultures. Ninety percent of PVE patients have positive blood cultures when antibiotics have not been given before. For a single blood culture with isolated coagulase-negative Staphylococcus and sterile samples, it may be necessary to perform a DNA test using pulsed-field gel electrophoresis to rule out PVE. PVE may have developed a polyclonal infection because of direct contamination of the operating environment.

PVE's side effects include consistently positive blood cultures, septic embolism, heart failure, and death. Persistent infection and heart failure are the most significant predictors of in-hospital mortality in prosthetic valve endocarditis patients. PVE issues are similar to those of NVE patients, but they are more difficult to treat. It is difficult to precisely define persistent bacteremia or relapse because the length of the bacteremia is determined by the bacterium. Methicillin-resistant S. aureus (MRSA) infections can still have bacteremia-positive blood cultures seven days after treatment, whereas S. viridans infections can become sterile after 48 hours. Septic embolism increases mortality and morbidity when PVE is used. It may present as a brain, splenic, or renal abscess, as with NVE. The risk of stroke was 23% in a sample of 111 PVE patients. In addition, 42% of PVE patients experienced a hemorrhagic transformation, most likely as a result of anticoagulant medication, which is frequently used in these settings.

Conclusion

A major potential complication of valve replacement surgery with a high death rate is PVE. It is difficult to establish a diagnosis. There are now a number of imaging techniques with a high sensitivity-to-specificity ratio. However, echocardiography continues to be the method of choice. PVE treatment is even more challenging because of its complexity and the high risk profile of these individuals due to numerous comorbidities. The etiology of Staphylococcus aureus, dense vegetation with a high risk of embolization, a paravalvular or myocardial abscess, a fistula, a valve dehiscence, or heart failure all necessitate immediate medical and surgical treatment. The type of heart valve used and the precise timing of the surgery are still up for debate. The treatment of PVE patients changed dramatically when transcatheter valve implantations and devices became available. More research is required to develop therapy strategies for this potentially fatal outcome.

Acknowledgement

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Conflict of Interest

None.

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