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ONE OF THE SERIOUS PATHOLOGICAL CONDITIONS OF CHILDREN'S CARDIOVASCULAR SYSTEM IS INFECTIOUS ENDOCARDITIS DISEASE

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Annotation Infectious endocarditis is the inflammation of the endocardium, the inner lining of the heart, as well as the valves that separate each of the four chambers within the heart. It is primarily a disease caused by bacteria and has a wide array of manifestations and sequelae. It is a rare condition that can lead to rapid and significant morbidity and mortality if not efficiently diagnosed and treated. This activity reviews the evaluation and treatment of infectious endocarditis and highlights the role of the interprofessional team in managing patients with this condition.

Keywords: heart disease, infection, endocardit, gram-positive, streptococci, staphylococci.

Introduction

Infective endocarditis in children is a rare entity that poses multiple challenges. A history of congenital heart disease is the most common risk factor, although in recent years, other emerging predisposing conditions have gained relevance, such as central venous catheters carriers or children with chronic debilitating conditions; cases in previously healthy children with no medical history are also seen. Diagnosis is complex, although it has improved with the use of multimodal imaging techniques. Antibiotic treatment should be started early, according to causative microorganism and risk factors.

The vast majority of infectious endocarditis cases stem from gram-positive streptococci, staphylococci, and enterococci infection. Together, these three groups account for 80% to 90% of all cases, with Staphylococcus aureus specifically responsible for around 30% of cases in the developed world. In addition to various streptococci species, other common colonizers of the oropharynx, such as the HACEK organisms (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, and Kingella) can less frequently be the culprit bacteria. Numerous other bacteria have been previously identified as well but comprise only about 6% of total cases. Finally, fungal endocarditis represents only about 1% of cases but can be a typically fatal complication of systemic Candida and Aspergillus infection in the immunocompromised population. Risk factors and the environmental setting of bacterial acquisition, healthcare versus community, provide hints towards the underlying infectious etiology. The definition of nosocomial infections remains controversial, but in general, healthcare-related cases emerge in the setting of early prosthetic valve endocarditis (typically defined as occurring within the first 60 days since surgery) or following recent vascular catheterization, hemodialysis, hospitalization, or extra-cardiac operative procedures. In these situations, S. aureus represents the predominant pathogen, responsible for around 50% of nosocomial infections. The less virulent coagulase-negative staphylococci, such as S. epidermidis, stereotypically stem from indwelling vascular devices or recently implanted prosthetic valves. Enterococcal infection emerges with similar frequency in both nosocomial and non-nosocomial infections, comprising about 15% and 18% of cases, respectively. Community-acquired infections tend to develop in the setting of immunosuppression, intravenous drug use, poor dentition, degenerative valve disease, and rheumatic heart disease. Intravenous drug use, which underlies almost 10% of infectious endocarditis cases, suggests repeated inoculation with skin flora such as S. aureus and S. epidermidis, with S. aureus demonstrating

a predilection for healthy, native tricuspid valves. While fairly uncommon in healthcare-related infections, viridans group streptococci underlie about 20% of community-acquired infections. Classically, infections with Streptococcus gallolyticus (bovis) organisms should raise suspicion for underlying colon carcinoma.

Early histopathologic studies in humans and decades-long investigations that have included an animal model of experimental endocarditis have confirmed 2 critical histopathologic findings: Damaged or denuded endothelium is necessary for initial pathogen colonization of a cardiac nidus; and Gram-positive cocci, the predominant pathogens in both native and prosthetic value infections, express multiple adhesins that serve as virulence factors through their ability to enhance host cell/substrate attachments that are important in both the initiation and propagation of endocardial infection. (Adhesins are discussed further in a separate section.) Denuded cardiac endothelium can occur when there is turbulence caused by abnormal cardiac structures, in particular stenotic or regurgitant valves, that results in high-velocity jets of blood. Once the endothelium is damaged, the host response includes platelet and fibrin deposition, leading to so-called nonbacterial thrombotic endocarditis (NBTE), at the wound site. NBTE serves as an excellent nidus for subsequent bacterial or fungal colonization in a patient with bacteremia or fungemia. The prevailing notion is that activities of daily living, such as chewing food, toothbrushing, and flossing, account for most bloodstream seeding of an NBTE site. There are additional mechanisms involved in endocarditis pathogenesis. Right-sided endocarditis can occur when there are intravenous catheters, illicit intravenous drug use, or cardiovascular implantable electronic device leads that dwell in the right side of the heart. Damage to the endothelium occurs by 2 mechanisms. One involves direct damage produced by the foreign body “rubbing” directly against the endothelial surface. The other is via an indirect effect, such as when a foreign device interferes with normal tricuspid valve function and causes regurgitant jets of blood. Bacteremia may be caused by entry of organisms at the skin site of percutaneous catheters or leads, via the catheter lumen, or in contaminated infusate. Microorganisms carried by the bloodstream enter the right side of the heart, potentially causing IE on preexisting NBTE. IE can also occur as a result of direct infection of an indwelling device. This occurs at the time of device placement into a cardiac locus (eg, valves, leads, other types of devices) and is an example of surgical site infection. These infections can occur despite the administration of antibiotic prophylaxis at the time of placement of cardiovascular devices such as heart valves, pacemaker leads, or left ventricular devices.

Adhesins Virulence factors that are involved in bacterial adherence, socalled adhesins, have received the bulk of recent investigative attention. Advances in molecular biological techniques have been crucial in characterizing these cell surface structures, with attention specifically to staphylococcal, streptococcal, and enterococcal species, which account for the large majority of IE cases. These adhesins attach to either host cell structures or extracellular molecules that bind to host cells or to extracellular matrix. The availability of an experimental animal model of endocarditis has been a pivotal aspect of these pathogenesis investigations. It has served as the ultimate evaluation of in vitro molecular techniques to obtain mutant and recombinant isolates that are developed to examine the effects of a single purported virulence factor expressed by a wild-type strain. Considering the fact that Gram-positive cocci typically express multiple adhesins, the ability to demonstrate the role of a single adhesin in infection pathogenesis is remarkable. For example, this approach demonstrated pilus involvement in attachment to collagen by Streptococcus gallolyticus. This was the first time that a virulence factor was demonstrated in an animal model of endocarditis.30 Interestingly, strains that expressed pil1 did not adhere to either fibronectin or fibrinogen but did form biofilm in vitro. A nonpathological Lactococcus lactis strain that by recombinant techniques expressed Pil1 in vitro was examined with its parent strain that did not express Pil1 in a rat model of experimental endocarditis. The results suggested that Pil1 was important in vivo as a virulence factor; 82% of rats challenged with the Pil1+ strain developed experimental endocarditis, in contrast to the animals that received the Pil1− strain (36%, P=0.03).30 The “big 3” pathogens (viridans group streptococci [VGS], S aureus, and Enterococcus species) that account for the large majority of endocarditis cases have been the primary focus of pathogenesis studies.31 Adhesins of S aureus, which have been referred to as MSCRAMMs (microbial surface components recognizing adhesive matrix molecules), are surface molecules involved in staphylococcal attachment to collagen, thrombospondin, laminin, fibrinogen, and fibronectin.32 These interactions with host proteins not only may be important in the initial adherence of bacteria to a site of endothelial damage but also may be operative in bacterial persistence and engulfment by the host cell (endothelial cells, platelets). Similarly, there have been several bacterial surface structures identified in strains of VGS and Enterococcus species that appear critical in endocarditis pathogenesis. Study of pathogenic mechanisms in IE is pivotal as we consider potential advances in infection treatment and prevention in the future.

This knowledge serves as a foundation for the development of novel clinical tools that include therapeutics and vaccines. Indeed, identification of a virulence factor resulted in development of a vaccine that reduced the risk of endocarditis development in an animal model.

Conclusion, infectious endocarditis is a serious pathological condition with heart tissue damage. A good understanding of the pathological condition is very important to prevent severe consequences of this disease and to treat it. Uncomplicated treatment makes it possible to further improve the quality of life of children.

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