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REVIEW

Percutaneous pulmonary valve endocarditis: Incidence, prevention and management

Endocardite sur bioprothèses pulmonaires percutanées : incidence, prévention et prise en charge

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Summary The epidemiology of infective endocarditis is changing rapidly due to the emergence of resistant microorganisms, the indiscriminate use of antibiotics, and an increase in the implantation of cardiovascular devices including percutaneous valves. Percutaneous pulmonary valve implantation has achieved standard of care for the management of certain patients with right ventricular outflow tract dysfunction. With its expanding use, several cases of early and delayed infective endocarditis with higher morbidity and mortality rates have been reported. This review summarizes the trends in percutaneous pulmonary valve infective endocarditis, postulates proposed mechanisms, and elaborates on the prevention and management of this unique and potentially fatal complication.

Abbreviations: CHD, congenital heart disease; IE, infective endocarditis; PPV, percutaneous pulmonary valve; PPVI, percutaneous pulmonary valve implantation; RVOT, right ventricular outflow tract.

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Résumé L'épidémiologie des endocardites infectieuses change rapidement en raison de l'émergence de micro-organismes résistants, à l'utilisation large des antibiotiques, et à l'augmentation des implants cardiovasculaires. L'implantation de valves percutanées fait désormais partie de la prise en charge conventionnelle des patients avec dysfonction de la voie d'éjection droite. Avec son expansion rapide, plusieurs cas d'endocardites infectieuses précoces et tardives ont été rapportés avec un risque accru de morbi-mortalité. Cette revue résume les connaissances actuelles sur le sujet, propose des mécanismes physiopathologiques et élabore des conduites à tenir pour prévenir et traiter cette complication unique et potentiellement fatale.

Introduction

Percutaneous pulmonary valve implantation (PPVI) (Melody® valve; Medtronic, Minneapolis, MN, USA) has become established as a valuable treatment option for patients with right ventricular outflow tract (RVOT) dysfunction since its clinical introduction in 2000 [1—4]. Excellent early, mid-term and even long-term success rates, with improvements in functional status, peak exercise capacity and ventricular function, have been reported since then [5—12]. Unfortunately, because of factors that are not clearly understood, several cases of early and delayed percutaneous pulmonary valve infective endocarditis (PPV IE) have been reported. IE is a burden in the congenital heart disease (CHD) population, particularly in patients with prosthetic valves in whom diagnosis is more difficult, prognosis is worse and the need for surgery is more frequent compared with CHD patients with native valves. Around half of the patients with CHD-associated IE develop severe episode-related complications and the mortality rate for surgery for IE is very high (40—50%) [13—15]. In this review article, we summarize the trends in PPV IE, postulate proposed mechanisms, and elaborate on the prevention and management of this unique and potentially fatal complication.

Clinical presentation and diagnosis

IE is a clinical diagnosis and requires a high index of suspicion, especially in the CHD population, as patients may present with non-specific symptoms. The Duke criteria for the diagnosis and management of IE were initially drafted in 1994; they were later modified in 2002 to include echocardiography criteria, and again in 2007 for optimal clinical use. The two major clinical criteria are abnormal blood cultures and evidence of endocardial involvement. The five minor clinical criteria are a predisposition to IE, a fever of > 38 °C, vascular phenomena, immunological phenomena or microbiological evidence of IE not meeting major criteria. Requirements for a clinical diagnosis of IE are: two major clinical criteria; one major and three minor clinical criteria; or five minor clinical criteria [16—18]. Although these criteria are still used universally for IE, the patient characteristics, timing and presentation of PPV IE are quite complex, and one may have to keep a high index of suspicion in such patients. It is important to note that the clinical presentation with atypical organisms, such as *Coxiella burnetii* endovascular infection, is usually insidious, lacks the typical features of bacterial endocarditis and often results in delayed diagnosis. Patients are often afebrile and vegetations are usually absent or small [19,20].

Definitive IE is diagnosed based on modified Duke criteria, although in patients with prosthetic or Melody valves, true valve involvement may be difficult to determine with a high degree of certainty, because of the acoustic shadowing artefacts from the prosthesis and the unusual anatomy of the RVOT. Transoesophageal echocardiography might be needed. However, the anterior position of the RVOT makes its visualization very difficult and the result is not always inconclusive. An increase in RVOT gradient from postprocedure echocardiogram to hospital admission is present in most patients with IE. The Duke criteria should be modified for this particular substrate (i.e. PPVI) because, in our opinion, any degree of increase of RVOT gradient (unexplained by a structural complication, such as stent fracture) demonstrates the valvular involvement and should be considered as a major criterion similar to new onset of pulmonary regurgitation, unless proved otherwise. Based on these features, blood stream infection with a rise in RVOT gradient may be reclassified as definitive endocarditis. This criterion alone may very well explain the difference noted in the incidence of PPV IE between reported studies. Some other diagnostic tools may be helpful, such as intracardiac echocardiography, three-dimensional echocardiography or positron emission tomography. Positron emission tomography-computed tomography fusion imaging is a good diagnostic tool in case of difficulties in assessing hot spots and slow mouldering cardiac involvement.

Incidence

Several types of presentations and risk factors have been reported. The first series in 2008 by Lurz et al. [21] reported five cases of PPV IE out of 155 patients with an age range of 7—71 years. The possible risk factors reported included dental treatment ($n = 1$), septic wound after arm trauma ($n = 1$), reactivation of previously treated fungal infection