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Recurrent infective endocarditis causing heart valve failure: A case report



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A R T I C L E I N F O

ABSTRACT

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Keywords: Infective endocarditis Bioprosthetic heart valves Heart valve Bioprostheses Pannus Vegetation EPIC® valve Infective endocarditis (IE) is an infection that does not usually respond rapidly to treatment, often because its early symptoms are non-specific. The diseased valves (native or bioprosthetic) may be calcified and the thrombotic vegetations on them typically friable and embolize easily. Left untreated IE leads to damage to the infected valve and to congestive heart failure (CHF). Its treatment usually requires heart valve replacement. Our 69-year-old patient had IE, and underwent aortic valve replacement (AVR) with a bioprosthesis. This case stresses the complications of IE and its tendency to recur in patients with bioprosthetic heart valves (BHV) who previously had IE.

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1. Introduction

Infective endocarditis (IE) is a potentially fatal condition defined by infection and inflammation of heart valves, usually functionally abnormal due to underlying disease [1]. Untreated, the patients progressively worsen and develop congestive heart failure (CHF). IE is more common in the elderly, 25%–50% occurring in those over 60 years old [2] with a higher incidence when the valve replacement was for IE [3]. IE has an incidence of 6–7 cases per 100,000 in developed countries, likely higher (6–10 cases per 100,000 life years) in developing countries [4]. In patients with IE, there is no significant difference in mortality rate whether a mechanical heart valve or a bioprosthetic heart valve is implanted, however patients are usually recommended to receive a mechanical heart valve if they are younger in age and a bioprosthetic heart valve if they are greater than 60 years of age.

The infection is caused primarily by bacteria, and in some cases fungi. The infection may be acute or subacute depending on the infecting microorganism and often develops slowly, initially with vague and non-specific symptoms such as low grade fever, aches, pains and fatigue, making diagnosis difficult [5,6]. Acute onset is usually with more virulent bacteria, significant symptoms and rapid destruction of the infected valve(s) tissues [5]. IE can also cause myocardial, paravalvular, or annular abscesses, new intracardiac shunts, new cardiac murmurs, embolic infarcts [7] and other life threatening complications [8], potentially with a mortality of 30% to 50% [9]. The bacteria's "adaptive intelligence" allows it to consistently resist the most efficacious and recently engineered drugs. Prevention of IE needs an emphasis on the diagnosis of valve disease, dental hygiene and avoidance of street drugs as well as a high incidence of suspicion of IE [6].

Native valve endocarditis (NVE) is predominantly caused by Streptococci and Enterococci [10], although the trend is changing and Staphylococcus seems to be increasingly common. Prosthetic valve endocarditis (PVE) is caused more often by Staphylococci, bacteria of the HACEK group (*Haemophilus, Actinobacillus, Cardiobacterium, Eikinella* and *Kingella*) and fungi [10]. A diagnosis is made on positive blood cultures as well as echocardiographic evidence of valvular/prosthesis infection, including but not limited to vegetations, paravalvular regurgitation, thromboembolic events and abscesses [10].

PVE is most common in the first two years after bioprosthetic heart valve (BHV) implantation with an incidence of 1-6% [7]. This has been decreasing, especially with the judicious use of perioperative antibiotics. In consideration of the bacteria's strong resistance to antimicrobial and antithrombotic treatments, in highly infectious cases, particularly in those with PVE, early surgery is recommended to improve survival. A major remaining concern is the prevention of high recurrence of IE.

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We report a patient who suffered from NVE and PVE on a porcine bioprosthesis, and who had to undergo two heart valve replacements with one of the newer porcine BHV.

2. Case presentation

A 69-year-old woman presented with recurrent IE. She had suffered complications associated with the IE and had undergone multiple surgical procedures over 4 years. The patient had a history of type II diabetes mellitus, chronic kidney disease, obesity, hypertension, asthma and dyslipidemia. She had received a permanent pacemaker VVI Medtronic in 2007 for sick sinus syndrome and coronary artery catheterization in 2007 and in 2012. She was diagnosed with IE and following medical management underwent aortic valve replacement (AVR) with a porcine bioprosthesis (EPIC® by St Jude Medical Inc., Minneapolis, USA).

The patient was in good condition for about fifteen months following surgery when she developed symptoms of fever and dizziness, which led to a diagnosis of PVE with blood cultures positive for Enterococcus. She was immediately started on antimicrobial therapy. Aortic PVE was confirmed by a transesophageal echocardiogram (TEE), which showed prominent thickening of the leaflets as well as signs of an infected pacemaker lead, moderate tricuspid regurgitation, an aortic annular abscess and tricuspid vegetation. Following the diagnosis, the patient underwent a second AVR with a pericardial bioprosthesis (21 mm Mitroflow pericardial bioprosthesis, Sorin Inc., Milan, Italy). There was confirmed hypokinesia of the right ventricle, dilatation of the right atrium and a large abscess impeding the left ventricular outflow tract. The patient's aortomitral junction was repaired with a pericardial patch and the infected pacemaker lead removed. The postoperative course was complicated, but she is back at home.

3. Pathology

The explanted specimen consisted of a porcine bioprosthesis (EPIC®, SJM, Minneapolis, USA) with flow surface dimensions of 2.7 cm by 2.5 cm; the difference in dimension was due to a loss of sewing cuff. The porcine valve showed evidence of pannus and thickening of the cusps due to significant vegetation.

On the flow surface, the cusps were thickened, had a greyish-white to pale brown discoloration and a roughened appearance (Fig. 1). The cusps did not open or close completely, on mild digital pressure. In at least one commissural region, thrombotic material was present. Pannus was visible on the sewing ring along with 7 small, white pledgetted sutures (length 0.3 cm).

On the non-flow (or aortic) surface, pannus was found in the bias areas and on all three stent posts, but significantly greater on one



Fig. 1. Explanted bioprosthetic porcine valve (EPIC®, SJM, Minneapolis, USA) at 18 months. The flow surface shows thrombus (arrow) on the valve's leaflets.



Fig. 2. Explanted bioprosthetic porcine valve (EPIC®, SJM, Minneapolis, USA) at 18 months. The non-flow surface has pannus (P) on the sewing cuff extending onto the leaflets as well as large vegetations (arrow) on the cusps and in the sinuses.

(Fig. 2). All three commissural regions showed blue pledgetted sutures with some appearing to have been taken through the stent post fabric (Fig. 3). All three cusps showed soft grey-brown vegetations on the sinus surfaces (Fig. 4) as well as focal calcification (Fig. 5). The cusps were thickened and stiffer than normal. One commissural region was narrowed by the presence of vegetations (thickness of 0.3 cm). Histologically, microorganisms were seen in the thrombotic vegetations on the infected cusps (Fig. 6), as were small foci of calcification.

4. Discussion

This case highlights the recurrence of IE in the BHV of older patients who previously had NVE [11]. The recurrent infection led to the patient's symptoms and to the marked destruction of annular cardiac tissues. In addition, the vegetations and the infection with superimposed pannus lead to bioprosthesis dysfunction and to replacement of the porcine bioprosthesis. Untreated, this infected heart valve would have led to CHF and death.

Infection of a BHV is a life threatening condition. It may occur early or late post valve replacement. Its early incidence has been decreasing, likely due to improved surgical techniques and the increasing use of preoperative antibiotic prophylaxis. However, the incidence of late



Fig. 3. Transverse sections through the commissural region of the bioprosthesis shows the junction of two cusps (black arrows), suture sites (black broken arrows) and the commissural end of the cusps (C). Pericardium (P) covers the porcine aortic tissue (A). (Original magnification $\times 2.5$; Movat pentachrome stain)

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