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Prosthetic Valve Endocarditis, A Case Report: Challenges and Advances

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ABSTRACT

Infective endocarditis on valve prostheses accounts for 10-30% of all cases of endocarditis, with an in-hospital mortality of 20-40%. It is present in approximately 1-6%4 of patients with valve prostheses, with an incidence of 0.3-1.2% of patients per year. We present the case of a patient who underwent his third sternotomy secondary to acute heart failure due to dysfunction of the mechanical prosthesis, rupture of the annulus and severe paravalvular leak.

KEYWORDS: infective endocarditis, mechanical heart valve prosthesis, prosthetic valve endocarditis, *staphylococcus.*

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INTRODUCTION

Infective endocarditis (IE) is a multifactorial disease secondary to microbial colonization of the valvular endothelium that can lead to destruction of the heart valves, involvement of adjacent myocardium, development of embolisms from vegetations and severe persistent sepsis[1].

Infective prosthetic valve endocarditis (PIVE) is defined as when the infection settles on any mechanical, biological, autologous or heterologous substitute for native valves. It accounts for 10-30% of all cases of endocarditis[2] and is one of the most severe forms of this disease, with an in-hospital mortality of 20-40%[3] .

It is estimated to occur in about 1- 6% of patients with valve prostheses, with an incidence of 1-3% in the first year and 3-5% at 5 years; it is most frequent during the first three months after surgery and then gradually decreases from 0.3% to 0.6% at 12 months[1,3-4] .

Factors that have been associated with an increased risk of endocarditis are: multiple valve replacements, valve replacement due to active endocarditis, nosocomial bacteremia in the perioperative period, prolonged cardiopulmonary bypass, and male sex[1,5] .

IVPD occurs in two forms: Early prosthetic IE that occurs in the first year after the surgical procedure, and late prosthetic IE that appears after the first year of surgery[6,7] .

The early form is acquired in the perioperative period, in the operating room during surgery or by infection of the surgical wound or through intravascular catheters in the post-surgical care unit, in which coagulase-negative staphylococci such as Staphylococcus epidermidis, Staphylococcus aureus (S. aureus), enterococci, fungi and gram-negative bacilli predominate[7]. In late prosthetic IE the microorganisms involved are similar to those found in native valve IE, mainly staphylococci, viridans group streptococci and enterococococci[4,8] .

The evolution of the disease can have an acute, subacute or chronic clinical course. In the acute form, symptoms appear from a few days to 2 weeks prior to diagnosis, usually with high fever, shivering and heart failure. The most frequent microorganism in this form of presentation is S. aureus. In the subacute form, constitutional symptoms (weight loss, asthenia, anorexia) and febrile fever predominate. In this case, the causative microorganisms are usually coagulase-negative streptococci or staphylococci, or Propionibacterium acnes[9,10] .

Diagnosis is established according to the modified Duke criteria[4,11], which are based primarily on clinical, echocardiographic and microbiological findings. Major criteria include culture isolation of typical microorganisms and the 7 echocardiographic signs[4] . Minor criteria include the presence of predisposing valve disease (including valve prostheses), parenteral drug use, fever, and the existence of vascular and immunological phenomena[7] **.**

Antibiotics for empirical treatment should be chosen according to the most likely causative microorganism, patient characteristics and clinical presentation of the infection. In

early PIDV, mainly methylcycline-resistant coagulase-negative staphylococci and S. aureus should be covered, after the first year Staphylococcus epidermidis is particularly frequent[4,8] .

In PIVP, more than 50% of cases will require surgery for definitive eradication of the infection[4,12] , and the vast majority of patients with early prosthetic IE with severe prosthetic dysfunction or heart failure will require surgery[13,14] , however, patients with uncomplicated late PIVP, not caused by staphylococcal or fungal infection, may be amenable to conservative treatment[3] .

CLINICAL CASE PRESENTATION

A 66-year-old male with sick sinus syndrome managed with metoprolol and amiodarone; with a history of moderate tricuspid insufficiency with placement of a definitive epicardial pacemaker approached by sternotomy in 2020, severe tricuspid insufficiency and severe mitral stenosis managed with 25 mm mitral valve replacement and tricuspid plasty with 34 mm mechanical prosthesis on April 12, 2023. He went to the emergency department for fatigue, progressive dyspnea until presenting paroxysmal nocturnal dyspnea, tachypnea, chest discomfort of intensity 8/10, going to UMAE Puebla, where he was found with distant audible rales, jugular ingurgitation and tachypnea, dyspnea at rest, tachypnea, use of respiratory accessory muscles, He required supplemental oxygen at 15 liters per minute, saturating at 86%, an X-ray was performed (Figure 1), and early orotracheal intubation was decided, with hemodynamic instability, with a tendency to hypotension secondary to cardiogenic shock, for which inotropic support was started with dobutamine.

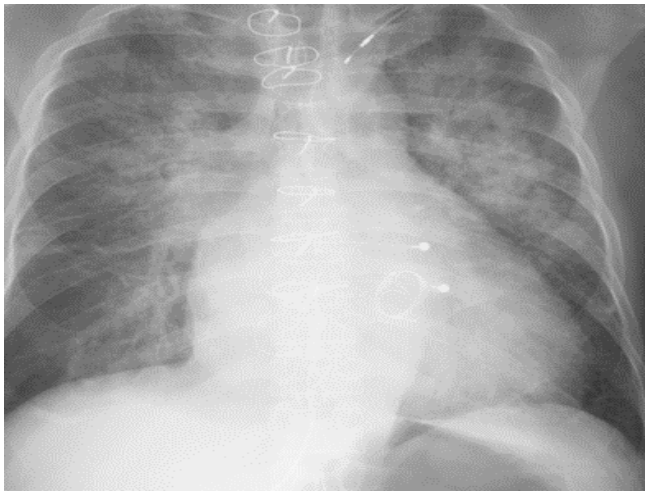

Figure 1 Pattern in butterfly wings and Kerley B lines

A TTTTUS was performed, reporting a mechanical prosthesis in dysfunctional mitral position due to partial detachment of 25% of its circumference that generates prosthetic Rocking, torrential jet with dehiscence located between 6 and 9 o'clock in surgeon's view, maximum length of dehiscence of 13.4 mm, normal valvular area, the three pillars of the prosthetic ring and valvar opening and closing (trivalva) are appreciated. Moderate tricuspid regurgitation, left ventricle with normal diameters, LVEF 72%.

The patient underwent emergency sternotomy due to mechanical prosthesis dysfunction, rupture of the annulus and severe paravalvular leak.

Sternotomy was performed, inactivating definitive pacemaker, mitral prosthetic valve was found with perforation of the mitral annulus at the level of the anterior leaflet of approximately 25% of the total circumference, the rest of the valve with an intact annulus without leaks, very friable and with scarce fibrin creations, a sample of the mitral valve was taken. The valve was repaired with 4 ethibond stitches, obliterating the leak site (Figure 2), endocarditis pacemaker was placed to stimulate cardiac contraction and mediastinal probes.

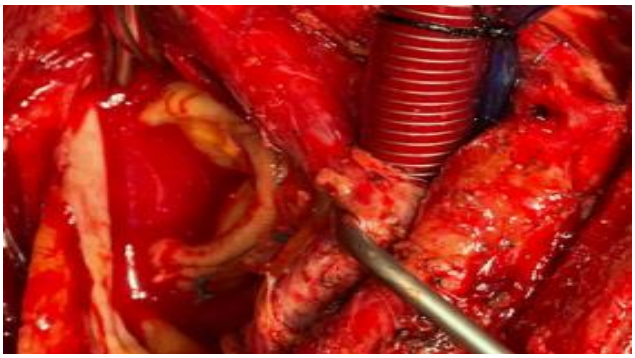

Figure 2 Obliterated paravalvular leak site

In the intensive care unit (ICU) empirical management with piperacicline/tazobactam was started, extubation was decided on the second day of stay in ICU with successful tolerance, vasopressor amines were withdrawn, and due to low expenditure of mediastinal probes were withdrawn. On the third day, a permanent pacemaker was activated and the patient was admitted to the floor for further management.

A culture was taken and Staphylococcus epidermidis was obtained, indicating that the dysfunction and detachment of the prosthetic valve was secondary to prosthetic endocarditis; an evaluation was requested by infectiology, who indicated a triple scheme with vancomycin, rifampicin and gentamicin for 6 weeks.

The patient is progressing satisfactorily, hemodynamically stable and without systemic inflammatory response syndrome.

DISCUSSION

Patients with PIVS have a history of prosthetic valve replacement in which an inflammatory process may develop near the prosthetic stitches, creating conditions for thrombus formation and its infestation, with the development of vegetation and perivalvular abscesses[15] .

PIVSD is a more severe form of endocarditis whose mortality remains high despite advances in diagnosis and changes in the therapeutic approach. Therefore, in order to reach an early diagnosis, IE should always be considered in patients with

prosthetic valve disease with fever, prosthetic dysfunction (new valvular or perivalvular regurgitation) and embolisms[7] . Currently the patients are older and predominantly male, although the reason for this is not clear, the theory that it could be a consequence of shaving the chest hair in the preoperative period and subsequent folliculitis is advocated. Another possible theory would be urethral catheterization, since in the male the urethra is longer and curved, and the procedure is more traumatic, which could cause more bacteremia[16] .

In PIVSD the extension of the infection to the annulus and adjacent myocardium results in paravalvular abscess formation and partial valve dehiscence with paravalvular leakage. If a large vegetation forms, it can enter the valvular orifice and cause functional obstruction, or it can prevent valve closure causing valve incompetence[17] .

The indications for surgery in patients with active PVI can be grouped into 3 types: a hemodynamic indication, an infectious indication, and another whose main objective is to prevent embolism. Of the 3 types of indication, hemodynamic indication due to heart failure is the most frequent. In most cases, the clinical manifestations are secondary to valvular destruction due to infection in biological prostheses and severe periprosthetic failure in mechanical prostheses[18] .

Antibiotic treatment of EIPV is more prolonged (at least 6 weeks) but is useful to avoid relapses. In staphylococcal PIVS, the use of vancomycin and gentamicin combined with rifampicin is recommended, due to its significant and rapid anti-staphylococcal potency and great capacity to penetrate the biofilms formed by these microorganisms. Rifampicin should be initiated 3-5 days after the start of effective antibiotic treatment once the bacteremia has been eliminated[4] .

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CONCLUSIONS

We present the case of a male patient who underwent a third sternotomy secondary to infective endocarditis of early prosthetic valve with acute clinical course, meeting three major Duke criteria for diagnosis. It was decided to close the valve dehiscence without removing the prosthetic valve because the perivalvular tissue was very friable and at high risk of rupture. She was managed with a combined regimen of vancomycin, rifampicin and gentamicin for 6 weeks, evolving satisfactorily.

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