Endocarditis in Bicuspid Aortic Valve with Extension and Perforation of the Anterior Leaflet Mitral

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Abstract

Young patient, hospitalized for infective endocarditis in bicuspid aortic valve with extension periannular infection (abscess with perforation of mitral leaflet) presenting significant aortic and mitral insufficiency. Evolved with mixed shock, low cardiac output effective, metabolic acidosis, respiratory failure and acute renal failure requiring dialysis secondary to glomerulonephritis by immune complex deposition. Established treatment for heart failure, broad-spectrum antibiotic therapy, noninvasive ventilatory support and hemodialysis, with clinical improvement. Underwent to aortic valve replacement and mitral valve bioprosthesis with good outcome. There was no report of previous dental procedure, other ports of entry for microorganisms and drug addiction. Spontaneous bacteremia can be certainly involved in these cases. There seems no reduction in the incidence of valvular infections with use of antibiotic prophylaxis in patients with native valves changed, according to current recommendations.

Keywords: **Heart Valve Diseases; Aortic Valve/abnormalities; Endocarditis/therapy.**

Case Report

Male patient, RSR, 23 years old, born and raised in Santo André, SP, single, student, sought medical service in his city, complaining of fever, palpitations and dyspnea. He was prescribed antibiotics (sic), with partial improvement of symptoms. After a few days, he returned to the hospital with worsening, showing chills, pallor, fever, and fatigue under minimal stress. On clinical examination heart murmur was heard, being hospitalized for further investigation. It was performed transthoracic echocardiography, which showed left ventricular dilatation, significant aortic regurgitation, and anomalous image attached to the aortic valve. The patient reported a history of blowing murmur diagnosed in childhood.

He denied recent dental procedure, tattooing, or drug addiction. Afterwards, he underwent transesophageal echocardiography, showing bicuspid aortic valve with severe aortic regurgitation, vegetation of 1.4 cm X 0.9 cm on the ventricular face of one of the valves, with extension to the base of the anterior mitral leaflet, showing an image suggestive of abscess and presence of perforation associated, visualized with color flow mapping.

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He was referred to Hospital Paulo Sacramento - Jundiaí-SP for surgical management for reasons of medical insurance. He progressed in the ICU to mixed shock (septic and cardiogenic), requiring vasoactive drugs and ventilatory support. Prescribed dobutamine, volemic expansion, blood cultures collected, and administered broad spectrum antibiotics (teicoplanin and meropenem). There was improvement of the shock, reducing acidosis and respiratory distress. After a few days, he progressed to oliguria, lower limb edema, and systolic hypertension requiring Nipride and clonidine, being diagnosed nephritic syndrome, requiring dialysis.

Physical examination: Patient with poor general state, pale +++ / 4++, sleepy, HR: 120 bpm, RR 28 irm, BP 113 x 48 mmHg, edema of lower limbs, abdomen with voluntary stiffness, painful on palpation, pulmonary auscultation with reduced adventitious sound in the bases, and cardiac auscultation with third heart sound and systodiastolic murmur at the base 3+/4+, and mitral systolic murmur 2+/ 4+. No skin lesions or signs of peripheral embolization.

Laboratory data

CBC: significant leukocytosis (24,500 leukocytes) with left shift (Rods 7% metamyelocytes 2%), anemia (Hb 8.5 g/dL) normocytic and normochromic, and platelets 166,000.

Na 141mEq/L, K 3.9 mEq/L, Calcium ion 1.04 mmol/l, Magnesium 1.7 mg/dL, CK 126U/l, CK-MB 46U/l, ESR 70 mm (normal lower than 15mm), Urea 115mg/dL, Creatinine 4.90 mg/dl, AST 26U/L, ALT 40U/L, Albumin

1.7 g/dl (3.5 to 5:2), Bilirubin 0.59 mg/dL, Fibrinogen 342 mg/dL.

Blood gases: pH 7.15, pO2 127 mmHg, PCO2 13.7 mmHg, 96% saturation (O2 mask 10 l/min).

INR 2.47, R 1.23, C-reactive protein 12 mg/dL (normal lower than 0.30).

Complement: C3 67 mg/dL (90-180), C4 12 mg/dL (10 to 40), Urine 1: Protein ++, Hb ++, WBC 43.000/mL (< 10,000), RBC 12,000/ml (< 8,000/mL).

Hemoculture: negative

Chest X-ray: Cardiomegaly ++, signs of pulmonary congestion and right pleural effusion. (Figure 1).

Abdominal ultrasonography: Signs of liver congestion. Kidneys of normal size showing ascites. ECG: left ventricular volume overload and presence of transient third-degree atrioventricular block (AVB) (Figure 2).

Echocardiography: aorta 30 mm, left atrium 43 mm, LVDD 58 mm, LVSD 38 mm, ejection fraction 61%, septum 9 mm, posterior wall 9 mm, bicuspid aortic valve calcified with mobile vegetation on the ventricular face measuring 1.5 x 0.7 cm, with significant aortic regurgitation, with equalization of diastolic mesodiastolic pressures (acute) and reverse flow in the abdominal aorta, abscess and fistula at the base of the anterior mitral leaflet with severe mitral regurgitation. Pulmonary artery systolic pressure of 47 mmHg measured by tricuspid reflux. Presence of mild pericardial effusion (Figures 3, 4, 5 and 6).

The patient, after improvement of leukocytosis and inflammatory markers, underwent surgery for aortic valve replacement by bioprosthesis, with anterior mitral leaflet valvuloplasty and satisfactory surgical outcome and progress. During surgery was confirmed the presence of abscess and perforation in the anterior mitral leaflet. The material was sent to pathology laboratory, which confirmed endocarditis in aortic and mitral valves, but unfortunately without isolation of the causative agent.

Discussion

Endocarditis are infections that affect the valvular endocardium and occasionally the wall, intact or injured, and, even today, have a high mortality rate[1]. In high-risk patients, such as patients with prosthetic valve, congenital heart disease, and previous endocarditis, the mortality is about 50%[2]. Even after discharge, there is a higher mortality of patients with IE, because of the recurrence of infection and the surgical procedure. In a study with 328 patients, survival rates related to the 1st, 3rd and 5th year were 92% (95% CI, 88% -95%), 86% (95% CI, 77% -92%), and 82% (95% CI, 59% -91%), respectively. Excess mortality was observed throughout the follow-up period, but was higher during the first year after hospital discharge[3].

Clinical manifestations are nonspecific and result from the presence of vegetations composed of platelets, fibrin, and microorganisms. These vegetations can determine perforation in valve leaflets, destruction of chordate tendineae, rupture of aortic sinuses, and thromboembolic phenomena. The presence of heart murmurs, especially diastolic, in the presence of fever is an indication for echocardiography to rule out endocarditis[4].

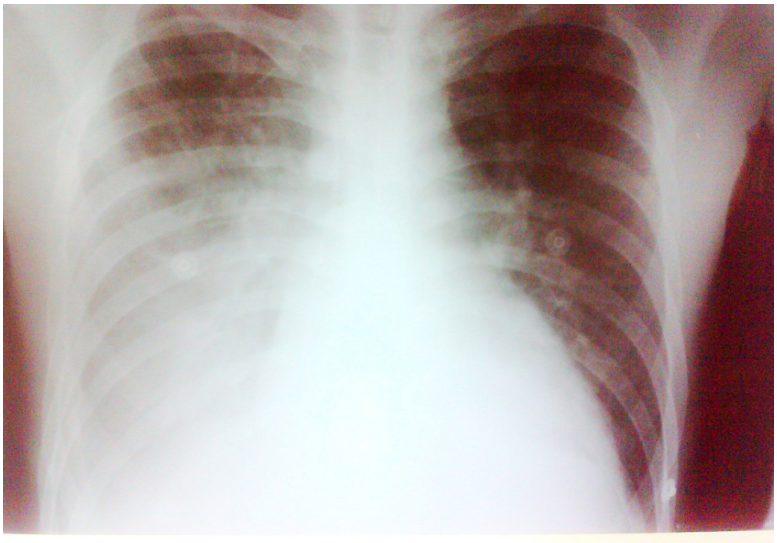

Figure 1 - *Chest w-ray showing extensive right pleural effusion.*

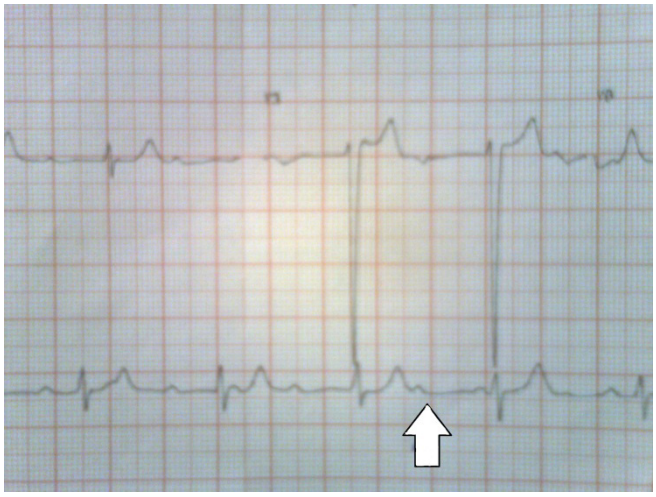

Figure 2 - *P-wave regularly dissociated from QRS complex, indicating AVB.*

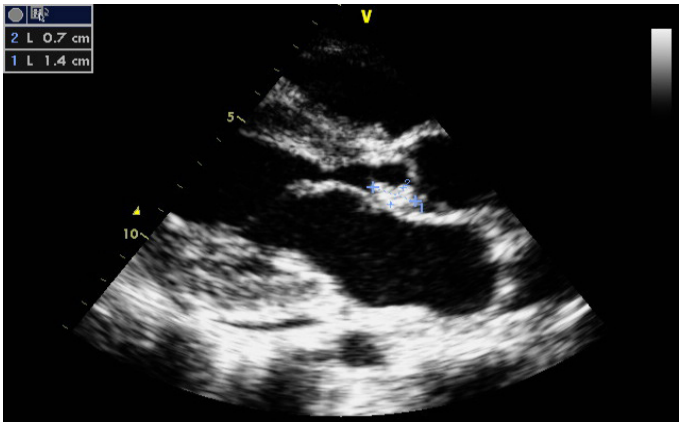

Figure 3 - *Anomalous echo in aortic valve, parasternal longitudinal axis.*

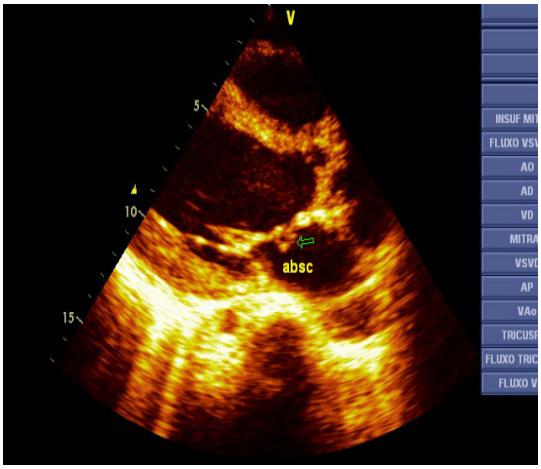

Figure 4 - *Image suggesting abscess in the anterior mitral leaflet.*

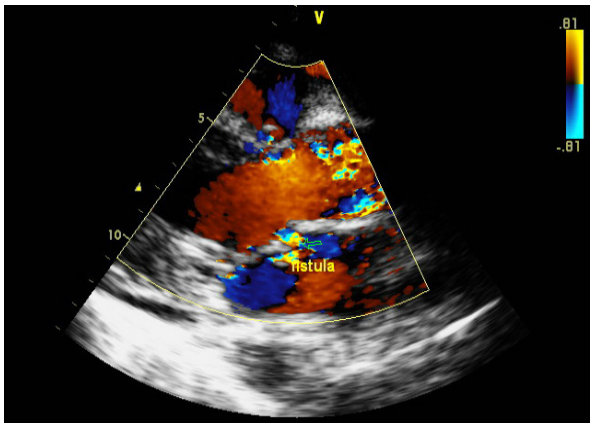

Figure 5 - *In the region of the abscess, there is perforation associated, evidenced by color Doppler.*

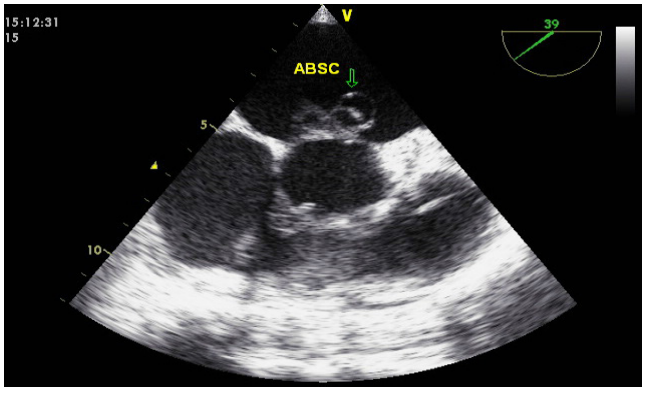

Figure 6 - *Transesophageal echocardiography, cross-sectional view, with visualization of lesion in the mitral valve, below the aortic valve.*

Risk factors for this infection are prior valvular lesions, cardiac prosthesis, intravascular catheters, dental procedures, and urologic, gynecologic, and gastrointestinal surgeries involving mucous membranes. The course of the disease depends on the etiologic agent. Generally, infections by Staphylococcus aureus, group B streptococci or gram-negative bacilli are usually more aggressive. On the other hand, alpha-hemolytic streptococci, enterococci, and fungi usually present a more indolent course. In developed countries, there is an increased incidence of infections caused by Staphylococcus aureus as a result of nosocomial infections and intravenous drug use, overcoming streptococci as the most common agent[5].

Patients may present with fever, chills, joint pain, muscle pain, night sweats, and anorexia. These symptoms reflect the release of inflammatory mediators common to infections, such as tumor necrosis factor alpha and interleukins. Clinical Hemorrhagic manifestations such as petechiae and gingival bleeding may be present, in addition to brain and systemic embolic events. The diagnosis based on symptoms can be frustrating, and implies complementary tests. It is common leukocytosis with left shift, normocytic and normochromic anemia (anemia of chronic disease), thrombocytosis, and elevated inflammatory markers such as ESR, alpha-1 acid glycoprotein, and C-reactive protein.

The request of blood cultures prior to antibiotic use is ideal and should be collected three sample pairs in the first 24 hours. The percentage of negative hemocultures can reach 20% as a result of prior antibiotic therapy[5]. There may be changes in urinary sediment with hematuria, proteinuria, and leukocituria, reflecting, in most cases, glomerulites secondary to deposition of circulating immune complex in the kidney.

The electrocardiogram should be requested routinely, and in case of abscess in the aortic ring, varying degrees of atrioventricular block may be observed. The echocardiogram is the most important imaging test and it can detect

vegetations in 60-80% of patients, reaching up to 96% in esophageal mode. The specificity of both transthoracic (TTE) and transesophageal (TEE) echocardiography is higher than 90%[6]. Examination also plays a role in prognosis (risk stratification and prediction of embolism), detects early perioperative complications, and provides support[7]. The use of antibiotics based on the causal agent and for periods of 4-6 weeks is essential, with hospitalization of patients for intravenous therapy.

Treatments for heart and renal failure with drugs and even hemodialysis may be considered in the presence of such complications. Patients indicated for valve surgery are those with refractory heart failure, signs of persistent infection, abscess formation, fungal infections, and mobile vegetations with diameters larger than 10 mm on echocardiography, multiple emboli, endocarditis in prosthesis installed earlier than 2 months, dehiscence of infected prosthesis, and relapse of infection in prosthetic valve. The most recent guidelines have suggested that surgical intervention to be performed as early as possible, especially because of the higher incidence of S. aureus infections, and in elderly patients[7].

In suspected cases of infective endocarditis, echocardiography is an essential test and should not be postponed[8]. Patients with previous history of endocarditis, new murmur, acute heart failure, having prosthetic valves, congenital heart disease, and unfavorable thoracic windows would be the candidates for initial TEE. Patients considered as low risk, in the presence of fever without other conditions of endocarditis, and absence of heart disease, would be the candidates for initial TTE[9]. In this case, the findings on TEE were observed initially in TTE. It must be considered the favorable acoustic window of the patient in question.

Although there is similar specificity in the detection of vegetation in both techniques, the sensitivity of the transesophageal method is higher compared to transthoracic method, especially in the presence of complications such as abscesses, aneurysms, and fistulas[4]. The patient presented showed clinical aspects suggestive of endocarditis complicated by perivalvular abscess, and important leukocytosis with left shift, and atrioventricular block on electrocardiogram.

Prophylaxis

The American Heart Association (AHA) publishes recommendations for prophylaxis of endocarditis, for over 50 years[10]. The effectiveness of such procedure has never been demonstrated in a randomized controlled trial. The document from the AHA 1997 stratifies conditions as high, medium, and low risk, with prophylaxis being not indicated in the latter group. This document states that most cases of endocarditis is not attributed to invasive procedures, occurring due to daily spontaneous bacteremia, such as toothbrushing and even chewing[11]. The AHA 2007 guideline caused intense discussion around the world, as it suggested a drastic change in the prophylaxis, which is now indicated only for some groups (Table 1) and no longer indicated before simple dental procedures and genitourinary, pulmonary, and gastrointestinal diagnostic procedures[12], meaning that for most patients prophylaxis is not indicated.

The rationale behind antibiotic prophylaxis is theoretical: bacteremia can cause IE in patients with valvular abnormalities; invasive procedures may lead to bacteremia, and, in animal models, antibiotics administered prior to induced bacteremia may reduce the risk of IE. However, it is unclear if results from animal models can be extrapolated to humans. A study of 275 patients in the Netherlands has shown that most cases of IE have been attributed to spontaneous bacteremia and not to invasive procedures[13].

What has been discussed is that the number of patients who have to receive prophylaxis is extremely high (NNT) to prevent an IE, and the use of antibiotics is not exempted of risks. For example, anaphylaxis to β-lactam can occur in 15-40 per 100,000 doses, being potentially fatal in 1-3 per 100,000 doses[14].

Table 1

Recommended Prophylaxis for Endocarditis ACC / AHA 2008 (no indication of class I)	
Valve Prosthesis	IIA / B
Prior IE	IIA / B
Congenital Heart Disease	IIA / B
Congenital Heart Disease repaired by prosthetic material (up to 6 months later)	IIA / B
Valve disease in transplanted heart	IIA / C
Endoscopy, TEE, colonoscopy	IIA / B

In conclusion, the indication of antibiotic prophylaxis before dental procedures would be suitable only in certain patient groups (Table I), being not indicated before invasive diagnostic procedures. The infectious endocarditis related to dental procedures is usually caused by streptococci viridans, bacteria found in the oral cavity. Intuitively, there would be an increase in infections by such agent[15]. Recent population-based study conducted in Minnesota, USA, retrospectively collecting cases of endocarditis by s.viridans, during the period from 1999 to 2010, the researchers found no increased incidence of endocarditis by this agent after the publication of the 2007 guideline. They warn, however, that continuous observations, and in other locations, are needed to substantiate this preliminary conclusion.

The interpretation of these recommendations should be made with caution, because we are in a country where there is still a high incidence of rheumatic fever, and the poor dental condition of the population needs to be considered. A frank conversation with patients, discussing the current guidelines and individualizing the cases, may be valid.

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Case Report