CAS CLINIQUE / CASE REPORT

SIMULTANEOUS TRICUSPID AND AORTIC VALVE ENDOCARDITIS

http://www.lebanesemedicaljournal.org/articles/66-2/case2.pdf

Tony BECHARA[1*], Randa TABBAH[2], Rachoin RACHOIN[1]

Bechara T, Tabbah R, Rachoin R. Simultaneous tricuspid and aortic valve endocarditis. J Med Liban 2018 ; 66 (2) : 112-114.

ABSTRACT • Simultaneous tricuspid and aortic valve endocarditis can occur, although rarely, in patients without any predisposing factors for endocarditis and without the presence of an aortic sinus rupture, most probably via the presence of bacteremia for a prolonged period, like the patient presented in this case report who had suffered from a recurrent fever for several weeks which had not been investigated nor treated till the person was admitted to the hospital with both pulmonary and systemic septic emboli.

Keywords : simultaneous; endocarditis; aortic valve; tricuspid valve

Bechara T, Tabbah R, Rachoin R. Endocardite simultanée des valves tricuspide et aortique. J Med Liban 2018 ; 66 (2) : 112-114.

RÉSUMÉ • L'endocardite simultanée des valves aortique et tricuspide peut se produire, bien que rarement, chez des patients sans aucun facteur prédisposant d'endocardite et sans la présence d'une rupture du sinus aortique, résultant très probablement d'une bactériémie prolongée, comme ce fut le cas chez la patiente présentée dans cet article qui a souffert d'une fièvre récurrente pendant plusieurs semaines et qui n'a pas été investiguée ni traitée jusqu'à l'hospitalisation de la patiente pour embolies septiques pulmonaire et systémique.

Mots-clés : endocardite simultanée; valve aortique; valve tricuspide

LEARNING OBJECTIVE

Our case report teaches us that a simultaneous bilateral endocarditis can occur on native normal valves without any predisposing factor, so the patient presents with systemic and pulmonary septic emboli associated with fever and vague flu-like symptoms associated with dyspnea.

INTRODUCTION

Bilateral, right and left, valvular endocarditis are truly rare, especially when there is no evidence of an aortic sinus rupture in a patient who is not an IV drug abuser neither known to have any past cardiac history of congenital or non-congenital valvular disease, as in the present case.

CASE PRESENTATION

A 72-year-old female patient, known to have diabetes mellitus and dyslipidemia since eight years, was admitted to the hospital because of chills and severe dyspnea. The onset of illness, 3 to 4 months before admission, started with intermittent chills, fever, dyspnea, headache, back pain, nausea, anorexia and chronic fatigue until the patient became bedridden. Note also that before admission, the patient had recently a non productive cough; she had not undergone any invasive treatment or diagnostic procedures recently.

[1] Cardiology & Echocardiography Departments, Notre Dame des Secours University Hospital (NDS), Faculty of Medicine, Holy Spirit University of Kaslik, Byblos, Lebanon.
[2] Cardiology Department Resident, NDS.
*Corresponding author : *Tony Bechara,* MD.
e-mail: dr.tony.bechara@hotmail.com

On physical examination, her temperature was 38.5 ºC, her heart rate 120 bpm and respiratory rate 40 breaths per minute. The patient's blood pressure was 150/80 mmHg. A systolic murmur, grade 2 over 6, was heard at the left upper sternal border with no pericardial friction rub. The lungs had crackles on the right base. There were no peripheral stigmata of infective endocarditis. The patient had no peripheral venous catheters or other infectious focus.

The laboratory findings included hemoglobin of 7.7 g/l, a white blood cell count of 18.000/m^3. The patient presented hypochromic anemia with thrombocytopenia (42,000 platelets/m^3) with normal urea and creatinine. C-reactive protein was 289 mg/l.

Arterial blood gas showed hypoxia and hypocapnea. Urine analysis showed 15 RBC and 15 WBC; uroculture, done unfortunately after the beginning of antibiotherapy, was sterile.

Blood cultures were positive for Gram positive *Enterococcus faecalis*.

Plain chest radiography reported a right inferior lobar condensation with bilateral pleural effusion.

Thoracic and abdominal CT scanning showed pulmonary embolism of the right proximal pulmonary artery and a splenic large infarction.

Venous Doppler ultrasound of lower limbs indicated the absence of deep venous thrombosis.

The patient was treated first with ceftriaxone 2 g IV per day, gentamicin 120 mg IV per day and teicoplanin 100 mg IV per day. No good response was noted with this combination. Teicoplanin was stopped and substituted with vancomycin 1 g IV per 8 hours; fluconazole 200 mg IV per day was added for a tongue fungal infection.

Transthoracic echocardiography showed bivalvular vegetations; the first vegetation 17 mm in length, mobile, protruding into the aorta during systole was detected on the ventricular side of the non coronary cusp of the aortic valve (Figure 1); the second vegetation was visualized as an hyperechogenic image of 13 mm behind the anterior leaflet of the tricuspid valve, associated with a severe thickening of all leaflets of the tricuspid valve (Figure 2). The left ventricle was hyperkinetic with an ejection fraction of 82% with moderate dilatation of the right chambers; a moderate pulmonary hypertension with an estimated systolic pulmonary artery pressure of 57 mmHg was associated with a moderate tricuspid regurgitation and stenosis, and a dilated inferior vena cava. A trivial posterior pericardial effusion of 2 mm was also seen.

Bivalvular replacement with double bioprothesis was indicated.

A transthoracic echocardiography was repeated after the surgery to evaluate the hemodynamic function of the heart and the bioprothesis function. The right heart chambers were no more dilated with a mean pulmonary artery pressure of 17 mmHg.

The patient presented an episode of dysarthria 24 h after the surgery; a CT scanner of the brain was done showing no signs of emboli.

DISCUSSION

Infective endocarditis occurs in 2 to 6 persons per 100,000 person-years; it is certainly higher in patients suffering of valvular heart diseases and those with intravenous drug abuse [1].

Endocarditis usually starts as a flulike illness with a dry cough, myalgia and asthenia that follow a subacute or chronic course. Low-grade fevers, night sweats and weight loss are cardinal manifestations. Unexpectedly, patients often, especially younger ones, do not consult their physician up to the time they develop a major complication, such as an embolic event or heart failure [2].

Enterococci are currently found in urinary, biliary and gastrointestinal tract infections and are increasingly being recognized in nosocomial bacteriuria and bacteremia. Actually, enterococci are the third most common cause of infective endocarditis (after streptococci and *Staphylococcus aureus*) causing 5 to 15% of cases, often occurring in older patients who usually are more at risk of having a degenerative valve disease; the presentation is typically subacute, infrequently associated with peripheral stigmata of endocarditis, and carries a relatively low short-term mortality rate; since enterococcal isolates are often resistant to antibiotics, therapy is based on synergistic drug combinations.

Isolated native non-rheumatic tricuspid valve endocarditis is rarely diagnosed, but can be encountered in the absence of intravenous drug use, intracardiac catheters of underlying cardiac anomalies. The presence of right-sided infective endocarditis might be a predicting factor of drug abuse, even if the injection sites are not obvious, so a toxicological analysis is reasonable [3].

Fever, multiple pulmonary emboli and sustained bacteremia are signs of clinical alert for right-sided endocarditis. Symptoms related to pulmonary emboli usually force patients to seek medical attention and dominate the clinical picture. Pulmonary events occur in 80% of these cases, generally involving the lower lobes. Peripheral stigmata of infective endocarditis are not consistently present. There is a paucity of cardiac symptoms and signs, as is common with most cases of right-sided endocarditis. Regurgitant systolic murmurs develop late in the course of the illness. Some authors suggest that clinical suspicion of tricuspid valve endocarditis should be raised in the presence of recurrent pulmonary events, ane-

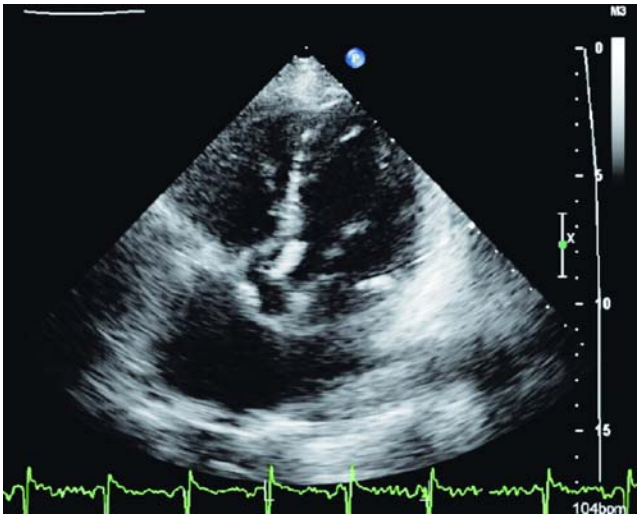

Figure 1
Echocardiography apical 5-chamber B-mode view showing the vegetation attached to the ventricular aspect of the aortic valve

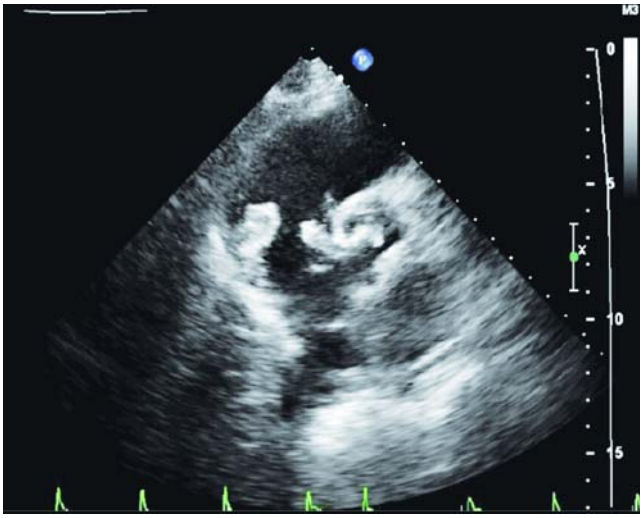

Figure 2
Echocardiography short axis B-mode view showing the severely thickened tricuspid leaflets

mia and microscopic hematuria, the so-called "tricuspid syndrome".

Our patient had vegetations on both aortic and tricuspid valves. This is, even in the case of multiple valve endocarditis, an unusual presentation, in the absence of a communication between left and right chamber. Involvement of both sides of the heart may give rise to different possible symptoms, according to the possibility of embolization to different territories, as was the case of our patient. In the reported case, there was important embolization, from the aortic (splenic infarction) as well as from the tricuspid (pulmonary embolism) vegetations. Septic pulmonary embolism is a serious and often underdiagnosed insidious disease. It usually presents with fever, respiratory symptoms and pulmonary infiltrates. Thoracic X-ray findings are nonspecific, but CT scanning can confirm the diagnosis.

The occurrence of multivalvular endocarditis is uncommon. The majority of cases involve a single valve, and demonstration of two-, triple-, or even quadruple-valve involvement by echocardiography is less frequent. The most common etiologic micro-organism is *S. aureus* (43%), which was responsible for multivalvular endocarditis. The mortality rate is comparable with single-valve infective endocarditis. Among the complications, only congestive heart failure was statistically more common in the multivalvular versus the univalvular group [4].

We conclude that bilateral native valve endocarditis are truly rare especially in a population not known to have predisposed factors and without a communication between the right and left heart chambers, most probably due to a prolonged untreated bacteremia from a complicated subacute or chronic valve endocarditis predisposing the other valves to be infected over time.

ACKNOWLEDGMENTS

Acknowledgments for the Department of Cardiology and Echocardiography in Notre Dame des Secours University Hospital, Byblos, Lebanon.

REFERENCES

1. Keys TF. Infective endocarditis. Cleveland Clinic, August 2010.
2. Heydari AA, Safari H, Sarvghad MR. Isolated tricuspid valve endocarditis. International Journal of Infectious Diseases 2009 May; 13 (3): e109-e111.
3. Lourenço S, Caeiro F, Ramos A, Pacheco H, Malhado JA. Aortic and tricuspid endocarditis due to *Streptococcus gallolyticus* in an immunocompetent patient with a normal heart. JC Cases 2010 Apr; 1 (2): e95-e97.
4. Kim N, Lazar JM, Cunha BA, Liao W, Minnaganti V. Multi-valvular endocarditis. Clinical Microbiology and Infection 2000 Apr; 6 (4): 207-12,