

Tricuspid valve endocarditis by histoplasma in immunocompetent patient

*Endocarditis de válvula tricúspide por
histoplasma en paciente inmunocompetente*

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Endocarditis,
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Palabras clave:

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ABSTRACT

Fungal endocarditis is a rare entity reported from 0 to 2% in many series but, although not the only one, *Candida* is the etiologic agent with the highest incidence. Although cases of fungal endocarditis due histoplasmosis have been described in immunocompromised patients, in the case that we are presenting, the pathogen was found in the tricuspid valve of an immune competent patient that received clinical and surgical treatment.

RESUMEN

La endocarditis fúngica es una entidad poco frecuente reportada entre el 0 a 2% en varias series y de ésta la *Candida* es el agente etiológico con mayor incidencia, pero no el único. Así se describe el caso de endocarditis fúngica debido a histoplasmosis que ha sido descrito en pacientes inmunocomprometidos, sin embargo, en este caso este patógeno fue encontrado en válvula tricúspide nativa de un paciente inmunocompetente que recibió tratamiento clínico y quirúrgico.

CLINICAL CASE

Twenty one years old female patient, with no relevant personal medical history. She did not refer any drug or intravenous catheter use, excessive alcohol consumption, risk behaviour for sexually transmitted diseases, recent oral cavity manipulation and had no history of rheumatic fever. She was transferred to our institution referred from a provincial hospital, with symptoms of fever, polyarthralgia, myalgia, generalized edema, hematuria and nodular lesions on the chest Rx which were classified as complicated pneumonia. According to the transfer sheet, quinolone treatment was started with no response. At our center, we performed an echocardiogram that reported a vegetation in the posterolateral leaflet of tricuspid valve. The patient developed shock and was admitted to the ICU for hemodynamic support and antibiotic treatment with vancomycin.

PHYSICAL EXAM

Lucid, oriented in time and space, without neurological deficit, Glasgow 15/15 pts. Hemodynamically tachycardic, hypotensive, normophonetic R1-R2, R3 with gallop rhythm, regurgitant tricuspid systolic murmur, lower and upper limbs edema, jugular venous distension ++, hepatomegaly, spontaneous breathing, dyspnea and tachypnea, and with bilateral rales crackles to the vertices.

COMPLEMENTARY TESTS

WBC: 11.00 K/uL; Hct: 36.9%; Hb: 12.9 g/dL; Plt: 94,600/mL; Bun: 53 mg/dL; creatinine 1.78 mg/dL; Na +: 145 mEq/L; K +: 3.53 mEq/L; pH: 7.45; pCO₂ 22 mmHg; paO₂: 79.9 mmHg; HCO₃- 15.3 mol/L; BE: -8; SatO₂: 96.9%. Proteinuria 24 hours 1,982.62 mg/24 h GGT: 270 U/L, total proteins 5.67 g/dL, HIV (-).

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Posteroanterior thorax Rx: cardiomegaly grade I, effacement of bilateral phrenic cost angle, radiopaque image at the right middle lung lobe.

ECG: microvolt, FC 115 lpm. 0° axis, changes in ventricular repolarization, suggesting right ventricular overload.

Blood and urine cultures taken at admission: negative.

Transthoracic echocardiography: image suggestive of a pedicled vegetation in posterolateral tricuspid valve leaflet, of 2.66 x 0.8 cm, discrete dilation of the left atrium, diffuse left ventricular hypokinesis with LVEF 55%.

Transesophageal echocardiogram: echo-genic mass of 3.8 x 1.7 cm, compatible with a vegetation located in atrial region of the lateral tricuspid valve; severe tricuspid regurgitation in color; discreet mitral insufficiency.

CLINICAL EVOLUTION

With these findings and the development of shock, it was decided to treat the endocarditis surgically, so we performed a bicuspidization of the tricuspid valve, an annuloplasty of De Vega, and a closure of atrial septal defect.

The histopathological results of the tricuspid valve microscopical examination describe fragments of fibrous connective tissue and fibrinoid materials in various stages of organization, with necrosis foci, important polymorphous inflammatory infiltrate, multiple gram positive bacterial colonies, as well as numerous spores whose diameters range from 2 to 5 microns, in relation to histoplasma.

Due to the appropriate response, the patient completed vancomycin treatment, and then continued with itraconazole for a year.

DISCUSSION

Native tricuspid valve endocarditis is a rare pathology in the non intravenous drug user adult. In most cases (70%) it is associated with underlying medical conditions (oral septic processes, alcoholism, skin or genital infections, abortions, immunodeficiency processes, indwelling catheters)¹⁻⁴ none of which was identified in this case.

Fungal infection is a rare etiology, and is associated with high morbidity and mortality. It

affects mainly the right valves,^{5,6} and *Candida* is the most prevalent causative agent, although not the only one. *Histoplasma capsulatum* can affect normal, sick and prosthetic valves, as well as cardiac tumors, or even ateromas.⁷

Aortic valve is the most frequently affected (58%), followed by mitral valve (31%) and tricuspid (8%)² valve. In the literature, only few series of endocarditis by histoplasmosis have been reported, and it has not been previously reported in our country.

Signs and symptoms include dyspnea, edema, heart compromise shown by a new murmur or changes in previous auscultation; pneumonia emboli, frequent involvement of large vessels including brain, limbs and gastrointestinal tract, as well as general symptoms such as persistent fever, weight loss and night sweats, that make presentation insidious and hinder the definitive diagnosis. Therefore, diagnostic suspicion and echocardiography play a key role.

The recommended therapeutic regimen is combined therapy of antifungal agents and valvular replacement.⁸ In the preoperative period, amphotericin B in doses 0, 7-1 mg/kg/day or amphotericin B liposomal 5 mg/kg/day should be started, continuing for at least 6 weeks in the postoperative period. The mortality rate is 50%.⁹ Relapses are common, even with combined, medical and surgical therapy, thus strict monitoring is essential even after an apparently successful complete treatment.⁹

In patients who cannot undergo surgery, initial treatment with amphotericin B should be continued, and then, oral itraconazole should be given for a prolonged time.

In this case, we considered the patient immunocompetent due to the lack of addiction history and the negative specific serology. Therefore, initially the patient received a combined therapy with vancomycin and surgery, until the histopathological results were obtained. Then, during the first weeks after the surgery, she received itraconazole antifungal treatment. This treatment continued for a year. Amphotericin B was not administered because she was an immunocompetent patient and in pre-surgery blood culture the causative agent was not identified. Furthermore, after the valve replacement, even with vancomycin, the patient's improvement was entirely satisfactory.

But we did start antifungal treatment after the histopathologic results. Currently, the patient is in functional class I with no recurrence.

CONCLUSIONS

Histoplasmosis endocarditis is a rare disease in immunocompetent patients and timely medical and surgical treatment allow greater survival.

Interest conflict

The author declares no conflict of interest.

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