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Fungal Endocarditis in Non Neutropenic Patient: A Case Report

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Abstract

Fungal disease of cardiac valve is rare and occurs mostly in patients with predisposing conditions. Clinical presentation is variable and could lack classic signs and symptoms of bacterial endocarditis, as well as the onset of disease vary from nonspecific signs to severe systemic embolic complications, leading to a changeling early diagnosis and higher mortality.

In this case report, a 50-year-old man presenting to emergency room for stupor and fever, rapidly worsen with respiratory failure, shock and admission to intensive care unit next day. Twelve days after, transthoracic echocardiography identifies a vegetation on native mitral valve, and blood cultures positive for yeast like fungi leads to Fungal Endocarditis diagnosis.

Abbreviations: TEE: Transoesophageal Echocardiography, SID: Stroke-Induced Immunosuppressive Syndrome

Introduction

Fungal endocarditis is an extremely debilitating disease associated with high morbidity and mortality **[1,2]**. Immunosuppression and intravenous drug abuse are the most common risk factors.

Candida and Aspergillus species are the etiologic fungi more commonly seen. They can be isolated from surgically removed emboli, resected valves, or infected foreign bodies. Candida albicans is responsible for 24-46% of all the cases of FE and for 3.4% of all the cases of prosthetic valve endocarditis, with a mortality rate of 46.6-50%. After Candida, the Aspergillus species are the second most frequent pathogens of fungal infection, accounting for approximately 25% of all FE cases in cardiac valve prostheses and the great vessels.

Early and accurate diagnosis of infective endocarditis is crucial because delayed treatment negatively affects outcome **[3]**. Clinical diagnosis of infective endocarditis is largely based on the modified Duke criteria and echocardiography.

However, both transthoracic echo cardiography and transoesophageal echocardiography (TEE) miss infective endocarditis sequelae in 30% of patients.

Clinical features of FE are similar to Bacterial Endocarditis or culture-negative endocarditis, but their reported incidence varies. Fever occurs in the majority of patients, often accompanied by cytokine-mediated phenomena such as chills, sweats and fatigue. Heart failure due to regurgitation rather than myocarditis or conduction defect occurs less often. The incidence of reported splenomegaly is much more variable- between < 10 and > 60%.

FE therefore has no consistent or pathogenomic characteristics. However, certain features or absence of features predict more for fungal aetiology **[4,5,6,7,8]**.

Histology and culture of surgically removable emboli often provides the means of diagnosis of FE. In Aspergillus FE these are often positive when blood cultures are negative. Deeper end organs such as the brain, mesentery and myocardium are also targeted. Neurological complications, either focal and secondary to cerebral embolization or more diffuse, occur in up to 30%.

1,3 β-D-Glucan detection has high specificity and makes it a fair diagnostic adjunct for invasive fungal disease in patients with appropriate pretest probability and a suggestive clinical syndrome. However, unlike the mannan and anti-mannan antibody test, exposure of patient to antifungals does not lower the levels of 1,3 β-D-Glucan.

Galactomannan is an important constituent of the cell wall of Aspergillus, however treatment with antifungals reduces the sensitivity of galactomannan antigen.

Several case reports support the use of PCR detection methods in fungal endocarditis especially in culture negative infective endocarditis (Candida albicans and Aspergillus spp).

Molecular diagnostics are complex and innovative tests which, at the expense of their high cost and limited availability, have a promising role in the early diagnosis of endocarditis **[9,10,11]**.

Case Report

A 50-year-old man comes to the emergency room for 3 days persistent fever, mild cognitive impairment, confusion, irritability and amnesia. In remote pathological history there are smoking and arterial hypertension. Initial assessments in the emergency room detect peripheral saturation 93% in ambient air, sinus tachycardia, blood pressure 100/60 mmHg, respiratory rate 23 acts/min, body temperature 38.4 C°, neutrophilic leukocytosis (22.3 x 10^3/mm^3), increased troponin levels (128 pg/ml), C-reactive protein 178 mg/L. Chest X-ray detects lung thickening in the right lower field.

Culture examinations are carried out, oxygen and empiric antibiotic therapy for suspected community acquired pneumonia are administered.

After 12 hours after hospitalization, the patient's clinical condition worsens. Respiratory failure appears with marked reduction in the levels of peripheral oximetry and partial pressure of oxygen in the blood, severe tachycardia and hypotension, temperature 39.2 C°. A cardiological evaluation describes a globally preserved contractile cardiac function, slightly dilated aortic root, mild tricuspidal and mitral insufficiency, TAPSE 33 mm, PAPS 35 mmHg. Non-invasive ventilation is applied, blood tests are repeated and show a worsening of leukocytosis, increased indices of renal function; hemodynamic parameters worsen and serum lactate values rapidly increase; the SOFA score increases by 5 points. The intensive care team is alerted and the patient is transferred to ICU, where invasive mechanical ventilation is applied.

The patient is then treated for septic shock associated with lobar pneumonia. Although the treatment provided, the clinical conditions do not improve, high-dose vasopressors is necessary, pulmonary gas exchanges remain poor, and in the following days the axial chest tomography detects a moderate ARDS.

Microbiological examinations show a S. Aureus infection on bronchial sample, and target antimicrobial therapy is provided.

After seven days of hospitalization in intensive care, no substantial clinical improvements are observed: culture examinations are repeated.

On the tenth day, bronchial cultures detect the presence of yeast-like fungi and A. Baumannii. Blood culture tests are repeated. Transthoracic echocardiographic examination detects a worsening of mitral insufficiency and a vegetation on the valve, indicating a transesophageal examination; it describes a gross vegetation on the atrial side of the posterior limb of the mitral valve, (about 2 x 0.8 cm), extremely mobile, inhomogeneous echogenicity, partial loss of coaptation and severe valve insufficiency; no evidence of flap perforation or perivalvary complications; absence of thrombi and spontaneous echo in the left atrium and auricle, no abnormalities on the remaining valvular systems examined, normal Doppler velocity in the left auricle, intact interatrial septum, absence of detectable shunts, normal left ventricle contractility, absence of plaques in the thoracic aorta.

1,3 β-D-Glucan and galactomannan tests are performed and are negative at this time.

A collegial evaluation of antibiotic therapy is required; it is decided for an eradicating therapy for A. Baumannii and empirical therapy with Daptomycin and Caspofungin; procalcitonin levels are monitored and prophylaxis of thromboembolism therapy is enhanced.

Axial tomography of the brain and an MRI are performed and detect several acute and subacute ischemic stroke signs on the right hemipartement of the splenium of the corpus callosum, the right occipital region, the ventro-medial thalamus and lower tonsillar subtentorial site.

After 5 days of treatment the clinical conditions improve; pulmonary thickening is reduced, weaning from mechanical ventilation begins, and neurological examination reveals that the state of consciousness is intact and there are no pathological neurological signs. Physiotherapy is provided.

Echocardiographic control shows severe mitral insufficiency and valve vegetation persist, but the global contractile function is performing.

Leukocytosis and CRP are still high, while PCT is low.

Cardiac surgery consultancy suggests mitral valve replacement surgery.

Two days later the patient is transferred to the cardiology department in stable clinical conditions; the antibiotic therapy continued until the eradication of A. Baumannii; cultures of blood and bronchial aspirate are negative, then antibiotic descalation is carried out, Daptomycin and Caspofungin continue.

After 5 days the patient is transferred to the cardiac surgery unit to plan the surgery.

The surgery is performed successfully, and the patient is discharged from the hospital 15 days later.

Finally histological and microbiological examination of the mitral valve confirmed Candida Albicans fungal endocarditis.

Discussion

Studies about endocarditis in non-neutropenic patients are limited to patient populations with known risk factors such as oncological or immunosuppressive diseases. The scientific literature is very limited, and the reasons include the difficulty in diagnosing this pathology, the high probability of symptomatic clinical pictures without fungemia or without proven systemic clinical signs of systemic fungal disease **[12,13,14]**. At the same time, it is difficult to decide when to undertake empirical therapy and which drug to use.

We draw attention to this case report in particular for the unusual presentation of endocarditis, but also for the subsequent therapeutic management.

The initial presentation of symptoms, with fever, amnesia, irritability, and confusion, is included in the clinical manifestations of an acute inflammatory state involving the central nervous system. Differential diagnosis includes neuroinflammatory diseases, thromboembolic disease, and rare manifestations of ischemic stroke.

In fact, the neuroradiological diagnostics carried out in the days following the onset of symptoms confirms the presence of multiple foci of ischemic damage, both acute and subacute, probably linked to septic thromboembolic events whose origin could be endocarditis; however, there were no signs of cerebral micro abscesses, nor did any relevant neurological sequelae occur at the end of the case.

Initially, biochemical and blood chemistry showed no signs of immunosuppression, on the contrary there were signs of an intense activation of the immune system. We do not believe that endocarditis was related to stroke-induced immunosuppressive syndrome (SID), although pneumonia and septic shock rapidly developed in the following hours.

In 2012, ESCMID published guidelines for the diagnosis and treatment of Candida disease in non-neutropenic adult patients; despite the invaluable help of these indications, much indecision remains about the management of these patients in the context of emergency departments and intensive care units. Even more so in this case, where the symptomatology was rather misleading from the beginning and the indices of fungal infection as well as the culture tests were negative, we consider it courageous to undertake in a patient at high risk an empirical therapy with an echinocandin and daptomycin **[15,16,17,18]**.

We share the collegial decision taken by the team of doctors who treated the patient, as the clinical conditions were very serious and the evidence of severe acute cardiac and respiratory failure required aggressive and timely treatment, providing for a positive clinical outcome.

Further studies are scheduled to update diagnostic and therapeutic strategies of this rare pathology.

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