An Aspergillus Myocardial Abscess Diagnosed by Echocardiography

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This is a rare case of Aspergillus myocardial abscess in 19-year-old woman with acute lymphoblastic leukemia treated by chemotherapy. During pancytopenia she developed invasive aspergillosis with myocardial abscess. The presence of specific antigen in the pericardial effusion was diagnostic. She died despite vigorous antifungal therapy.

Keywords: Aspergillosis, Myocardial abscess, Pericardial effusion, Myocarditis

We present here the case of a 19-year-old woman. She was treated 10 months for a Philadelphia-chromosome positive acute lymphoblastic leukemia. She had chemotherapy and allogeneic hematopoietic stem cell transplantation from an HLA-matched unrelated donor in August 2005. She reached complete remission after a posttransplantation relapse treated with a triple donor lymphocyte infusion.

A few weeks later she developed an acute graft-versus-host disease that required high-dose steroids. The patient remained in pancytopenia as a result of various complications. She experienced several bacterial and viral infections successfully treated by antibacterial and antiviral therapy.

Two weeks before the current admission, she experienced fever and chest pain; investigations revealed pulmonary aspergillosis with several nodules and pleural effusion, and the presence of specific antigen galactomannan in bronchoalveolar lavage fluid.

Antifungal treatment with caspofungin was started. The patient did not improve despite the shift into another antifungal drug, voriconazole; she developed pleural and pericardial effusion with hypotension.

Echocardiography revealed pericardial effusion, with echolucent masses inside the interventricular septum and the posterolateral wall of the left ventricle, there was no valvular vegetation (Figures 1-3 and Videos 1-3). Because of clinical instability, pleural and pericardial drains were inserted. Blood pressure improved. Immunoassay found specific antigen of Aspergillus fumigatus in pleural and pericardial fluids. Bacterial culture in the pericardial fluid, fungal culture, culture of Cytomegalovirus, and culture of Mycobacterium produced negative results. Polymerase chain reaction was tested for Mycobacterium, Toxoplasma gondii, and viruses (Cytomegalovirus, human herpesvirus 6, and Epstein-Barr); none of them were detected. Histologic analysis of pericardial tissue showed serofibrinous pericarditis. No malignant cell was seen.

Later, new nodules appeared in the lung, the skin, and the brain. Histologic analysis of a cutaneous nodule showed fungal forms characteristic of Aspergillus with large septate hyphae and angular dichotomous branching.

The patient finally developed a fatal cerebral hemorrhage. Necropsy was refused by the family. We concluded an invasive aspergillosis with pulmonary, cutaneous, cardiac, and probably cerebral involvement.

DISCUSSION

Despite necropsy not being allowed, the diagnosis is very probably a cardiac aspergillosis with pericardial involvement and myocardial abscess, without endocardial involvement. This kind of echolucent mass has been described in cardiac metastasis of leukemia. Others diagnosis could be evoked, such as bacterial infection such as Staphylococcus aureus and Escherichia coli—also viral infection—and other fungal infections such as Candida species.

In this case all those causes have been ruled out, bacterial and viral culture produced negative findings, and no parasites were found; no neoplastic cells were found.
Here we emphasize the role of immunologic detection of *Aspergillus* antigen in the diagnosis of cardiac aspergillosis because the culture has low sensitivity.

There are several species of *Aspergillus* (e.g., *fumigatus, niger, terreus*). *Aspergillus fumigatus* is the most frequent.5-7 The transmission to the patient is mostly by air especially in the presence of demolition or construction work.5

Cardiac aspergillosis is rare; it may develop after cardiac surgery (valvular surgery, cardiac transplantation, pacemakers, central catheter)5,8,9; in those cases sterility rules in operating departments seems very important.5

It may also develop in the setting of malignancies, immunodeficiency, broad-spectrum antibiotics, and intravenous drug use.5,6 The infective form of *Aspergillus* is the conidia; cellular defense against conidia is through the macrophages, neutrophils, and monocytes.6 Consequently, impaired cellular immunity favors aspergillosis in leukemia.

Cardiac aspergillosis may involve several sites: valvular lesions, endocardial nonvalvular lesions, myocardial infection, and pericardial involvement.

The valvular aspergillosis and the other endocardial aspergillosis result usually in large vegetations on the valves and the cardiac cavities; it is a source of embolic events even in large vessels. Those lesions are often visible by imaging techniques such as echocardiography.7,10,11 Pericardial effusion is frequent and may turn in tamponade.12 Myocardial abscess and mural involvement account for up to 40% of cardiac aspergillosis.13 They are source of embolic events with undetected vegetations.

Most of them are echocardiographically silent and revealed only after cardiotomy or necropsy.13-15 When clinically suggested, blood culture, *Aspergillus*-specific antigen should be assessed in the blood sample and in the pericardial fluid, and antifungal therapy should be started quickly. When diagnosed, the recommended treatment of myocardial abscess and mural endocarditis is surgical excision. Despite antifungal therapy, almost all cases of cardiac aspergillosis are fatal. It has been advocated that serial echocardiography should be performed early after the diagnosis of pulmonary aspergillosis.12,16 Successful treatment with combined surgical and medical treatment has been described in pericardial involvement.12

REFERENCES