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Aortic thrombus during invasive aspergillosis in a kidney transplant recipient

Sir,

Involvement of the large vessels has been rarely described during invasive aspergillosis (IA), especially after solid organ transplantation. It affects poorly the prognosis of this fungal infection and was often reported as a postmortem finding.

We report the case of a massive floating aortic thrombus diagnosed simultaneously with a pulmonary IA in a 54-year-old kidney transplant recipient. Kidney transplantation was performed in June 2003 with an uneventful early course. Maintenance immunosuppressive therapy consisted of prednisone, mycophenolate mofetil (MMF) and cyclosporine A (CsA). The occurrence of many complications, including extensive skin herpetic infection, cytomegalovirus (CMV) disease and post-transplantation diabetes mellitus (PTDM), resulted in prolonged hospital stay. In September 2003, two weeks after successful treatment of acute graft-rejection, the patient developed fever with a dull chest pain. Arterial gasometry revealed a moderate hypoxemia and hypocapnia. Chest X-ray showed pneumonic infiltration with right paracardiac cavitations; angio-CT scan confirmed the pulmonary nodules [Figure 1] and showed a massive floating thrombus of the ascending aorta [Figure 2]. The diagnosis of IA was made in view of the isolation of *Aspergillus fumigatus* on the culture of the fluid of bronchoalveolar lavage. Electrocardiography, transoesophageal echocardiography and serum tests ruled out other predisposing factors for thrombosis, mainly endocarditis. Therapeutic regimen, associating reduction of the doses of MMF and CsA with initiation of Voriconazole, resulted in prolonged apyrexia with regression of the pulmonary radiological abnormalities. The substantial reduction in the size and mobility of the aortic thrombus, documented after...
the initiation of antifungal therapy, is an indirect argument in favor of the causative role of aspergillosis in the thrombotic process. However, this etiology is difficult to establish without microbiological or pathologic studies of the thrombotic material. Indeed, surgical removal of this thrombus was considered highly risky and our patient, who died suddenly five months later, did not have necropsy verification.

The mechanism of arterial obstruction during IA remains hypothetical. It was suggested that aggregation of the fungus cells in the intima stimulates endothelial cells to become prothrombotic by expressing thromboplastin that activates Factor II and initiates the extrinsic coagulation cascade.[6]

The vasculotropic character of aspergillus explains the disseminated forms of IA as was observed in our patient who developed adrenal insufficiency and multiple small areas of cerebral and splenic infarcts. All these disorders improved after initiation of antifungal therapy.

In our experience, this is the first case of IA among 330 renal transplantations performed since 1986. Of note, the diagnosis of IA was made during the same period of works of building renovation around our transplant unit. In addition, our patient had many of the reported predisposing factors to this fungal infection including prolonged hospitalization, PTDM, CMV infection and acute graft-rejection.[2]

This case is of clinical interest because of the unusual occurrence of aortic thrombus in a kidney transplant recipient with IA. Voriconazole was effective on fungal infection without substantial interference on graft function or CsA blood levels. Sudden fatal issue could be related to rupture or complete occlusion of the aorta. As a result, surgical removal of infective thrombi should be considered even in cases where the antimicrobial therapy seems to be effective.

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