Mucormycosis masquerading as an endobronchial tumor

Sir,

A 58-year-old woman with poorly controlled diabetes mellitus presented with a 2-week history of high grade fever with rapidly progressive shortness of breath 1 day prior to admission. She was a known case of dilated cardiomyopathy and diabetic nephropathy. Systemic examination revealed tachypnea, decreased breath sounds bilaterally with basal rales, decreased intensity of heart sounds and audible S3, tender hepatomegaly with fluid thrill.

The complete blood cell count showed a hematocrit of 30% and a leukocyte count of 32,000/mm^3. The serum glucose level was 245 mg/dl, serum urea nitrogen level was 168 mg/dl, serum creatinine was 4.6 mg/dl and urinalysis revealed 20-30 pus cells with proteinuria. Serum procalcitonin was 6.77 ng/ml. Arterial blood gas

value showed metabolic acidosis (anion gap-26 mmol/l) with negative blood and urine ketones. The chest radiograph demonstrated cardiomegaly and blunting of both cardiophrenic angles [Figure 1]. Ultrasonography of chest and abdomen showed bilateral moderate pleural effusion and small ascites. Transthoracic echocardiography revealed ejection fraction of 25% with global hypokinesia and moderate pericardial effusion.

Clinical symptomatology suggested a provisional diagnosis of urinary tract infection with septic shock and cardiac failure. The patient was started on broad spectrum antibiotics and vasopressors. Urine output remained between 500 and 800 ml/day. By the fifth day, she was afebrile, weaned off inotropes and non-invasive ventilator support but required alternate day hemofiltration. Blood and urine cultures sent at admission were sterile.

It was on ninth hospital day that she again became febrile and her chest X-ray revealed left upper lobe collapse [Figure 2]. Her oxygen requirement increased, she was intubated and put on mechanical ventilator. Computed tomography (CT) of the chest revealed heterogeneous mass measuring 5.3 × 3.2 cm in the left hilum, completely obliterating the left upper lobe [Figure 3]. Fiberoptic bronchoscopy revealed reddish polypoidal growth causing almost complete obstruction of left upper lobe bronchus. Histopathology examination of the bronchial biopsy showed irregular broad aseptate hyphae branching at right angles, consistent with mucormycosis. [Figure 4]. The patient was started on liposomal amphoterecin and surgical resection of upper lobe was planned, but patient's condition deteriorated. She developed multi-organ failure and had massive hemoptysis followed by cardiac arrest and succumbed to treatment on the 24th day of hospitalization.

Mucormycosis is an opportunistic infection causing deep tissue infection. It is a potentially fatal opportunistic fungal infection caused by certain fungi of the order Mucorales. It occurs commonly in immunocompromised hosts but has also been demonstrated in patients with normal immune

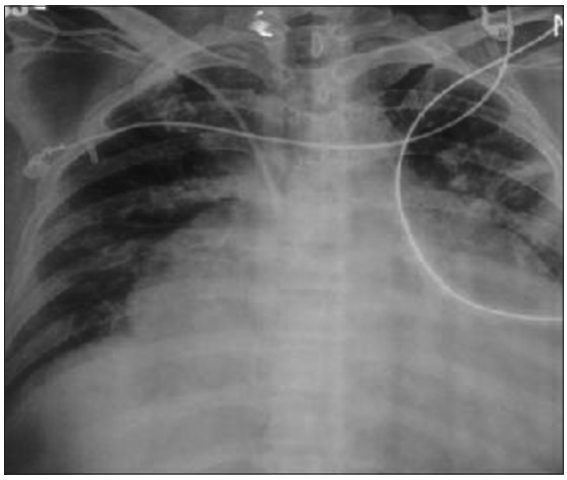

Figure 1: Chest X-ray at the time of admission showing cardiomegaly and blunting of both the cardiophrenic angles

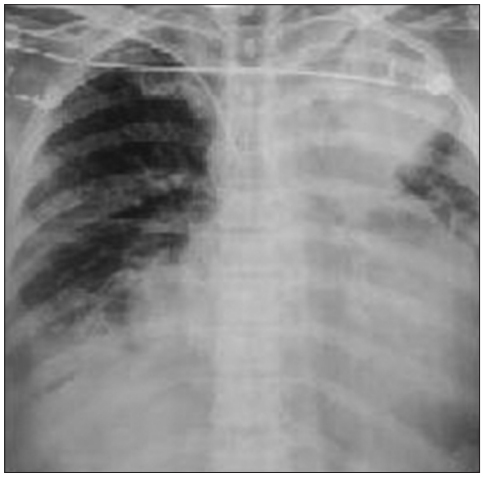

Figure 2: Chest X-ray on the 9th day of admission shows collapse of the left upper lobe

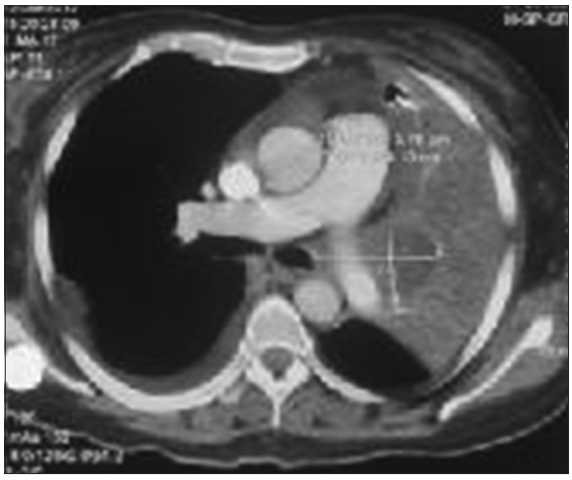

Figure 3: CECT chest showing an endo-bronchial mass lesion in left upper lobe bronchus causing distal atelectasis

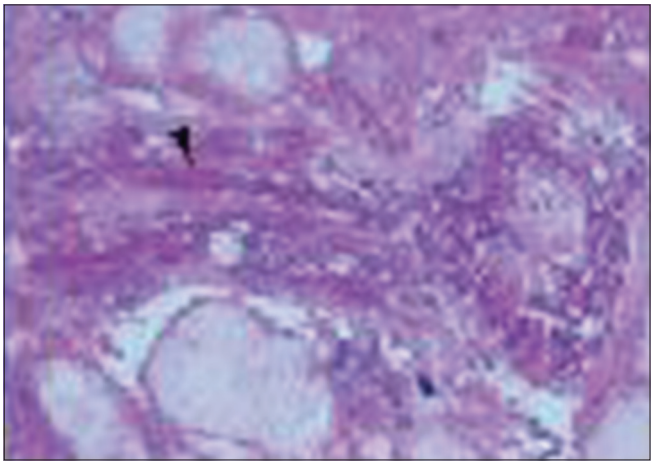

Figure 4: Microphotograph of histopathology section from the lesion (hematoxylin-eosin stain) shows broad aseptate hyphae with frequent right-angle branching suggestive of mucormycosis

status. Five predominant forms of mucormycosis seen clinically are: Rhinocerebral, pulmonary, disseminated, cutaneous (particularly burn wounds), and gastrointestinal mucormycosis. Pulmonary mucormycosis is commonly seen in patients with hematological malignancies. Common pulmonary manifestations include consolidation (66%) or cavitation (40%), less commonly as a solitary pulmonary nodule and multiple mycotic pulmonary artery aneurysms.[1]

Presentation as an endobronchial mass lesion is extremely rare. The production of spores, which become airborne, leads to the primary route of inoculation in the respiratory tract. Risk factors for mucormycosis are neutropenia, lymphopenia, hyperglycemia, pre-existing renal failure and prolonged steroid use. Our patient had uncontrolled diabetes with renal failure, a combination that made her an ideal host for mucormycosis.

The clinical manifestations of pulmonary mucormycosis cannot be easily distinguished from those of bacterial infection. The most prominent physical finding is bilateral fixed rales or pleural rubs. Rapidly progressive pneumonia is seen in those patients with underlying hematological malignancies. Few case reports in the literature have mentioned a distinct clinical syndrome of mucormycosis in diabetics, as endobronchial polypoidal lesion.[2] Nearly, 78% of patients of pulmonary mucormycosis can have an acute presentation and our patient had a very rapidly progressive clinical course as well.

Definite diagnosis is made by a biopsy and histopathology. The characteristic histological feature is tissue invasion by aseptate, broad, right angled branching hyphae with a propensity to invade blood vessels. Culture is considered as the gold standard for disease diagnosis and species identification. However, unfortunately the recovery of fungi from culture is less sensitive due to hyphal damage during processing of the specimen. Hence, it has been agreed that microscopic identification of characteristic fungi invading affected tissues should be considered significant [Figure 4].[3]

Therapy for pulmonary mucormycosis should be aggressive as the fungus produces a locally invasive infection with a high fatality rate. According to IDSA Guidelines, amphoterecin B deoxycholate (0.7-1.0 mg/kg/d) or its lipid formulations (5 mg/kg/d) remain the primary antimicrobial agent for mucormycosis.[4]

Mucormycosis has an extremely high mortality rate ranging from 25% to 80%. Prognosis of pulmonary mucormycosis depends on the underlying immune-compromised state. Survival rate is better in diabetics (45-60%), organ transplant recipients than patients with renal insufficiency.[3] Our patient had the two most important predisposing factors that made the chances of her survival worst. The common causes of death are fungal sepsis (42%), respiratory insufficiency (27%) and hemoptysis (13%). Massive hemoptysis may be due to vascular invasion, multiple pulmonary artery pseudoaneurysms and can lead to death due to asphyxiation.[5]

Patients on medical therapy alone have a mortality of 55% compared to 27% in patients who get surgical treatment with or without medical treatment.[3] Surgical therapy is highly recommended and must be performed without delay because of the aggressive nature of the disease with propensity for vascular invasion. We managed our patient conservatively and planned for lobectomy, but she developed multi-organ failure and did not give time for surgery. In conclusion, pulmonary mucormycosis is a rare, rapidly spreading disease and fatal disease, which can masquerades many common conditions including lung cancer. A high level of index should be kept in diabetics and immune-compromised hosts who fail to respond to antibacterial therapy. Early recognition and aggressive management requires systemic antifungal therapy, surgical resection, coupled with control of underlying disease.

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