Empyema with Pleuropulmonary Mucormycosis

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Abstract
Pleuropulmonary mucormycosis is a relatively rare disease and that too localised disease is very rare but has got better prognosis. Maintaining a high level of suspicion is important in right clinical setting with pleuropulmonary involvement that fails to antibacterial agent either clinically or radiologically.

Introduction
Mucormycosis is a serious, relatively uncommon invasive fungal infection and one of the most aggressive and lethal invasive mycoses. Physicians caring for patients with specific risk groups (Table 1) should be acutely aware of the enhanced susceptibility of infection with the mucormycosis. Here we report a case who initially was treated as a case of empyema with antibiotics and antitubercular drugs and finally turned out to be mucormycosis and was successfully treated with amphotericin, antibiotics and surgery.

Case Report
58 yr old MR known diabetic was admitted with fever, cough and breathlessness x 6 months. Fever was low grade, continuous, evening rise, no chills/rigors with max temp 100.4° F. Cough was associated with yellow expectoration no haemoptysis. Breathlessness was gradually progressive, MRC grade II. He also gave history of decreased appetite and generalised weakness. There was no history of pain chest, hoarseness of voice, wt loss. He had past history of pulmonary TB in 2001 and 2007. He was non smoker, denies consuming alcohol and exposure to sex workers. On examination: average built, ht - 174 cm, Wt – 62 kg, Temp - 99.8°F, Pulse - 90/min, RR - 18/min, BP - 110/70 mm Hg, Clubbing present, grade III. No lymphadenopathy, pallor, icterus, cyanosis, pedal oedema. Spo2 94%. Respiratory system- Inspection- decreased movement right side, chest expansion: 2 cms. Palpation: Trachea: slight deviation to left. Tactile vocal fremitus decreased right infrascapular region, Percussion: -Stony dull note right infrascapular region. Auscultation-decreased breath sounds right infrascapular region -occ crackles, no wheeze/rub. Other systems- Normal. Investigations : Complete blood count- normal, Blood sugar F-266 mg%, PP-336 mg%, bun-19 mg%, Creatinine-1.2 mg%, S.Liver function test- normal, LDH-386 IU/L, Na-145 meq/l, K-4.5 meq/l, Serum Proteins- 6.5 g%, Albumin- 3.7 g%, Globulin -2.8 g%. Sputum for AFB/Fungal stain- negative. Sputum for malignant cells- negative. Mantoux test –Negative. Chest X Ray- non homogeneous opacity right lower zone. USG Chest- Large pocket of about 300 ml seen in right thoracic cavity. Pleural fluid aspiration : Pus like appearance, pH- 7.2, WBC- 16,200 cells/ micro L, RBC- 1400/ micro L, Protein- 3.6 gm/dl, Glucose- 42 gm/dl, LDH- 220 IU/L, ADA - 42 IU/L, ZN Stain- no AFB seen, Gram stain- numerous neutrophils, no organisms seen, KOH mount- no fungal hyphae/yeast forms seen, Culture- sterile after 07 days. CT Chest (Figure 1). Effusion right lower zone with subjacent air bronchogram. He underwent medical thoracoscopy under LA after 3 weeks of hospitalisation because of poor response to antibiotics and ATT. Medical thoracoscopy (Figure 2) revealed extensive pleural thickening with soft necrotic and shaggy pleural surface. Indications of thoracoscopy (Table 2). Multiple biopsies were taken. Intercostal tube was inserted. Biopsy sample were sent for histopathological examination (Figures 3 - 6) which revealed mucormycosis.

Patient was initially treated on iv antibiotics, insulin and other supportive measures for 1 wk of hospitalisation with no response. He was thereafter put on empirical ATT on basis of clinical and radiological picture and ATT continued for 3 weeks. After getting the diagnosis of mucormycosis on pleural biopsy he was put on liposomal amphotericin and broad spectrum antibiotics continued. Liposomal amphotericin B was continued for 3 weeks at the dose of 150 mg/day and thereafter put on plain amphotericin due to financial constraints. Patient responded after 2 weeks and became afebrile. His condition remained critical for about 1 month. Amphotericin B continued.

Table 1: Risk Factors for Mucormycosis

| 1. Diabetes mellitus esp. with ketoacidosis |
| 2. Steroid therapy |
| 3. Neutropenic patients |
| 4. HIV patients |
| 5. Haematologic and solid malignancies |
| 6. Malnourished individuals, especially children |
| 7. BM transplant recipients |
| 8. Persons in Renal Failure |
| 9. Intravenous drug abusers (at risk for cerebral Mucormycosis) |
| 10. Desferoxamine therapy and all causes of iron overload |

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Fig. 1: CT Chest showing right pleural effusion with subjacent consolidation
tools. Three factors are key to a successful therapy for mucormycosis: (1) reversal of the underlying predisposition; (2) aggressive surgical debridement; and (3) aggressive antifungal therapy, with early initiation and high drug doses. Use of lipid formulations at doses of 15–20 mg/kg per day maximizes the amount of amphotericin B delivered to the tissues as well as the speed of its delivery. Posaconazole, an experimental triazole antifungal agent, has been shown to be active against mucormycosis. The optimal duration of therapy for mucormycosis is not known precisely. If possible, antifungal administration should be continued for at least 3 months after clinical and radiological cure.

This case stresses the fact that presentation of pulmonary mucormycosis depends on the associated clinical disorder. Patient with leukaemia and lymphoma often have diffuse parenchymal disease refractory to medical and surgical therapy. Occasional patients with serious but non malignant underlying medical condition may develop diffuse disease. Diabetics are different from others in sense that they may have local disease amenable to therapy with surgery and amphotericin. This case is perfect example that localised disease can be successfully treated and managed. Another peculiarity in this case is that here there is localised lower lobe involvement whereas case reviews have shown that this disease has predilection for involvement of upper lobe. This case was also not treated with liposomal amphotericin B/ amphotericin plain in adequate dose as described in various literature due to cost factors and fluctuating azotaemia and dyselectrolytaemia. There were no evidence of vascular invasion in this case either clinically or radiologically. The delay in therapeutic decortication was due to diagnostic uncertainty and in the belief that the patient will respond to antibiotics and for 3 months and the drug was interrupted 4 times in between because of electrolyte derangement and azotaemia. His condition improved gradually and was referred to CTVS for decortication surgery which he underwent successfully. He is at present doing well and is on follow up for diabetes.

Discussion

Fungi from the order Mucorales are the aetiologic agents of mucormycosis. Despite the name of this infection, mucor is not the most common genus recovered from patients. In the normal human lung, mucorales spores are inhibited from germinating into hyphae by alveolar macrophages. However, in high risk groups (Table 1) the spores germinate, hyphae develop and invades blood vessels and surrounding tissues. Microscopic examination and culture of biopsy samples from the involved area are critical in making an accurate diagnosis. However negative cultures are the rule in pulmonary mucormycosis. Molecular methods of speciation are still used only as research tools.
thereafter ATT. This patient was also not in position to undergo any surgical procedure initially due to poor general condition. Ideally one decortication would have been better option both for diagnosing as well as for therapy.

Conflicts of Interest
None identified

References

Fig. 6: Gomorie Methamine silver stain shows broad non septate hyphae with irregular width with branching at right angles.