Case report

Pseudomembranous necrotizing bronchial aspergillosis

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Abstract. We report a case of pseudomembranous necrotizing bronchial aspergillosis in a patient with acute myelocytic leukaemia who died of massive haemoptysis. Lobar collapse was demonstrated on chest radiography. CT showed a marked necrotic thickening of the lobar bronchus with extension of the disease in to the peribronchial region.

Pulmonary aspergillosis presents with a wide spectrum of pathology in humans. This includes allergic bronchopulmonary aspergillosis, aspergiloma, and semi-invasive and invasive aspergillosis, depending on the immunological state of the host. Invasive aspergillosis in an immunocompromised patient with severe neutropenia is a dreaded form of aspergillosis. Most cases of invasive aspergillosis present in an angioinvasive form, but less commonly an airway form of invasive aspergillosis can also be present [1]. Pseudomembranous necrotizing tracheobronchial aspergillosis has been recently reported in the clinical literature as a rare type of airway invasive aspergillosis [2–6], but little attention has been given to the radiological findings. To our knowledge, there has been only one case report describing the radiographic and CT findings of invasive tracheal aspergillosis [7]. We describe the radiographic and CT findings of a case of pseudomembranous necrotizing bronchial aspergillosis which resulted in massive haemoptysis and death.

Case report

A 56-year-old man presented with fever, cough, and dyspnoea. He had been recently diagnosed as having acute myelocytic leukaemia, for which he received chemotherapy including daunorubicin and cytarabine. After the completion of chemotherapy, the patient became neutropenic with a total leucocyte count of 300/mm³, followed by the development of fever and dyspnoea. On chest radiography an homogeneous opacity was noted along the medial portion of the left upper lung zone with deviation of the trachea to the involved side as well as elevation of the left hilum and the left hemidiaphragm, consistent with collapse of the left upper lobe (Figure 1). Bronchoscopy demonstrated a whitish pseudomembrane, almost completely covering the entire mucosa of the left upper lobar bronchus. Bronchoscopic biopsy was performed on this area. Microscopic examination of the specimen (Figure 2) showed that the pseudomembrane was composed predominantly of inflammatory cells and fungal hyphae, with destruction of the underlying respiratory epithelium. Transmural extension of the inflammation and hyphae to the level of the bronchial cartilage was also noted. Typical hyphae were septate, with progressive and dichotomous branching. Culture of the bronchial washing fluid grew Aspergillus species.

The patient was promptly treated with intravenous amphotericin B. His general condition gradually improved, but cough with blood-tinged sputum was still present 7 days after commencement of treatment. His white blood cell count at this time was 3500 per mm³. Follow-up chest radiography showed partial re-expansion of the left upper lobe (Figure 3). Chest CT (Figure 4) on the same day demonstrated extensive thickening of the left upper lobar bronchial wall with luminal narrowing. No contrast enhancement was noted in this thickened bronchial wall. On lung window setting, peribronchial infiltration was demonstrated in the parahilar region of the left upper lobe. In spite of gradual improvement of his symptoms with continued amphotericin B infusion, the patient died of sudden massive haemoptysis 4 weeks after the treatment had started.

Discussion

Pseudomembranous necrotizing tracheobronchial aspergillosis is a new clinical entity that is manifested as an extensive transmural necrotizing
bronchitis with pseudomembrane formation in immunocompromised patients [2–6]. It differs from aspergillary bronchitis, which is a relatively common and indolent process showing a superficial saprophytic growth of hyphae on the bronchial mucosa [1]. Since the first description by Pervez et al [2], sporadic clinical cases of pseudomembranous necrotizing bronchial aspergillosis have been reported [3–6]. However, the radiological findings have been poorly described.

Logan et al reported the radiographic and CT findings of nine patients with pathologically proven invasive aspergillosis of airways [8]. Seven of nine patients showed peribronchial consolidation and centrilobular nodules on CT. All of these seven patients had invasive aspergillosis of the peripheral airways. Two had combined tracheal or main-stem bronchial involvement, but CT was normal. The only report describing CT findings of invasive airway aspergillosis involving the trachea, showed increased paratracheal opacity, tracheal narrowing and pneumomediastinum [7]. Our case shows unique appearances of CT findings of invasive pseudomembranous bronchial aspergillosis, including marked necrotic thickening of the involved bronchial walls with peribronchial extension of the process and adjacent bronchopneumonia.

The pseudomembrane is composed of necrotic tissue, exudate and fungal hyphae. It is usually characterized by completely destroyed mucosa, transmural bronchial necrosis and the limited

**Figure 1.** Chest radiograph shows a homogeneous opacity along the medial portion of left upper lung zone with deviation of the trachea to the involved site and elevation of left hilum and left hemidiaphragm, suggesting collapse of the left upper lobe.

**Figure 2.** (a) Photomicrograph of bronchoscopic biopsy specimen shows the eroded respiratory mucosa, focally covered by granular pseudomembrane composed of inflammatory exudate and hyphae. Bronchial cartilage was exposed (arrows) (hematoxylin eosin stain; original magnification ×100). (b) Typical hyphae are septate, and branching is progressive and dichotomous (original magnification ×400).

**Figure 3.** The follow-up chest radiograph after 1 week shows re-expansion of the left upper lobe with peribronchial infiltration.
extension of inflammation and hyphae into the peribronchial tissues [2, 3, 5]. In our case, although the specimen was small, it was sufficient to diagnose airway invasive aspergillosis, which showed eroded respiratory mucosa accompanied by the partial exposure of bronchial cartilage with inflammatory cells and exudate containing hyphae. In addition, CT showed marked bronchial wall thickening and peribronchial air density (Figure 4a), which strongly suggested involvement of the entire bronchial wall and peribronchial tissue.

There has been a reported case of fatal haemorrhage during bronchoscopy when attempting to remove the obstructing pseudomembrane in a patient with extensive necrosis which extended from the bronchial wall into the pulmonary artery [4]. Massive haemoptysis was also seen in our patient and led to death. Based on the observation that pulmonary vessels are intimately associated with diseased bronchi on CT, arterial invasion can be expected as a cause of massive haemoptysis.

In conclusion, when CT of an immunologically compromised patient presenting with acute respiratory symptoms such as fever, cough and dyspnoea, shows an extensive necrotic thickening of the bronchi or trachea with invasion to the adjacent structures, radiologists should consider the possibility of pseudomembranous necrotizing tracheobronchial aspergillosis.

Acknowledgments

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References