Lobar Necrosis from Pulmonary Embolism

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Figure 1. Evaluation of the middle lobe cavity. (A) Initial chest radiograph revealed a middle lobe cavity. (B) Chest computed tomography (CT) showed the large middle lobe cavity surrounded by the medial and lateral segmental bronchi. (C) Gross tissue analysis demonstrated an occlusive thrombus in the pulmonary artery branch to the middle lobe (black arrow). Histologic analysis revealed (D) organizing thrombus within the pulmonary artery showing signs of recanalization (4× magnification), and (E) diffuse alveolar damage with loss of architecture, septal thickening, and neutrophil infiltration (10× magnification). (F) New pulmonary emboli (green arrows) were found on CT angiogram performed after resection of the middle lobe.

A 63-year-old male heart transplant recipient with recurrent lower extremity deep vein thrombosis, receiving warfarin, with chronically reduced glomerular filtration rate (30 ml/min), presented with worsening cough and dyspnea. Laboratory testing revealed normal blood cell counts, mildly elevated C-reactive protein, and international normalized ratio of 1.8. Subsequent radiograph revealed a middle lobe cavity (Figure 1A). Bronchoalveolar fluid cultures revealed *Candida albicans*, but antifungal therapy cleared the organism after 2 weeks. Nevertheless, the cough persisted, and the cavity progressed over the following 6 weeks (Figure 1B). Given the uncertain etiology of the cavity and persistence of symptoms, thorascopic middle lobectomy was performed, during which the majority of the lobe was found to be necrotic and filled with ~10 ml of thick brown liquefied debris. Major and minor fissures were “complete,” preventing any collateral blood supply to the middle lobe. Gross and histological examination revealed an occlusive thrombus in the pulmonary artery branch to the middle lobe. The majority of the lobar parenchyma had undergone necrosis, leading to the cavity formation, and the walls of the cavity adjacent to the minor and major fissures revealed microscopic features of alveolar

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damage/hemorrhage, septal thickening, and neutrophil infiltration (Figure 1C–1E), suggesting ongoing ischemic damage. Bacterial, viral, mycobacterial, and fungal cultures of the lobar tissue, as well as bronchoalveolar lavage fluid and cavity debris, were negative. Postoperatively, computed tomography pulmonary angiogram confirmed new bilateral pulmonary emboli (Figure 1F) but preserved right bronchial artery. Pulmonary infarction from segmental artery occlusion can be associated with cavitation, especially in the setting of underlying infection in up to 10% of cases (1–5), but complete lobar necrosis does not usually occur. Because the middle lobe is usually supplied by only one branch of the pulmonary artery, it may be uniquely susceptible to lobar necrosis in the setting of an occlusive thrombus within the middle lobe artery. This case, therefore, highlights the importance of pulmonary circulation in maintaining viability of lung parenchyma. ■

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