This ten-year-old white boy underwent operative correction of Fallot's tetrad. Immediately postoperatively, copious amounts of yellow secretions were aspirated from the endotracheal tube. Within six hours, the secretions became frothy pink. Progressive tachypnea and cyanosis culminated in death approximately 36 hours following operation.

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Diagnosis: Pulmonary Alveolar Hemorrhage

The chest roentgenogram (Fig 1) shortly prior to death shows both lungs to be homogeneously opacified. The cardiac contour is obscured and prominent air bronchograms are seen throughout. The findings are typical of alveolar involvement. At necropsy, the ventricular septal defect appeared securely closed and the obstruction to right ventricular outflow had been ablated. The lungs showed many consolidated areas of alveolar hemorrhage in each lobe. There were no pulmonary emboli or hemorrhage in other organs. Figure 2A shows the gross appearance of the lung. Figure 2B is a photomicrograph of a histologic section taken from one of the consolidated areas (H and E × 400). It shows alveoli which contain many erythrocytes with a few macrophages and clumps of fibrin.

Pulmonary alveolar hemorrhage is a nonspecific response which may occur in association with various conditions including hemorrhagic hypotension, severe renal disease, alteration of bleeding and clotting mechanisms, gram negative sepsis, and operations utilizing extracorporeal circulation. It may also occur idiopathically, as in primary pulmonary hemosiderosis. The bleeding may be produced by the release of humoral agents, acting either at the pulmonary venule or arteriole level, which cause intense vasoconstriction and resultant damage of capillaries with extravasation of erythrocytes.

The clinical course is characterized by progressive dyspnea, tachypnea, and cyanosis without manifestations of circulatory failure. Patients frequently produce large amounts of frothy red secretions. Death usually occurs within 24 to 36 hours. The severe respiratory distress without ventricular gallop rhythm helps distinguish pulmonary hemorrhage from pulmonary edema. The roentgen findings are: (1) large areas of opacification with or without air bronchograms; (2) nonsegmental distribution; and (3) occasional perivascular infiltrates progressing to diffuse pulmonary opacification.

The appearance of pulmonary opacification shortly after operation helps to distinguish pulmonary alveolar hemorrhage from pneumonia and oxygen toxicity. Pneumonia of this magnitude so soon after operation would be quite unusual unless the patient were receiving immunosuppressive medications. The "timing" does not exclude pulmonary infarction; however, this does not usually result in extensive involvement of all lobes. Obstructive atelectasis can also closely resemble pulmonary hemorrhage. However, signs of volume loss (i.e., diaphragmatic elevation, mediastinal shift, overexpansion of adjacent segments) are not present in pulmonary alveolar hemorrhage. Pulmonary edema is the principal condition to be considered in differential diagnosis. In pulmonary alveolar hemorrhage, Kerley B lines and pulmonary venous distention are absent and perihilar distribution of the infiltrate is less likely.

References


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