Inhalational Anthrax
Gross Autopsy Findings

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A 61-year-old Vietnamese woman with a history of hypertension had rapidly worsening dyspnea, chest pain, and a blood-tined productive cough for 2 days. She had immigrated to the United States more than 20 years ago, had not traveled recently, and was previously in her regular state of good health. She worked in a hospital stockroom, adjacent to the mail room. She did not have direct contact with patients nor did she handle the mail.

On admission to the hospital, she was afebrile, with an oxygen saturation of 92% on room air. Physical examination results were notable for bilateral apical rales and edema of the lower extremities. A chest radiograph revealed pleural effusions and increased pulmonary vascular markings, and initially she was treated with diuretics for congestive heart failure and antibiotics for a possible pulmonary infection.

Worsening respiratory failure required emergency intubation, and a chest computed tomographic (CT) scan showed mediastinal adenopathy. Bronchoscopy disclosed friability and hemorrhage of the tracheobronchial mucosa, and bronchi contained purulent secretions. A follow-up CT scan revealed marked mediastinal hemorrhage and worsening effusions. Bilateral chest tubes drained 2000 mL of serosanguinous fluid. She was treated for septic shock followed by disseminated intravascular coagulation and multiorgan system failure. Blood cultures grew Bacillus anthracis. Polymerase chain reaction studies for B anthracis on blood, pleural fluid, and bronchial specimens further confirmed the diagnosis. She died 3 days after admission.

At autopsy, there were numerous foci of hemorrhage of the mediastinum. The hilar and peribronchial lymph nodes were enlarged, soft, and confluent hemorrhagic (Figure 1), and hemorrhage tracked along the peribronchial parenchyma (Figure 2). The trachea was encased in hemorrhagic soft tissue (Figure 3). Microscopically, the mediastinal lymph nodes showed acute hemorrhagic effacement with necrosis and karyorrhexis (Figure 4). Touch preparations of these lymph nodes revealed rare, scattered, gram-positive bacilli without definitive sporulation. Although the lungs were edematous, there was no bronchopneumonia. She did not have gastrointestinal or skin lesions, meningitis, splenomegaly, or mesenteric lymphadenopathy. The localization of the findings to the mediastinum and the absence of mesenteric or intestinal findings excluded the gastrointestinal tract as the portal of infection (ie, gastrointestinal anthrax). The cause of death was certified as inhalational anthrax and the manner of death was homicide.

This death from inhalational anthrax demonstrates the ability of this infection to mimic other diseases: congestive heart failure, influenza, and community-acquired pneumonias. With a high clinical suspicion and blood cultures, the clinical diagnosis of anthrax can be made. In such hospital deaths, the autopsy is important to confirm the clinical diagnosis and the portal of entry.

Unfortunately, as most forensic pathologists can attest, some people, no matter how ill, do not seek medical attention. Forensic pathologists see the natural pathologic progression of many untreated diseases. The extent of the disease may be so advanced that the pathologist wonders how the person could not have sought medical care. Substance abuse and psychiatric illness are frequent common denominators in such deaths. If a person died at home from inhalational anthrax, it would be up to the medical examiner or coroner to make the diagnosis. In addition, with increasing numbers of “suspected” or “possible” cases of anthrax (eg, a postal worker who is found dead at home), a pathologist must quickly be able to exclude and include inhalational anthrax.

The key to the diagnosis of inhalational anthrax at autopsy is to recognize the distinct gross findings that then will lead to further testing. The hemorrhagic mediastinitis observed at autopsy in this death was striking. In addition to the mediastinal hemorrhage, the hilar or peribronchial lymph nodes were enlarged, soft, and blue due to necrosis of the lymphoid tissue with replacement by hemorrhage. Grossly, the hilar lymph nodes had the appearance and consistency of postmortem clot or of accessory spleens. Once these initial autopsy findings are noted, touch preparations and cultures of the lymph nodes or pleural fluid must be performed to identify the bacillus. The bacilli are abundant in nontreated persons but may be sparse in antibiotic-treated patients.

References