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Case report

Lepidic predominant adenocarcinoma with aerogenous spread of mucin in a young patient — A case report

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ABSTRACT

We present a unique case of a 26 year-old female non-smoker who expired following treatment for presumed pneumonias. At autopsy, lepidic predominant adenocarcinoma with aerogenous spread of mucin without evidence of invasion, a rare diagnosis that previously would have fallen under the umbrella of "bronchioloalveolar carcinoma," was found. Histopathology showed mucin-secreting neoplastic cells lining the alveolar walls, as well as exfoliated and dense aggregates of mucinous debris filling the alveoli. The immediate cause of death was respiratory failure, most likely due to the significant amount of tumor-produced mucin that filled the alveolar spaces, which literally drowned the patient.

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Introduction

Although lung cancer is the leading cause of cancer death for both men and women in the United States, lung cancer in young women is rare (U.S. Cancer Statistics Working Group, 2013). From 2006 to 2010, the incidence of non-small cell lung cancer in women aged 25–29 was 0.6% (Howlader et al., 2013). Furthermore, while adenocarcinoma is the most common type of lung cancer, tumors formerly called "bronchioloalveolar carcinoma" comprise a small subgroup of adenocarcinoma (Thompson, 2004; Travis et al., 2011). In this case, we present a young woman found at autopsy to have lepidic predominant adenocarcinoma with aerogenous spread of mucin without evidence of invasion, discuss her diagnosis in relation to the revised classification of adenocarcinoma, and highlight the significant amount of mucinous debris that lead to hypoxia, respiratory failure, and death.

Initial presentation

A 26 -year-old Hispanic female patient with a history of uncontrolled diabetes mellitus presented to the emergency room with chronic cough, following one month of progressive dyspnea. She endorsed eight months of dry cough and a history of 30-pound weight loss over two months. She denied fevers, chills, night sweats, or sick contacts. Her social history was negative for smoking, traveling, and environmental exposure.

She had been hospitalized twice, four and eight months prior to admission, at outside medical centers. She was treated with IV antibiotics and a course of corticosteroids for presumed pneumonia but with minimal improvement in her symptoms.

On physical exam, the patient was tachycardic and tachypneic. Her oxygen saturation was 82% on ambient air, which increased to 98% on 3 l of oxygen by nasal cannula. She appeared in mild distress without accessory muscle use. She had diffuse bilateral inspiratory rales, greater in the upper two-thirds of her chest without wheezing. Pertinent laboratory findings were WBC 13.6 K/cu mm (normal range: 4–10 K/cu mm), lactic acid 1.7 mmol/L (normal range: 0.5–2.2 mmol/L), and an arterial blood gas showing hypoxemia.

Imaging on presentation was significant for dense perihilar opacities in both lungs (Fig. 1), areas of consolidation (Fig. 2), ground glass pulmonary nodular infiltrates (Fig. 3), and cystic spaces (Fig. 3).

Hospital course

Upon admission, cultures were drawn, and the patient began broad-spectrum antibiotics for suspected community and/or hospital acquired pneumonia. She was placed on airborne precautions for suspected miliary tuberculosis given the diffuse nodular infiltrates, history of weight loss, and diabetes out of control. As antibacterial therapy failed to improve the patient's condition, super-infection with MRSA as well as a fungal etiology for the necrotic pulmonary manifestation were considered and appropriately covered. Despite the treatment efforts, the

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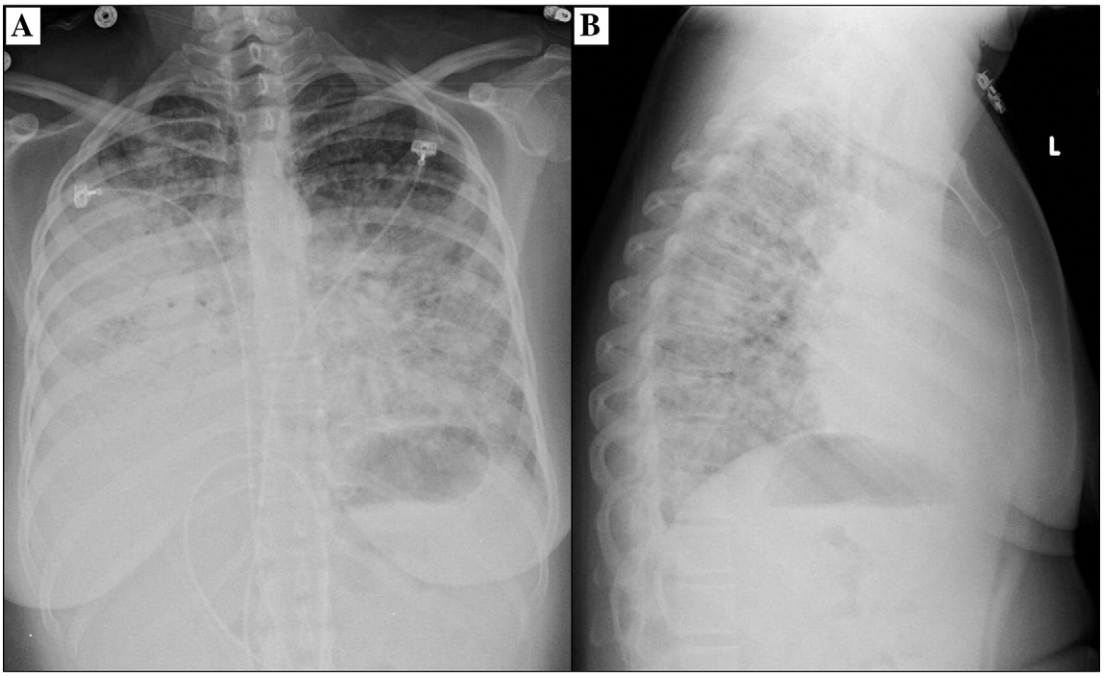

Fig. 1. PA (A) and lateral (B) chest X-ray on presentation showing dense perihilar opacities in both lungs. The pulmonary vasculature is mildly prominent.

patient developed progressive respiratory failure and was intubated on hospital day 3. A bedside bronchoscopy with bronchoalveolar lavage was performed which returned only mucopurulent exudate and macrophages, which had phagocytized neutrophils.

Details of her previous hospitalizations were obtained which revealed radiographic evidence consistent with the imaging from the current admission. She had undergone a bronchoscopic biopsy that was indicative of interstitial pneumonitis. She was treated with intra-venous antibiotics and a course of corticosteroids for suspected pneumonia. Together with a comprehensive infectious workup, including negative cultures, smear AFB's, and fungal serologies from this and prior admissions, the patient seemed to have an underlying interstitial lung disease, possibly cryptogenic organizing pneumonia. While the use of corticosteroids was strongly being considered, the patient developed shock and rapidly required increasing vasopressor and oxygen administration. She expired on hospital day 5.

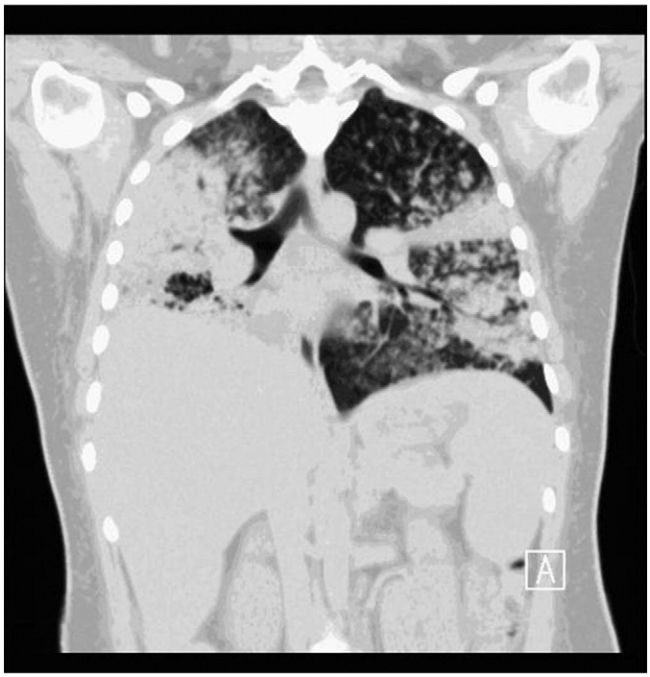

Fig. 2. CT chest without intravenous contrast obtained on admission. This coronal view shows dense consolidation in the left upper lobe, the right middle lobe, and the right lower lobe.

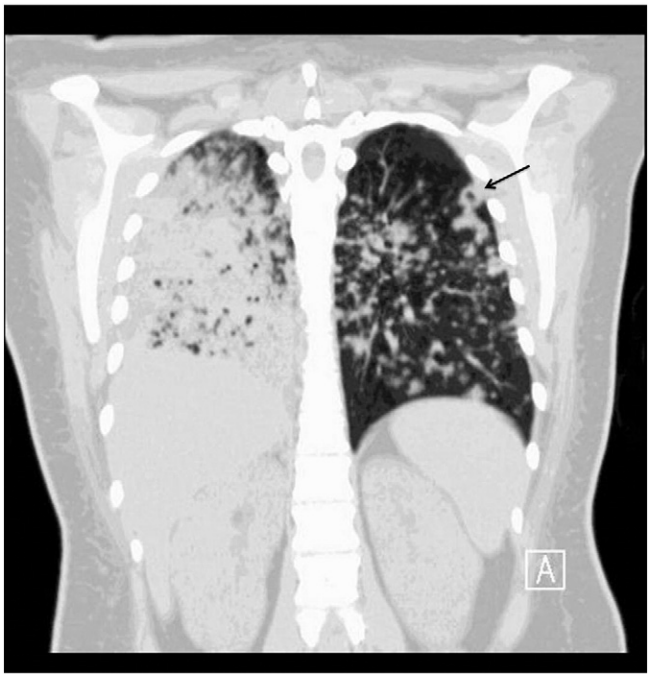

Fig. 3. CT chest in coronal view showing right lung consolidation with diffuse ground glass pulmonary nodular infiltrates in both lungs. The arrow highlights a pseudo-cavitation in the left upper lobe.