A 19-year-old black woman presented with a three-month history of malaise. She reported mild fever, slight cough, nasal drainage, sore throat, and chest pain for one week. She denied weight loss, chills, anorexia, or sputum production. There was a history of genital herpes and gonorrhea. Her mother had been treated for tuberculosis nine years ago. The patient had normal chest radiographic findings and negative PPD two and one year, respectively, prior to presentation.

The chest film now showed multiple, noncalcified nodules varying from 1-4 cm in diameter (Fig 1). Findings on physical examination, and laboratory data, including pregnancy test, were normal. Material obtained by percutaneous needle biopsy was nondiagnostic.

Wedge resection of a nodule from the left upper lobe was performed. The lesion was grayish-tan, thick walled, cystic, and contained yellowish, necrotic-appearing material. Microscopically, its wall contained collagen bundles and some chronic inflammatory cells (Fig 2 and 3). Results of stains and culture for fungi, acid-fast bacilli and routine organisms were negative.

More laboratory tests were obtained postoperatively. Westergren sedimentation rate was 64 mm/hr. Angiotensin-converting enzymes were 24 IU per liter (normal). Rheumatoid arthritis factor was negative. Complement fixation antibodies for blastomycosis and histoplasmosis were <1:8.

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FIGURE 1

FIGURE 2

FIGURE 3
Diagnosis: Pulmonary hyalinizing granuloma

The term pulmonary hyalinizing granuloma (PHG) was introduced by Engleman et al. to describe a clinical complex of pulmonary nodules of unknown etiology. Although over 50 cases have been reported, the disease is generally unknown. It may be incidentally discovered by chest radiography. The first considered diagnosis is usually malignancy, metastatic or primary (Hodgkin's disease). Other differential alternatives are infection (histoplasmosis), amyloidosis, rheumatoid nodules, and granulomatous disease (Wegener's, sarcoidosis, lymphomatoid, plasma cell).

Diagnosis is made by evaluation of adequate biopsy material. Nodule centers have characteristic haphazard hyalinized collagen lamellae. Chronic inflammatory cells are seen at the nodule's periphery and interspersed between lamellae. Other types of nodules have distinguishing features, including amyloid, plasma cells, vasculitis, necrosis, and organisms. Electron microscopy may be helpful in differentiating PHG from amyloidosis.

Etiology of PHG is unknown. The nodules probably represent an immune response to the antigenic stimulus of infection or an autoimmune process. Sclerosing mediastinitis, which has a microscopic picture much like that of PHG, can be caused by histoplasmosis or tuberculosis. Previous exposure to fungal or mycobacterial disease can be documented in the majority of patients with PHG. Histoplasma capsulatum has (rarely) been seen with PHG nodules. Sixty percent of patients in the series of Yousem and Hochholzer had some clinical or laboratory evidence of autoimmune disease. Still, there is no consistent relationship of PHG with any other illness, infectious or immune.

Clinical presentation is usually a mild malady with pulmonary (cough, dyspnea, chest pain) or general systemic (fever, sinusitis, pharyngitis, fatigue) complaints. Most patients are middle-aged. There is no apparent racial or sexual predilection.

The usual radiographic appearance is multiple, frequently bilateral, nodules. They are larger than tuberculous or histoplasmosis nodules and have poorly defined borders. The lesions grow slowly, can coalesce, and show no calcification or necrosis. Pulmonary hyalinizing granuloma is not mentioned in recent radiologic texts, reflecting its clinical obscurity.

The prognosis of PHG is usually benign. Normal longevity is common. With enlargement and fibrosis, pulmonary insufficiency may occur. More worrisome is the occasional evolution into retroperitoneal fibrosis or sclerosing mediastinitis with entrapment of vessels or airway.

The current patient is apparently the youngest to be reported with PHG. The primary preoperative diagnosis was metastatic malignancy. There was a close family history of tuberculosis, but no evidence of that or histoplasmosis. Similarly, there was no indication of an autoimmune disease.

References