Chest radiographs revealed superior mediastinal enlargement. Computed tomography of the thoracic inlet and neck revealed a large (12 cm in diameter), low-density mass, which involved the right carotid artery to the right and the trachea to the left (Fig 1).

At surgery, the tumor was found to contain clear fluid and was completely excised via a cervical incision. Microscopic study disclosed a fibrotic membranous wall with thymic tissue (Fig 2) with no evidence of malignancy.

The patient recovered from surgery and six months later remained asymptomatic. On physical examination, ptosis had disappeared, and anisocoria was less evident than it had been initially.

**Discussion**

Horner’s syndrome may occur with a lesion anywhere along the sympathetic pathway, but it usually results from cervical sympathetic chain compression by a malignant tumor. With a lesion of the brain stem or cervical spinal cord, other signs of neurologic dysfunction usually predominate. Second-order preganglionic involvement may occur with lesions of the superior mediatinum, the apex of the lung, or the neck, whereas postganglionic involvement may be the result of an abnormality of the internal carotid artery, a lesion in the middle cranial fossa or cavernous sinus, or a migraine equivalent.

Thymic cysts may occur at any anatomic level, from near the base of the neck to the diaphragm. Although distant or discontinuous accessory lobes commonly occur within the anterior mediastinal and cervical regions, thymic cysts are often found near the base of the heart.

Thymomas often undergo cystic degeneration, at times so extensive as to result in a gross picture similar to that of a thymic cyst. Radiography does not allow distinction between benign thymic cysts and other potentially malignant lesions.

However, careful histologic examination of the cyst will invariably disclose residual tissue in its wall. Thymic cysts should, therefore, be excised, principally to differentiate them from thymomas.

We believe that the most important clue to diagnosis of a thymic cyst as the cause of Horner’s syndrome is the clinical history. Thus, diagnosis may be suspected in asymptomatic subjects with mediastinal enlargement in whom Horner’s syndrome is not accompanied by other neurologic signs or symptoms.

**References**

Lavage fluid collected from both sites was turbid, with black particulate matter resembling a dilute solution of activated charcoal. Black, granular particulate material was collected by passing the lavage fluid through filter paper. Centrifugation of the fluid produced a black pellet separated from clear fluid by a thin layer of cells (Fig 2). Analysis of the BAL fluid disclosed trace amounts of lidocaine but no cocaine or other toxins. The particulate matter was insoluble in organic and polar solvents and was considered by the toxicology lab to be inert carbon. The cell count of the BAL fluid from the right upper lobe was $17.2 \times 10^6$ cu mm, with 84 percent macrophages, 10 percent neutrophils, and 6 percent lymphocytes; from the left lower lobe, $4.7 \times 10^6$ cu mm, with 98 percent macrophages and 2 percent lymphocytes. Macrophages contained large amounts of darkly pigmented granules (Fig 3), which were negative for iron by Prussian blue stain.

On further questioning, the patient denied any inhalation exposure but reported heavy cocaine smoking for the previous three months, culminating in nearly continuous use for three days prior to presentation. She prepared her cocaine by “cooking” it in water and baking soda until a white paste formed. The paste was placed on a small piece of steel wool, heated with a cigarette lighter, and smoked until all that remained was a black, tarry residue. This residue was scraped free, reheated, and vigorously smoked until it was gone.

After three days of intravenous erythromycin administration, the patient was afebrile and felt much better. Her cough persisted but was less severe, and the amount of sputum decreased. Examinations of sputum and BAL fluid disclosed normal respiratory flora. Blood and urine cultures were negative. Serum Legionella titers were IgM <1:16 and IgG <1:32. Serum cytomegalovirus titers were IgM <1:16 and IgG <1:4,096. Her energy panel was nonreactive. Her
lymphocyte count was 374, with a total CD4 count of 3. A repeat chest film on the fourth hospital day showed resolution of the right upper lobe infiltrate and complete clearing of the infiltrate in the left lower lobe. Pulmonary function testing done on that day showed mild reduction in the FEV₁ and FVC (70 percent and 78 percent predicted, respectively) and a normal FEV₁/FVC ratio of 75 percent. On the seventh hospital day the patient was discharged in good condition, but did not return for follow-up appointments.

**Discussion**

Inhalation of volatilized cocaine is an increasingly popular form of habitual drug use in this country. Recent reports indicate that cocaine smoking is capable of producing a myriad of pulmonary complications, including chronic cough and shortness of breath, a decrease in FEV₁, FVC, and diffusion capacity for carbon monoxide; pneumothorax; pulmonary edema; alveolar hemorrhage; BOOP; asthma; and hypersensitivity pneumonitis. However, the mechanisms by which cocaine smoking causes pulmonary injury are not well understood. Other reports have suggested damage to the pulmonary capillary endothelium secondary to intense cocaine-induced vasoconstriction, a direct toxic effect of cocaine, and neurogenic or cardiogenic pulmonary edema, but none of these mechanisms has been proved.

Aside from adverse effects of the cocaine, pulmonary complications may result from impurities inhaled during the process of cocaine smoking. Cocaine smokers take frequent, heavy inhalations of cocaine smoke. Although cocaine alkali is easily volatilized, large amounts of particulate matter and smoke can be inhaled during cocaine freebasie. As cocaine free base is smoked, a dark, tarry residue forms on the inside of the bowl and the barrel of the pipe. This residue is considered by many smokers to be concentrated cocaine and is scraped free, reheated, and vigorously smoked. Inhalation of nonvolatile by-products of cocaine burning has been demonstrated by cocaine smokers in whom persistent cough productive of black sputum and at least one case of thermal burns in the posterior pharynx have been reported.

Our patient was producing clear sputum when she came to the emergency room. She did not receive activated charcoal, and bronchosopic examination of the nares, posterior pharynx, and upper airways showed no sign of carbon deposition or thermal injury. Nevertheless, BAL produced copious amounts of carbonaceous material and alveolar macrophages packed with pigmented granules, which stained negative for hemosiderin. These BAL findings suggest penetration of large amounts of particulate matter into the alveolar spaces, which probably resulted from overwhelming or impairment of normal clearance mechanisms by near-continuous cocaine smoking. The BAL findings in cocaine smokers include eosinophils, hemosiderin-laden macrophages, and an increase in protein concentration; but, to our knowledge, alveolar deposition of carbonaceous material has not been previously reported.

Whether the carbonaceous material found in the alveolar spaces of our patient was responsible for her symptoms is not clear. Previous case reports have attributed lung infiltrates in cocaine smokers to allergic alveolitis, pulmonary edema, and BOOP. However, our patient had no clinical evidence of pulmonary edema and improved rapidly without corticosteroid treatment. Her presentation was not consistent with community-acquired pneumonia, and no evidence of an opportunistic infection was found. It is likely that the cough, dyspnea, fever, and pulmonary infiltrates in our patient were due to an acute inflammatory reaction secondary to alveolar deposition of particulate material. Another possibility is metal fume fever secondary to repeated heating of the stainless steel wool inside her pipe and inhalation of the resulting fumes.

In a recent editorial, Lerner concluded that, in view of the increasing number of cocaine-related emergency room visits, there has been underreporting of the complications of cocaine smoking. This report demonstrates that heavy alveolar deposition of carbonaceous material may occur from smoking cocaine, which may explain in part one of the mechanisms by which cocaine smoking causes acute respiratory disease.

**References**