We describe identical, but mirror-image, twin brothers with cytologically similar pulmonary adenocarcinomas in mirror-image locations and nearly the same age at diagnosis. A literature search found only four publications citing bronchogenic carcinoma in twins. Two pairs of twins had squamous cell,2,3 one pair had anaplastic,4 and another pair had bronchioloalveolar cell carcinoma.5 We believe this report to be the first of identical twins with pulmonary adenocarcinoma of the nonbronchioloalveolar type. The environmental risk factors that these twins have in common, smoking habits, and the same occupation (carpenters), are similar.6 Their propensity to smoke is not only an environmental risk factor, but also may be genetic since it has been reported that monozygotic twins have a higher degree of concordance with regard to smoking than do dizygotic twins.7

First-degree relatives of lung cancer probands have a twofold to fourfold increased risk for lung cancer or other cancers, many of which are not related to smoking.8-11 Ooi and coworkers8 showed that the risk of familial lung cancer was increased even after allowing for age, sex, and indices of smoking and occupational exposures. Of course, cigarette smoking is a definite risk factor for the development of lung cancer of all histologic types, including adenocarcinoma.10 The twin cases we report are also of interest because of recent work implicating phenotypic mutation of the K-ras oncogene in the pathogenesis of adenocarcinoma of the lung.12 This oncogene is highly specific for this particular tumor, and mutational activation of the K-ras oncogene is rarely seen with either adenocarcinomas of extrapulmonary origin or with other bronchogenic neoplasms.12 Our twin cases underscore the importance of this work.

REFERENCES

Hemodynamic Compromise Secondary to a Mediastinal Bronchogenic Cyst*

Patrick M. Fratellone, M.D.; Neil Coplan, M.D.; Michael Friedman, M.D.; and Paul Stelzer, M.D.

Bronchogenic cysts are not commonly the cause of severe symptoms, and often present only as an abnormality on chest roentgenogram. We report an unusual patient with a mediastinal bronchogenic cyst associated with rapid hemodynamic deterioration secondary to compression of vital structures.(Chest 1994; 106:610-12)

Bronchogenic cysts are closed epithelial-lined sacs that develop from abnormal lung budding during embryologic development, and which account for 10 to 15 percent of all primary mediastinal tumors. The most common presenting symptom is chest pain.1 A patient presenting with dyspnea and back pain developed hemoptysis and rapid hemodynamic deterioration secondary to a large bronchogenic cyst.

CASE REPORT

A 27-year-old man with no significant medical history presented to the emergency department after experiencing several episodes of dyspnea and back pain. The blood pressure was 110/70 mm Hg, pulse was 84 beats/min while in a supine position, and there was no significant orthostatic change. There was no significant venous distention, the carotid upstrokes were full, and no bruits were present. Findings from chest, cardiovascular, and abdominal examination were unremarkable. There was neither peripheral edema nor cyanosis, and peripheral pulses were normal. Chest radiograph showed a large right-sided mediastinal mass, sharply demarcated posteriorly, measuring approximately 13x8 cm in maximal diameter (Fig 1). A two-dimensional echocardiogram demonstrated significant indentation of the left atrium.

The patient underwent computed tomography of the chest to further evaluate the mass (Fig 2 and 3). A large mass in the mediastinum in the subcarinal location was noted, compressing the root of the aorta and the left atrium anteriorly. There was also marked compression of the right and left pulmonary arteries and the main-stem bronchus, and the esophagus was displaced posteriorly. A thin septum within the mass was present. There were infiltrates present in the right lung field.

The patient began to have hemoptysis 2 h after initial presentation, and was noted to have diminished pulses in the right brachial artery.

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chial artery and carotid artery, hypotension, and a significant pulsus paradoxus. He became cyanotic, was intubated, and taken directly to the operating room for surgical exploration. The patient had a right posterolateral thoracotomy with the lung retracted anteriorly revealing a large posterior mediastinal cystic structure containing white creamy fluid. The structure was adherent to the lateral posterior border of the right main-stem bronchus and the esophagus, without adherence to the aorta. The mass was resected successfully and the patient was extubated without difficulty.

Culture of the fluid from the mass showed no growth. Pathologic findings were consistent with a bronchogenic cyst; with respiratory columnar epithelial lining containing chondroid, lymphoid, and neural components.

Seven days after presentation, the patient was discharged from the hospital.

**DISCUSSION**

Bronchogenic cysts are closed sacs that develop as supernumerary buds from the primitive respiratory system. Bronchogenic cysts usually occur along the tracheal bronchial tree, but they can occur within the lung parenchyma, preterinal area, supraclavicular space, or within the pericardium. Bronchogenic cysts represent 18 percent of all primary mediastinal tumors. The majority of the thoracic bronchogenic cysts occur in the posterior or middle mediastinum. The most common structures the cyst may be adherent to include the esophagus, lung, transbronchial tree, and the pericardium (49 percent, 33 percent, 32 percent, and 27 percent, respectively).

Since the first description in 1859, numerous reports of bronchogenic cysts have appeared in the literature. Most bronchogenic cysts are reported in children, where they frequently present as emergencies secondary to airway obstruction and respiratory distress. In contrast, early reports of the incidence of bronchogenic cysts in the adult population eluded to the fact that the cysts rarely caused symptoms, either because they are too small or due to their inferior location.

Reports after 1971 demonstrated that adults with bronchogenic cysts may present with serious symptoms. St. Georges et al reviewed the results of resection of 86 bronchogenic cysts over a 20-year period, 66 of which were mediastinal cysts. The majority of the patients with mediastinal cysts were symptomatic at the time of operation. Only 7 of 66 had an acute onset of symptoms, while 37 of 66 had progressive symptoms. The severity was reported as mild, moderate, and very severe in 9, 23, and 12 patients, respectively. The symptoms reported included chest pain...
We present an unusual case of a patient with chronic hepatitis C who experienced dyspnea, fever, and cough after 2/3 months' treatment with interferon. His radiograph demonstrated diffuse pulmonary infiltrates and bronchoalveolar lavage fluid showed an increase in lymphocytes, especially CD8-positive cells. The lung biopsy findings were bronchiolitis obliterans organizing pneumonia (BOOP). The pulmonary symptoms disappeared and the chest radiograph became normal after interferon therapy was discontinued and corticosteroid therapy was given. Interferon is suspected to be responsible for the BOOP.

(Chest 1994; 106:612-13)

**BOOP=bronchiolitis obliterans organizing pneumonia**

Drug-induced pulmonary diseases present a wide variety of clinicopathologic findings such as cough, fever, bronchospasm, noncardiac pulmonary edema, pulmonary effusion, interstitial pneumonia, and pulmonary fibrosis. Various kinds of drugs have been reported to be the causative agents. Recombinant alpha interferon has been developed and is now clinically used in patients with chronic type non-A, non-B hepatitis. However, several side effects have been reported. These include fever, myalgias, leukopenia, and nephritis. Herein we present a patient with chronic hepatitis C who experienced bronchiolitis obliterans organizing pneumonia (BOOP) after 2/3 months' treatment with recombinant alpha interferon.

**CASE REPORT**

A 64-year-old man who had a history of blood transfusion in 1953 was first suspected of having liver damage by chance in January 1992. He was diagnosed as having chronic hepatitis C by serologic findings and was treated with glycyrrhizin. However, his serum transaminase levels were worsening; aspartate aminotransferase was 244 IU/L, and alanine aminotransferase was 253 IU/L. His diagnosis of chronic active hepatitis was established by liver biopsy specimen in September 1992. He then received recombinant human alpha interferon (alpha-2b): 10 million units/d for 11 weeks, 6 days a week for the first 2 weeks and 3 days a week for 9 weeks, starting from the end of September. After 1 month, his serum transaminase levels decreased to within the normal range. In late November, he developed dyspnea, fever, and nonproductive cough. No remarkable abnormality was found in the laboratory data except for an enhanced erythrocyte sedimentation rate (45 mm/h). The white blood cell count was 5,900/mm$^3$ (segmented 38 percent; stab, 1 percent; lymphocytes, 40 percent; monocytes, 12%

**Table 1—Lung Function Tests and Arterial Blood Gas Studies**

<table>
<thead>
<tr>
<th></th>
<th>Before Therapy</th>
<th>After Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC, %</td>
<td>1.98 (66)</td>
<td>3.09 (100)</td>
</tr>
<tr>
<td>FEV$_1$, %</td>
<td>1.98 (99)</td>
<td>2.42 (82)</td>
</tr>
<tr>
<td>PaO$_2$, mm Hg</td>
<td>68.2</td>
<td>100.9</td>
</tr>
<tr>
<td>PaCO$_2$, mm Hg</td>
<td>47.3</td>
<td>44.2</td>
</tr>
</tbody>
</table>

Interferon-Related BOOP (Ogata, Koga, Yagawa)