**DISCUSSION**

Round atelectasis is an uncommon lung pseudotumor that was first described by Loeschke in 1928 in the German literature, and as “the folded lung” by Blesovsky in English in 1966. It is a peripheral lesion consisting of atelectatic lung surrounded by fibrotic pleura with vessels and bronchi extending from it in a curvilinear fashion. Asbestos exposure accounts for the majority of cases reported. In the case series of Hillerdal of 74 patients with round atelectasis, 64 patients (86 percent) were found to have asbestos exposure. Other reported etiologies include congestive heart failure, postthoracotomy, posttraumatic rib fractures with hemorrhagic effusion, and posttherapeutic pneumothorax. Round atelectasis is most often noted as an asymptomatic, incidental finding on chest radiograph.

There are two theories concerning the possible pathophysiology behind the development of round atelectasis. One proposed by Hanke and Kretzschmar theorizes that round atelectasis develops as a result of a pleural effusion causing a partially aerated portion of the lung to float. A cleft subsequently forms in the collapsed section and that portion of the lung is forced to tilt on itself. As the effusion resolves, fibrinous adhesions bind the tilted portion of the lung in place resulting in an atelectatic parenchymal mass from which the characteristic curvilinear bronchi and vessels extend. A second theory advocated by Menzies and Fraser is that the primary insult leads to inflammation and fibrosis of the visceral pleural surface, and as the fibrosis contracts, the parenchyma is forced to buckle on itself leading to round atelectasis. The lack of significant pleural effusion in our patient is more consistent with this second theory.

To our knowledge, our case is the first description of round atelectasis as a complication of a bacterial pleuropulmonary process. *Legionella pneumophila* is often associated with pleurisy, even when it presents without pulmonary infiltrates, and we believe that this was the instigating insult causing inflammation of the pleura with development of round atelectasis. By CT scan, the lesion in our patient fulfills the criteria for round atelectasis as outlined by Lynch and colleagues in that: (1) the mass is associated with pleural abnormalities, (2) there is evidence of volume loss in the affected area, (3) there is partial interposition of lung between the pleura and the mass, and (4) there is a visible “comet tail” of vessels and bronchi sweeping into the lateral aspect of the mass.

Because most cases develop many years after exposure to asbestos and present as incidental findings in asymptomatic individuals, round atelectasis is often assumed to develop slowly over a number of years after asbestos exposure. In our patient, however, the development of round atelectasis was clearly documented from the initial presentation with only minimal pleural abnormality on the CT scan with the findings suggestive of round atelectasis only 2 years later. Multiple chest radiographs obtained in the interim demonstrate the progressive pleuropulmonary disease.

Although this is a rare occurrence, we believe that our case is instructive for various reasons: first, it is the first description of round atelectasis as a complication of *L. pneumophila* or any bacterial process; second, the diagnosis of round atelectasis may become more common as imaging techniques improve and, therefore, we should become familiar with its clinical correlates. Most importantly, suspecting and identifying round atelectasis may prevent unnecessary thoracotomies.

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**REFERENCES**


**Mirror-Image Tumors in Mirror-Image Twins**

David Morison, M.D.; Cesar V. Reyes, M.D.; and Morton S. Skorodin, M.D., F.C.C.P.

Genetic factors are known to play a role in causing lung cancer. Twin cases of bronchioloalveolar, squamous, and anaplastic bronchogenic carcinoma have been previously reported. We describe mirror-image twins with adenocarcinoma of the lung occurring in mirror-image locations. They shared smoking and an occupational risk, carpentry, in addition to identical genetic backgrounds. (Chest 1994; 106:608-10)

Smoking and other environmental pollutants play a causal role in lung cancer. Genetic factors also predispose to lung cancer. This is a report of

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pulmonary adenocarcinoma in identical, but mirror-image, twins with similar age of onset, site of origin, and histopathologic features.

CASE REPORT

Twin 1, a 60-year-old left-handed white male carpenter from central Illinois with no significant medical history presented in February 1991 complaining of night sweats, pleuritic left-sided chest pain, and a cough producing a yellowish green sputum for about 1 week. The patient denied exposure to dust, asbestos, and other chemicals while working. He had an 80 pack-year history of smoking and was a social drinker. His father died of lung cancer at age 59 years. Physical examination revealed a tender, firm 5.0 x 5.0-cm mass in the left paraspinal area. Rhonchi and crackles were heard on the left side of the chest. A chest radiograph showed an ill-defined density in the left upper lung field (Fig 1, left). There was neither mediastinal nor hilar adenopathy. A fine-needle aspiration biopsy of the paraspinal mass revealed poorly differentiated adenocarcinoma (Fig 2, top). The patient underwent radiation therapy but died in October 1991.

Twin 2, a 62-year-old right-handed male carpenter also from central Illinois with a 1-year history of chronic obstructive pulmonary disease and a 93 pack-year history of smoking was admitted to the hospital in October 1992 complaining of shortness of breath, cough with sputum production, pleuritic pain, fever, chills, and night sweats. Inspection of the chest revealed that the left hemithorax was larger than the right hemithorax, which had limited respiratory excursions. Chest radiograph showed infiltrates in the right middle and upper lobes, a suprahilar mass, and an ill-defined density in the right lower lung field. The patient was treated for a community-acquired pneumonia and follow-up radiograph showed clearing of pneumatic infiltrates, but with a right upper lobe mass remaining (Fig 1, right). When his acute illness improved, a computed tomographic (CT) guided fine-needle aspiration biopsy specimen of the right upper lung mass was diagnosed as poorly differentiated adenocarcinoma (Fig 2, bottom) and appears to be cytologically akin to that of his twin sibling. A CT scan of the chest also demonstrated an enlarged lymph node in the pretracheal region. The patient is currently undergoing chemotherapy and radiation therapy for palliative treatment of stage III adenocarcinoma.

Figure 1. Left, Chest radiograph of twin 1. Note left upper lobe ill-defined density. Right, Chest radiograph of twin 2. Note right upper lobe mass.

Figure 2. Top, The neoplastic cells are large with pale and poorly defined cytoplasm and variable, vesicular nuclei. Often the cells are arranged in clusters, papillae, and pseudoacini (Papanicolaou stain, original magnification X400). Bottom, The tumor cells are closely similar with frequent papillary arrangement indicating poorly differentiated adenocarcinoma (Papanicolaou stain, original magnification X200).
**DISCUSSION**

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,12 one pair had anaplastic,8 and another pair had bronchioloalveolar cell carcinoma.5 We believe this report to be the first of identical twins with pulmonary adenocarcinoma of the nonbronchioalveolar type. The environmental risk factors that these twins have in common, smoking habits, and the same occupation (carpentry), 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 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 distension, 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.

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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