heart beats, and the blood pressure rose again, permitting sternal closure.

REFERENCES

Isolated Intrapulmonary Adenopathy in Leukemia*

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Localized adenopathy causing atelectasis is an unusual manifestation of leukemia. During exacerbation of acute lymphocytic leukemia, a patient developed atelectasis on the basis of exobronchial compression by an enlarged lymph node. More obvious causes for the adenopathy seem to be excluded on the basis of clinical and post-mortem findings.

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During the clinical course of acute leukemia, the lung can be involved directly or secondarily. Demonstrable radiographic changes occur with collections of malignant cells, infection, hemorrhage, or complications of therapy. Enlargement of lymph nodes within the hila and mediastinum also is a common manifestation. Physiologic abnormalities of clinical symptoms due solely to the presence of malignant leukocytes in pulmonary parenchyma or thoracic lymph nodes, or both, are rare.

We have recently reviewed 50 cases each of adult acute myeloblastic and lymphoblastic leukemia and have found that approximately 30 percent of these patients have obvious radiographic adenopathy in the thorax during some phase of their illness. There was not a significant difference in the frequency of this involvement between the various cell types. We were not able to establish in all cases whether the radiographic abnormality was the result of inflammatory or neoplastic nodes, or even nodular parenchymal involvement.

CASE REPORT

In July 1968 at the age of 26 years, the patient became ill with pancytopenia, hepatosplenomegaly, and lymphadenopathy. Aspiration of bone marrow showed acute lymphocytic leukemia. She was treated with 6-mercaptopurine and prednisone, which resulted in a complete remission. In June 1971, the patient suffered a relapse, and vincristine and prednisone were administered without response. She was then given a combination of cyclophosphamide, cytosine arabinoside, and methotrexate with folic acid-SF (Leucovorin) rescue, which produced a second complete remission. At this time the patient received prophylactic intrathecal therapy with methotrexate and brain irradiation, as well as splenic irradiation. She was maintained on therapy with cytosine arabinoside and methotrexate until September 1972, when the bone marrow showed numerous lymphoblasts. A combination of therapy with L-asparaginase and methotrexate was given, and the patient entered a third complete remission. These two drugs were administered every 10 to 14 days as maintenance therapy.

In April 1973, the patient developed a cough productive of yellow sputum, along with a low-grade fever. A chest radiograph showed a left lower lobe density with atelectasis. Sputum cultures grew Hemophilus influenzae, and ampicillin therapy was instituted. The patient improved symptomatically, but the abnormality on the chest radiograph did not change. In July 1973, the productive cough recurred. A chest x-ray film in November 1973 showed an increasing density in the left lower lobe extending into the lingula. The patient continued to have a cough with purulent sputum and experienced a 2.3-kg (5-lb) weight loss. The findings from physical examination were normal. Sputum smears and cultures were negative for acid-fast bacilli, including Nocardia species. Candida albicans was grown from one sputum specimen, but two subsequent specimens were negative for fungi. Serologic tests for histoplasmosis, blastomycosis, candidiasis, and cryptococcosis were negative. Review of the patient’s chest radiographs, which were unchanged, revealed a probable exobronchial lymph node compressing the left lower lobar bronchus and lingular bronchus; however, bronchoscopic examination demonstrated no narrowing of the bronchial lumen.

Over a four-week period the patient’s symptoms sponta-
neously abated. A chest radiograph showed some clearing of
the infiltrate, with probable decrease in size of the bronchial
lymph node. In May 1974, the patient developed signs of a
mass in the right cerebral hemisphere. Carotid arteriograms
demonstrated the mass, but a brain biopsy was not diagnostic.
The patient failed to respond to a course of chloramphenicol
and developed progressive hemiparesis with signs of in-
creased intracranial pressure. Exploratory craniotomy was
performed in July 1974, and a necrotic mass was partially
resected. Histologic examination of the tissue was not diag-
nostic. The patient continued to deteriorate neurologically
and died in August 1974.

Therapy with L-asparaginase and methotrexate had been
administered every 10 to 14 days without interruption from
September 1972 until June 1974. Examination of bone mar-
row shortly before death showed continued complete remis-
lossion with no evidence of leukemia.

Pathologic Findings

At autopsy, the major pulmonary fissure on the left was
carefully dissected. Immediately deep to the visceral pleura
and loosely invested by pleura and alveolar connective tissue
was an irregularly enlarged lymph node. The node was
abutting on two second-order bronchi, one to the lingula and
the other to the lower lobe (Fig 1A). In comparison, the
remaining thoracic lymph nodes were quite small, typical of
lymph nodes in treated leukemia. Histologically, the intra-
pulmonary lymph node here described showed ill-defined
follicular architecture, with sparse population of monocytes,
characteristic of the depleted posttherapeutic lymph node
(Fig 1B). There were no nodular or diffuse leukemic infiltr-
ates. The lesion in the central nervous system was a large
right-sided brain abscess, which was the immediate cause of
death.

Discussion

Common radiographic manifestations of leukemia in
the chest include mediastinal and hilar adenopathy,
pleural effusions, and radiodensities secondary to infec-
tion or hemorrhage. Less commonly seen are nodular
leukemic infiltrates and diffuse leukemic infiltrates.
Pathologically, the only manifestation of disease at

![Figure 1A (left). Irregular 1.2-cm intrapulmonary lymph node in fissure with overlying pleura reflected superiorly abuts lingular bronchus (upper arrow) (enlarged to 1.6 times natural size). 1B (right). Microscopically, node had depleted follicular cortical region (at upper left) and scarred medullary region (at lower right) (hematoxylin-eosin, original magnification × 100).](image1)

![Figure 2. Exobronchial lymph node compressing left lower lobar bronchus and lingular bronchus (arrows).](image2)
Systemic Arteriovenous Fistula Simulating Severe Valvular Aortic Stenosis*

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A patient with a renal arteriovenous fistula is described. She was thought to have valvular aortic stenosis because of a history of rheumatic fever, symptoms of congestive heart failure and syncope, and the presence of a harsh systolic murmur with a thrill in the aortic area. Cardiac catheterization revealed a left-to-right shunt of 8.7 L/min. Ligation of the fistula resulted in complete relief of the symptoms and attenuation of the murmur.

The diagnosis of arteriovenous fistula is straightforward when a continuous murmur is detected at the site of a previous surgical procedure or trauma. Similarly, a peripheral location of the fistula may produce local signs such as a mass or swelling of an extremity which alert the clinician to the presence of the fistula. Occasionally, however, the location of the shunt is central and the only sign is the bruit which can be heard only over a limited area. In such a case, the fistula may be overlooked and the hemodynamic alterations of a high output state will ensue insidiously, mimicking signs and symptoms of more common cardiovascular diseases. The usual presentation in these cases is that of refractory congestive heart failure, manifesting at variable intervals after trauma or surgery, ranging from months to as long as 57 years.

We report a patient in whom a large arteriovenous fistula secondary to a nephrectomy, presented with signs and symptoms which mimicked those of severe aortic stenosis.

Case Report

The patient, a 70-year-old woman, was referred to Temple University Hospital because of poorly controlled congestive heart failure, chest pain, and a recent episode of syncope. She gave a history of repeated episodes of rheumatic fever, the last one at age 35. At age 33, she had right nephrectomy performed because of recurrent nephrolithiasis. Following this she had recurrent episodes of dull, retrosternal chest pain and dyspnea on exertion. In 1964, at age 60, she was started on digitalis and diuretics. Two weeks prior to her admission she suffered an episode of non-exertional syncope associated with chest pain. The syncope was thought to be a manifestation of aortic stenosis and she was referred for further evaluation.

References


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