echocardiographic results were important in assessing the hemodynamic importance of the tricuspid stenosis in this patient. An important aspect in this regard is the performance of readings during expiration and inspiration, as the right ventricular inflow may change up to three times with inspiration. Therefore, comparison before and after exercise is required at the same stage in the respiratory cycle. Exercise Doppler echocardiography will also be useful for assessing the tricuspid valve at subsequent follow-up examinations.

REFERENCES


Anterior Mediastinal Tumor of 30 Years' Duration*

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A 67-year-old woman had a large mediastinal mass with calcification. On roentgenogram of 30 years ago, the same mass without calcification was seen. The mediastinal mass was removed by surgery and proved to be a non-Hodgkin's lymphoma (NHL), most probably as a transition from a giant lymph node hyperplasia (Castleman's disease).

(Chest 1991; 100:569-70)

NHL = non-Hodgkin's lymphoma

A giant mediastinal mass originally described by Castleman in 1954, also called giant lymph node hyperplasia, is most often located in the middle or posterior mediastinum and calcification is rare. It is a lymphoid tumor of unknown origin. Association with Hodgkin's disease or non-Hodgkin's lymphoma (NHL) has been described, especially in draining lymph nodes.

We describe a patient with a large mass in the anterior mediastinum that had been present for 30 years.

FIGURE 1. Chest roentgenogram of 1968, showing large mediastinal mass with calcification.

FIGURE 2. Histologic section of the border of the mediastinal tumor with lymphoplasmacytoid cells invading the surrounding fatty tissue.

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CASE REPORT

In January 1988, a 67-year-old woman was admitted to our hospital with a history of progressive breathlessness during the last two years. She had never smoked and had no history of pulmonary diseases. A chest roentgenogram was made 30 years ago because of a tuberculous contact. She remembered that she had been told that she had an innocent lesion on the chest roentgenogram.

Physical examination at the time of hospital admission revealed mild dyspnea but no other abnormalities. The chest roentgenogram showed an enormous mass with extensive central calcifications (Fig 1). The chest roentgenogram from July 1959 showed the same mass; however, it was smaller and without calcifications. Spirometry, laboratory, and electrocardiographic findings were normal. Computed tomography of the thorax confirmed a large calcified mass in the anterior mediastinum without connections with the heart.

At thoracotomy, a mediastinal tumor, 13 x 11 x 6 cm, weighing 700 g, was removed. The arterial and venous supply arose from subclavian vessels.

Histologic examination of the resected specimen showed a mainly diffuse and focally follicular proliferation of lymphoplasmacytoid cells divided by fibrous tissue with foci of calcified material. The lymphoid proliferation did not respect the borders of the central tumor and invaded the surrounding fatty tissue (Fig 2), leading to the diagnosis of lymphoplasmacytoid NHL of low malignancy, probably as transition from a giant lymph node hyperplasia (Castleman's disease).

DISCUSSION

Giant lymph node hyperplasia was initially reported as a solitary mediastinal mass, but multicentric and extranodal disease is now well known.  Although the localized form is occasionally asymptomatic, often general symptoms of fatigue, pain, fever, anemia, and sometimes hyperimmunglobulinemia are present. Histologically, two distinct variants are recognized: the hyaline-vascular type, showing hyalinized follicle-like structures with extensive capillary proliferation, and the plasma-cell variant with the presence of "sheets of mature plasma cells and normal-to-large-sized follicle centers."

Clinically, nodule changes like Castleman's disease have been found in association with a number of different diseases such as acquired immunodeficiency syndrome (AIDS), Kaposi's sarcoma, rheumatoid arthritis, autoimmune diseases, and in lymph nodes draining Hodgkin's disease or NHL. In terms of differential diagnosis, lymphoplasmacytoid NHL should be considered. Since it is very unlikely to survive for 30 years with an untreated NHL, we regard the initial mass in the anterior mediastinum as a giant lymph node hyperplasia with a malignant transformation to NHL. A relation between the two conditions has already been described, but a transition into NHL has not yet been reported (to our knowledge).

The treatment of choice of giant lymph node hyperplasia is surgical removal, but due to the presence of NHL in this particular patient, radiotherapy was instituted postoperatively. Two years after removal of the tumor, no signs of recurrence were present.

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Massive ST-Segment Elevation without Myocardial Injury in a Patient with Fulminant Hepatic Failure and Cerebral Edema*

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A 49-year-old woman presented in fulminant hepatic failure. The ECG showed dramatic ST-segment elevation, suggesting diffuse myocardial injury. However, echocardiography, creatine phosphokinase enzyme determinations, and examination of the heart at autopsy (six days later) failed to demonstrate any physiologic, anatomic, or histologic evidence of abnormality. The appearance of ST-segment elevation in this setting should not prompt treatment for cardiac disease or limit the candidacy for liver transplantation of such critically ill patients.

(Chest 1991; 100:570-72)

CPK = creatine phosphokinase, FHF = fulminant hepatic failure

The appearance of ECG changes suggestive of myocardial injury in the absence of myocardial cell necrosis, known as pseudoinfarction, has been well described in many textbooks and articles. To the best of our knowledge, however, this is the first report of its association with fulminant hepatic failure (FHF) and cerebral edema.

CASE REPORT

A previously well 49-year-old woman presented with nausea, vomiting, and malaise of four days' duration. The ECG obtained on admission and a chest x-ray film were normal. The patient was alert and oriented. Severe acidosis (pH, 7.02; serum bicarbonate, 4.1 mEq/L) and hypoglycemia (glucose, 14 mg/dl) were present. During the next 24 hours she became increasingly jaundiced and obtunded. Extremely high levels of liver transaminases (11,000 to 14,000 IU/L) and severe coagulopathy (prothrombin time, 41 s) were noted. The diagnosis of FHF was made. Large volumes of intravenous fluid, fresh-frozen plasma, and sodium bicarbonate were infused. Thirty-three hours after admission, the patient was transferred to our institution to be considered for liver transplantation.

Her laboratory results on admission to our hospital were remarkable for a serum sodium concentration of 160 mEq/L (normal, 136 to 146 mEq/L) and a serum osmolality of 367 mOsm/kg (normal, 275 to 299 mOsm/kg). Serum magnesium, ionized calcium, potassium, and creatinine and blood urea nitrogen levels were normal. The serum phosphate level was 1.3 mg/dl (normal, 2.5 to 4.5 mg/ dl). The total serum bilirubin level was 7.1 mg/dl (normal, 0.3 to 1.5 mg/dl). The chest radiograph showed pulmonary edema.

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