peel was removed, the underlying lung was seen to expand, except for the right upper lobe, which contained the old tuberculosis. After surgery the patient did require a nasotracheal tube and respiratory support for several days, eventually undergoing tracheostomy. After 14 days of respiratory support the patient was weaned from the respirator. Her strength, pulmonary reserve, and chest x-ray film slowly improved over the ensuing nine months (Fig 2).

DISCUSSION

Collapse therapy for tuberculosis was one of the oldest effective methods of treating the disease. Collapse therapy took several forms, including plombage. Eloesser described extrapleural plombage, with a high complication rate of fistula and empyema. Alexander used semisolid paraffin in the extrapleural position. This resulted in a very thin fibrous envelope with almost no reaction of the surrounding tissue. In spite of the use of plombage as a salvage maneuver, the rate of closure of cavities was reported to be as high as 85 percent.

In this patient, roentgenographic findings in the early postoperative period revealed hazy infiltrates in the newly expanded lung, which were believed to represent either interstitial fluid or hemorrhage related to the sudden expansion of the pulmonary tissue after 35 years of compression. This radiographic picture gradually cleared as the pulmonary function improved in the early postoperative period. In spite of the development of what was apparently a small bronchopleural fistula (which healed spontaneously), the patient's postoperative convalescence was relatively smooth, and at the time of this writing, she is asymptomatic and has resumed full activities. This experience suggests that with the probability of finding normal pulmonary tissue with extrinsic compression, even 35 years of compression is no contraindication to decortication in an attempt to improve respiratory function.

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REFERENCES


Reversible Pulmonary Hypertension and Cardiac Failure with Chronic Recurrent Pancreatitis

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Acute pulmonary hypertension causing acute right heart failure occurring in a patient with chronic recurrent pancreatitis is described. The mechanisms that may be responsible for the pulmonary hypertension and heart failure are discussed.

A variety of pulmonary complications of acute pancreatitis has been described. A rather unusual sequel of idiopathic chronic recurrent pancreatitis,

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acute heart failure secondary to pulmonary hypertension, occurred in our patient whom we shall describe.

CASE REPORT

A 60-year-old woman was admitted to the Brooklyn VA Hospital on October 23, 1975 because of chest and epigastric pain and fever of about six days' duration. The chest pain was bilateral, more on the left, aggravated by inspiration and bending over. The epigastric pain was persistent with periodic exacerbation, nonradiating, associated with occasional nausea and vomiting. Chills, dyspnea, orthopnea, or paroxysmal nocturnal dyspnea were not experienced.

In 1971, she was hospitalized for epigastric pain and discharged with a diagnosis of idiopathic pancreatitis. She experienced several episodes of epigastric pain following her admission in 1971. Past medical history revealed resection of colon followed by ileotransverse colostomy for carcinoma of the ascending colon in 1961. No evidence of gall bladder or pancreatic disease was present at this time. Right radical mastectomy for carcinoma was performed in 1967. There is specifically no history of cardiac or lung disease in the past.

The following physical abnormalities were noted: distended neck veins with patient positioned at 45°. Crepitant rales were heard at both lung bases. A pleural rub was present at the left base. A right ventricular heave was palpated. A loud P2 right sided S3 gallop, and a soft systolic murmur at the apex were heard. A very tender, slightly compressible mass, which moved with respiration, occupied the entire epigastrium. There was no rebound tenderness. The bowel sounds were normal. Results of rectal and pelvic examinations were normal within range. There was no peripheral edema. Calf tenderness and Homans signs were absent.

The following laboratory results were abnormal: WBC 16,500 with 78 percent neutrophils; hemoglobin 12.2 gm percent; hematocrit 37.2 percent; glucose 140 mg percent; calcium 8.4 mg percent; phosphate 2.7 mg percent; alkaline phosphatase 129 μ/l; amylase 262, 350, 400 Somogyi units on three successive occasions. Thyroid function studies gave normal findings. The ECG showed atrial fibrillation-flutter with a ventricular rate of 120 to 150 per minute, right bundle branch block with left anterior hemiblock. ST elevation was present in leads V1-V3 and V6. Chest x-ray film showed clear lung fields and a heart of normal size. Plain film of the abdomen revealed a density in the epigastrium displacing the stomach shadow to the left and the transverse colon downward. A GI series showed compression of the lesser curvature of the stomach by an extrinsic mass. On the lateral projection, the body of the stomach was displaced anteriorly. The echocardiogram was interpreted to show hypertrophy of the right ventricle. The lung scan was normal. Arterial blood gases determined under ambient conditions: pH 7.46; PO2 37 mm Hg; PO2 84 mm Hg; O2 saturation 93 percent; HCO3 25 mEq/L. Catheterization of the right heart gave these results in mm Hg: right atrial mean pressure 11, right ventricular systolic pressure 35 and a diastolic 16. The pulmonary artery mean pressure was 25 mm Hg. These values were determined daily. By the third day, the cardiac pressures had returned to normal and clinically the patient was out of failure. The Swan-Ganz catheter was removed on the fifth day (Fig 1).

Course in the Hospital

The cardiac failure and pancreatitis were treated in a conventional way. The patient's temperature declined to normal by the third day. After the fourth week, the epigastric mass and tenderness were no longer present. Initial technetium sulphur and gallium scans showed the presence of a space-occupying lesion in the left lobe of the liver. Liver scans employing the same isotopes repeated four weeks later showed complete clearing. The changes were attributed to the inflammatory process which had originated in the pancreas and had extended to the left lobe of liver. Sonographic examination of the pancreatic area was interpreted to show the presence of a pseudocyst in the body of the pancreas. One year after discharge, the patient was asymptomatic and doing well.

DISCUSSION

A number of pulmonary abnormalities have been described in pancreatitis.1-3 The majority of these are nonspecific except for the pleural fluid which is usually an exudate, hemorrhagic or not, usually left-sided but characteristically rich in pancreatic enzymes exceeding their concentration in the serum. Interiano4 and colleagues, in an analysis of the records of 50 consecutive patients with acute pancreatitis, showed that nine had diffuse pulmonary infiltrates. Dyspnea and shock were common in these patients. Arterial hypoxemia was present in six of the nine patients studied. Five of the nine died, a mortality of 55 percent, and autopsies performed on these showed pulmonary congestion, hemorrhage and diffuse bilateral pneumonia.

Pulmonary artery pressures and pulmonary artery wedge pressures were not determined in the nine patients. Interiano et al4 attribute the acute respiratory distress experienced by these patients to a chemical pneumonitis which is brought about through the action of pancreatic enzymes on the lung substance. Zieve and Vogel9 have shown that in man, serum phospholipase A activity (a lecithinase that splits one fatty acid off

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the lecithin molecule) is increased in acute pancreatitis. Lecithin is one of the main components of the lung surfactant. Disruption of the lecithin molecule may lead to vascular damage, pulmonary congestion and atelectasis. Halmagyi et al measured the pulmonary arterial mean pressure and pulmonary wedge pressure in three patients with acute hemorrhagic pancreatitis. They were free of symptoms of cardiac or pulmonary decompensation. The pulmonary arterial pressure was elevated, whereas the pulmonary wedge pressures were normal in three patients. The question remains: does the degree of severity of the pancreatitis bear upon the presence or absence of pulmonary hypertension? In addition to pulmonary hypertension, our patient showed evidence of right heart failure, as shown in the elevation of pressures in the chambers of the right heart, the presence of a right-sided S3 gallop, and the distention of the neck veins. The cardiac arrhythmia and failure cleared about the same time as the pulmonary hypertension. It is reasonable to infer from this temporal relationship that the pulmonary hypertension was the prime cause, but may not represent the sole cause of the cardiac failure and arrhythmias. For example, Lefer et al have isolated a peptide from the serum of animals with acute pancreatitis that depresses myocardial contractility (MDF).

REFERENCES

We present the findings in a 56-year-old man with idiopathic dilatation of the pulmonary artery who had a large saccular dilatation of the pulmonary artery on the angiogram and systolic fluttering of the pulmonic valvar leaflet and dilatation of the pulmonary artery on the echocardiogram. Although a number of cases of large dilatations of the pulmonary artery have been previously described, the echocardiographic findings of the disease have not been reported.

For the past several years, echocardiography has been widely used as a noninvasive tool for the assessment of various forms of heart disease. To our knowledge, there is no report about the echocardiographic findings in idiopathic dilatation of the pulmonary artery. We report a case of the abnormality proved by angiocardiographic studies and cardiac catheterization.

CASE REPORT
A symptomatic 56-year-old man was referred to Kyoto Prefectural University Hospital for cardiac examination because of an abnormal cardiac murmur and chest x-ray film. The patient appeared to be in good health. The blood pressure was 146/86 mm Hg. A systolic pulsation was present maximally at the middle left subclavicular line on the third intercostal space. There was a grade-5/6 harsh ejection systolic murmur, and a grade-2/6 high-pitched early diastolic murmur was heard best at the third left sternal border. The second heart sound was widely split, and its pulmonic component was not accentuated.

A chest x-ray film showed apparently normal pulmonary blood flow and a prominent protrusion of the pulmonary arterial segment in the posteroanterior view. An electrocardiogram was normal. Laboratory data, including the results of pulmonary function testing, a serologic test, and an optical fundus test, were normal.

Echocardiographic Findings of Idiopathic Dilatation of the Pulmonary Artery*

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FIGURE 1. Posteroanterior view of pulmonary arteriogram, showing large saccular dilatation of main trunk of pulmonary artery and dilatation of trees of pulmonary artery.