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 arrhythmia. For example, Lefer et al. have isolated a peptide from the serum of animals with acute pancreatitis that depresses myocardial contractility (MDF).

REFERENCES

Echocardiographic Findings of Idiopathic Dilatation of the Pulmonary Artery*

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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 valvular 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 angiographic 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-5/6 high-pitched early diastolic murmur was heard best at the third left sternal border. The second heart sound was widely split, and its pulmonary 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.

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

CHEST, 71: 5, MAY, 1977
Cardiac catheterization showed the following pressures: right ventricle, 25/7 mm Hg; pulmonary artery, 25/10 mm Hg; and mean pulmonary capillary wedge pressure, 10 mm Hg. The data from the intracardiac blood samples were within normal limits. The angiocardiogram revealed a large saccular dilatation of the main trunk of the pulmonary artery and the dilated pulmonary arterial trees (Fig 1 and 2). The width of the main trunk (Fig 2, arrow) is about 9 cm.

Echocardiographic examination was performed with an ultrasonic scope (Aloka SSD-90)** and a 2.25-MHz transducer. The tracings were recorded on a strip-chart recorder at a speed of 100 mm/sec. Echoes of the pulmonic valve were obtained with the beam set in an inferior and lateral direction through the left intercostal sternal border. The echoes of the pulmonic valve showed fine systolic fluttering. M-mode echocardiographic scanning from the pulmonic valve to the main trunk of the pulmonary artery showed that the echoes of the pulmonic valve disappeared in the increasing lumen of the pulmonary artery (Fig 3). The width of the lumen (Fig 3, arrow) is about 5.7 cm.

**Discussion**

Enlargement of the pulmonary artery occurs in a large number of different diseases, such as pulmonary stenosis, atrial septal defect, etc.; however, idiopathic dilatation of the pulmonary artery is less common. In 1949, Greene et al. established the following criteria for its clinical diagnosis: (1) simple dilatation of the pulmonary trunk, with or without involvement of the arterial tree; (2) absence of abnormal intrapulmonary or extrapulmonary shunts; (3) absence of chronic cardiac or pulmonary disease, either clinically or at autopsy; and (4) absence of arterial disease, such as syphilis or more than minimal atheromatosis or arteriosclerotic. In 1960, Deshmukh et al. advocated the addition of a normal pressure in the right ventricle and pulmonary artery as the fifth criterion in the diagnosis. This case fulfills these criteria.

The systolic murmur may be the result of the turbulence of the flow of blood in the dilated pulmonary trunk. Chisholm advocated the concept of "trigonoidation" of the pulmonic orifice as the mechanism of the murmur. The fine fluttering of the pulmonic valve in our case may be due to the turbulence of the pulmonary arterial blood flow or the "trigonoidation" of the pulmonic orifice. M-mode echocardiographic scanning from the pulmonic valve to the pulmonary trunk demonstrated the significantly enlarged pulmonary artery. In this study the beam was directed in a left lateral direction through the third intercostal space at the left sternal border to detect the pulmonic valve, so the width of the pulmonic trunk obtained from the echocardiogram was rather smaller than the real width calculated from the pulmonary arteriogram. In normal cases, it is usually difficult to detect the pulmonary trunk by M-mode echocardiographic scanning.

**Figure 2.** Left lateral view of pulmonary arteriogram reveals that width of main trunk (arrow) is about 9 cm.

**Figure 3.** Upper panel shows depth of pulmonic valve (PV) from chest wall and fine systolic fluttering of valve. Lower panel shows that M-mode echocardiographic scanning from valve to pulmonary trunk (PT) demonstrates gradually increasing width of lumen (arrow). Left atrium (LA) is detected under pulmonary root.
ly (two to six months), and some progressed to the usual chronic progressive type of lesions.

Our patient differs from those findings usually associated with chronic cavitary pulmonary histoplasmosis. There was an acute illness, with the rapid development of cavitation within one to four months. There was rather rapid closure of the cavity with therapy.

In contrast, the more frequent form of pulmonary cavitary histoplasmosis has a more insidious progression, developing over months to years, is usually apical and often bilateral, and is usually associated with definite and marked fibrosis. This form is usually classified as chronic. Without therapy, there is a high mortality; even with therapy a rate of relapse of 5 percent or greater may be expected.

Acute cavitary histoplasmosis may indeed be an early phase of chronic progressive pulmonary histoplasmosis, as suggested by Goodwin et al. If so, one must consider the role of reinfection, whether endogenous or exogenous, as reviewed and summarized by Schwarz and by Tosh. Taking another viewpoint, can cavitation be a manifestation of primary histoplasmosis? We believe this case to be highly suggestive of this.

Clinically, it is important to differentiate these two forms of cavitary histoplasmosis. The chronic form has been well established, and its natural history is known. The acute form is less well known. It remains to be determined whether this form is "benign" or whether it has the same implications pathogenically and prognostically as chronic cavitary pulmonary histoplasmosis.

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