Thrombosed Giant Left Atrium Mimicking a Mediastinal Tumor*

David Rimon, M.D.; Leon Cohen, M.D.; and Joseph Rosenfield, M.D.

A patient with rheumatic heart disease, mitral stenosis, and mitral insufficiency is described. The thrombosed giant left atrium paralyzed the left vocal cord and completely obstructed the bronchi to the middle and lower lobes of the right lung. The giant left atrium mimicked a mediastinal tumor on the chest x-ray film.

A huge left atrium is a rare finding in rheumatic mitral disease. The reported incidence is 0.2 percent of patients with rheumatic heart disease.1 Most of the cases have combined mitral disease with predominant insufficiency.1,3 Only in a minority of patients is pure insufficiency5,4 or pure stenosis6,7 of the mitral valve found. Most of the cases show a conspicuous elevation of the left main bronchus and a wide angle of the carina on the x-ray films. Different grades of pressure can be seen on the main bronchi.6,8 Only very rarely do complete bronchial obstruction with atelectasis been reported. A more common finding is atelectasis caused by

*From the Department of Medicine C, Beilinson Medical Center, Petach Tikva, Israel, and Tel Aviv University Medical School, Tel Aviv, Israel.
Reprint requests: Dr. Leon Cohen, Department of Medicine C, Beilinson Medical Center, Petah Tikva, Israel

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FIGURE 1. Chest x-ray film showing enlarged heart, bilateral pleural effusion, congested lungs, and enlarged hilum.

Direct pressure of the enlarged heart on the lungs.

A patient with a giant left atrium is described, in whom paralysis of the left vocal cord and obstruction of the main bronchi of the middle and lower lobes of the right lung were found. These findings, because of their rarity, can lead to the erroneous diagnosis of a mediastinal tumor.

CASE REPORT

A 61-year-old man was hospitalized because of dyspnea, productive cough, and chest pain. He was a heavy smoker and suffered from chronic cough.

FIGURE 2. Tomogram showing obstruction of bronchi to middle and lower lobes of right lung.

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Twelve years previously, a heart murmur, irregular pulse, and congestive heart failure were found. A year before the present hospitalization, progressive hoarseness appeared. Physical examination revealed a man in a bad nutritional state, with hoarseness, cyanosis of the lips, dyspnea, and orthopnea. The chest was emphysematomous. The respiratory sounds were well heard, even with heavy breathing. The blood pressure was 120/85 mm Hg. The pulse was completely irregular, with an average rate of 100 beats per minute. An uplift was found at the cardiac apex and at the right ventricle, with retraction in between the two. A pansystolic murmur was heard at the apex and lower sternum. The anterior lower part of the right side of the chest was flat on percussion. The liver was tender and was palpable 4 cm under the right costal margin. The electrocardiogram revealed atrial fibrillation. Cultures of the sputum grew *Pseudomonas aeruginosa* and *Escherichia coli*; no malignant cells were found.

On the x-ray films of the chest, congested lungs, enlarged hilar, and a bilateral pleural effusion were seen. The vascularization at the periphery of the lungs was poor. The cardiac silhouette was enormous (Fig 1). Tomograms of the right lung revealed a wide angle of the carina. The bronchus to the middle lobes narrowed progressively and was obstructed at its middle portion. The bronchus to the lower lobe was obstructed at its proximal part. Both of the obstructions seemed to be due to a mass in the mediastinum (Fig 2). On the upper gastrointestinal series, the lower esophagus and the fundus of the stomach were displaced by a space-occupying lesion on the right side.

The patient was treated with digoxin, furosemide, spironolactone (Aldactone), and ampicillin. On the third day, purpura appeared on the gluteal surface and the extensor surfaces of the limbs, accompanied by arthralgia and rectal bleeding. The blood urea level rose from 60 mg/100 ml to 200 mg/ml (normal, ≤ 40 mg/100 ml). A Henoch-Schölein syndrome was diagnosed. The therapy with ampicillin was discontinued, and treatment with hydrocortisone was started. The congestive heart failure progressively worsened, and despite increasing doses of furosemide, the patient died in a state of pulmonary edema.

At the postmortem examination, the heart was extremely enlarged and occupied most of the thoracic cage. After the pericardium was opened, the right atrium and both ventricles were seen. The left atrium could not be seen from this anterior view of the heart. Only after lifting the heart could the enormous left atrium be seen. This giant left atrium reached dimensions of 15 × 15 × 20 cm. The whole heart was rotated posteriorly and to the right and pressed the main bronchus of the right lung. The heart weighed 1.3 kg. The left atrium was almost completely filled with a mural thrombus (Fig 3). The mitral valve showed both stenosis and insufficiency. Hypertrophy and enlargement of the right atrium and both ventricles, with a relative tricuspid insufficiency, were observed. No tumor was found in the mediastinum. The kidney showed focal segmental necrotizing glomerulonephritis.

**DISCUSSION**

The differential diagnosis in this case was between rheumatic heart disease with mitral insufficiency with a giant left atrium and a mediastinal tumor which paralyzed the left recurrent laryngeal nerve, obstructed the lower and middle bronchi of the right lung, and displaced the lower esophagus and fundus of the stomach. Paralysis of the left vocal cord in patients with a giant left atrium is very rare. Only one out of ten patients described by DeSantis et al. and one out of 15 patients described by Daley and Franks had hoarseness.

Complete bronchial obstruction by a giant left atrium is even rarer. Daley and Franks described a patient with complete obstruction of the bronchus to the right middle lobe. This finding was proved by bronchoscopic and bronchographic studies. Paralysis of the left vocal cord and bronchial obstruction caused by a giant left atrium have never been described in the same patient.

Only mitral insufficiency was diagnosed before death; however, the autopsy revealed the presence of severe mitral stenosis. Surawicz and Nierenberg described four patients with mitral stenosis and a large thrombus in the left atrium, which were not diagnosed ante mortem because no diastolic murmur was heard; in two of their cases, even the opening snap was absent. The big thrombus in the left atrium of our patient could have been one of the reasons for the disappearance of the characteristic auscultatory findings of mitral stenosis. There is no doubt that echocardiographic studies would have been of value in making the correct diagnosis. Unfortunately, no echocardiograph was available at that time.

The absence of the clinical findings of mitral stenosis, the absence of thromboembolic phenomena, syncope, and changing murmurs did not permit us to diagnose the presence of a thrombus in the left atrium. No doubt, the right heart failure and the massive left atrial thrombosis present in our patient contributed to the pleural effusion. It is to be emphasized that the huge atrial thrombus so filled the enlarged left atrium that it was impossible to differentiate it from a mediastinal tumor.

The case described suggests that in the differential...
Chylopericardium, either isolated or in combination with chylothorax is an unusual clinical entity. The first report was by Hasebrock in 1888 and showed 20 ml of chylous fluid in the pericardium of a patient at autopsy. Since that time, there have been several case reports of isolated, primary, and secondary chylopericardium.

Recently, we have encountered a case of chylopericardium after open heart surgery and aortic valve replacement. To our knowledge, this has not been previously reported. The case is presented, and possible mechanisms, methods of diagnosis, and plans of treatment for this rare problem are discussed.

CASE REPORT

A 53-year-old white woman had a history of rheumatic fever at the age of seven years. She developed dyspnea and swelling of the ankles in 1972. Cardiac catheterization revealed significant aortic insufficiency and stenosis. In September 1973, the patient underwent open heart surgery with prosthetic aortic valve replacement. Bleeding from the pericardial cavity necessitated reexploration of the mediastinum and pericardial space. All of the clots were removed, and no tamponade was seen. On the same day a central venous catheter was inserted by way of the left subclavian vein. The patient was discharged on Oct 12, 1973 on a regimen of digoxin (Lanoxin), warfarin (Coumadin), and ferrous sulfate heptahydrate (Fesol).

The patient was readmitted five weeks later because of recurrent chest pain and increasing enlargement of the cardiac silhouette on the chest x-ray film. Her vital signs were normal. No pulsus paradoxus was detected. Hepatojugular reflux was present. There was an apical systolic murmur (grade 3/6), and a two-component pericardial friction rub was heard. The liver was 3 cm enlarged. Bilateral (+1) pretibial edema was present.

Isolated Massive Chylopericardium*

Complication of Open Heart Surgery for Aortic Valve Replacement

Emin Kansu, M.D.;** William Fraimow, M.D., F.C.C.P.;† and Stanton N. Smullens, M.D.;‡

Chylopericardium following open-heart surgery for aortic valve replacement in a 53-year-old woman is described. Five weeks after surgery, the chylosus pericardial effusion was detected when the patient developed recurrent chest pain and cardiomegaly. Treatment included drainage of the fluid and partial pericardietomy. No recurrence of the chylopericardium was observed in this patient up to 14 months after surgery.

*From the Departments of Medicine and Cardiovascular Surgery, Thomas Jefferson University, Philadelphia
**Clinical and Research Fellow in Hematology.
†Associate Professor of Medicine.
‡Assistant Professor of Surgery.
Reprint requests: Dr. Fraimow, 1025 Walnut Street, Philadelphia 19107

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Figure 1. Chest x-ray film taken on admission (Nov 27, 1973), showing enlargement of cardiac silhouette due to pericardial effusion. Pulmonary parenchyma is free of active disease.