Obliterating Left Ventricular Mural Thrombosis

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Herein is reported a case in which a left ventricular mural thrombus was so large as almost completely to obliterate the left ventricle. Such an occurrence, to our knowledge, has never been reported.

Case Report

A 64-year-old white man, blind since birth, considered himself well until March 1960. Because of progressive weakness, shortness of breath upon effort, and swelling of the ankles, he entered Episcopal Hospital on June 13, 1960. Cardiac therapy was intensified and left thoracentesis was done on several occasions. The fluid was serosanguineous in character. Pleural biopsy, with a Vim-Silverman needle, disclosed only fibrous tissue. He was discharged on July 18, 1960.

Shortly thereafter, shortness of breath again increased, and ankle edema and left pleural effusion reappeared. He was re-admitted to Episcopal Hospital on August 17, 1960. Findings were similar to those previously observed.

The blood pressure was 100/80 mm. Hg. The temperature was normal. There was bilateral optic atrophy. The jugular veins were slightly distended. The cardiac impulse was not palpable. Heart sounds were distant and normal in quality, rate, and rhythm. There were no murmurs. Signs of left pleural effusion were present. The abdomen was negative; the liver and spleen were not felt. There was moderate pitting edema of the legs and feet.

The hemoglobin and electrolytes were normal, the serologic tests for syphilis were negative, and the serum glutamic oxaloacetic transaminase was normal.

Roentgenogram of the chest (fig. 1) revealed increased size of the cardiac silhouette, involving mainly the left ventricular salient. The upper border of the left ventricle tended to be concave and bulged laterally into the density produced by a left pleural effusion. On fluoroscopy, following removal of most of the pleural fluid, no pulsation could be seen over the left ventricle. There were faint pulsations of the other borders of the cardiac silhouette. There was no change in the cardiac silhouette with either the Mueller or Valsalva maneuver. There was no intracardiac calcification. The pulmonary vascular markings were slightly increased. The findings were interpreted as consistent with pulmonary congestion, left pleural effusion, and enlargement of the heart predominantly of the left ventricle, which was probably aneurysmal and possibly associated with mural thrombosis. Additional pericardial disease could not be excluded.

The electrocardiograms (fig. 2) were interpreted, in the absence of change, as consistent with extensive anterior myocardial fibrosis, probable left ventricular aneurysm, left atrial enlargement, and digitalis effect.

Left thoracentesis on several occasions yielded serosanguineous and later dark amber fluid. The underlying lung appeared compressed but was otherwise not remarkable.

Dyspnea and edema of the lower extremities gradually lessened and the patient lost 13 pounds. On September 4, 1960, while resting in bed, he suddenly became taehynpeic, pale, and cyanotic, and died.

Autopsy Findings

The right pleural cavity was obliterated by a chronic fibrous pleuritis. The left pleural cavity contained 600 ml. of fluid, dark amber in color. The visceral pleura overlying the left lower lobe was thickened, and the underlying lung was firm, contracted, and congested. Microscopically the lung showed marked vascular congestion with extravasation of erythrocytes into the alveoli, which were edematous and contained "heart failure" cells. The pulmonary vessels were patent. There was some thickening of the walls of the smaller branches of the pulmonary artery.

The heart weighed 610 Gm. The pericardium was thickened and fused anteriorly with the epicardium of the left ventricle. The left atrium was slightly enlarged. The entire anterior wall of the left ventricle was thin and fibrotic, and tended to bulge somewhat. The walls of the remaining portion of the left ventricle were reduced in thickness.

Arising from the anterior wall of the left ventricle and extending into and almost obliterating the left ventricle was a large, firm, fibrotic, and layered thrombus. The diameter of the thrombus was 10 cm. The capacity of the remaining left ventricular cavity was estimated to be approximately 30 ml. Microscopically there was extensive myocardial fibrosis but no evidence of acute myocardial infarction. The thrombus itself was fibrotic, and its union with the myocardium was indistinguishable.

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No other thrombi were found. The cardiac valves were normal. The coronary arteries exhibited a moderate degree of generalized narrowing and sclerosis, and the anterior branch of the left coronary artery was completely occluded by an organized atheroma, 5 ml. in length, beginning 2.5 cm. from the ostium. The entire aorta showed mild atherosclerotic changes.

The liver weighed 1,100 Gm. It was nutmeg in appearance and revealed dilatation and congestion of the central veins and adjacent sinusoids. The surrounding hepatic cells showed degenerative change. The spleen was firm and weighed 120 Gm. There was marked congestion and a slight increase in fibrous tissue.

Sections of the heart, liver, and spleen are shown in figure 3.

Discussion

It was apparent from the chest roentgenogram that there was an aneurysm of the left ventricle.¹ The basis for the aneurysm was considered clinically to be myocardial fibrosis secondary to occlusive coronary artery disease, the absence of pain notwithstanding. Indeed, many cases of cardiac aneurysm have been reported to sustain a silent myocardial infarction and to present with congestive heart failure of insidious onset.² Interestingly, however, although the patient steadfastly denied having any pain, his family, following his death, related, in response to direct questioning, that he had had bouts of substernal pain for several months prior to his demise. Curiously, it was his wish that no physician should be informed of this distress because he feared hospitalization.

The aneurysm displayed no motion. A cardiac impulse was not palpable, and the left ventricular portion of the cardiac silhouette was seen by fluoroscopy not to pulsate. The absence of pulsation was thought to be due to intrinsic myocardial disease and possibly to mural thrombosis and even pericardial restriction, which frequently occur in left ventricular aneurysms arising from occlusive coronary artery disease.³-⁴ Unfortunately, his condition did not permit study of the heart by contrast roentgenography, by which diagnostic conclusions might have been possible.⁵-⁷

At autopsy, the entire anterior wall of the left ventricle was reduced to a fibrous shell. Arising from it and almost filling the left ventricle was a huge, organized, and layered thrombus.

We have been unable to find in the literature a similar occurrence, although in the case reported recently by Dexter, Lawton, and Raynolds,⁸ the left ventricular mural thrombus may possibly have been as large. Their case had an aneurysm, 18 cm. in diameter, described as being completely filled with a mural thrombus. Another might be the case reported by Shennan and Niven.⁹ In both of these cases, although it is not clear if the thrombus was restricted to the aneurysm or filled the ventricular cavity, illustrations show, indeed, a huge thrombus.

Such an obliterating thrombus of the left ventricle would be anticipated to produce severe restriction of inflow similar to that
occurring in mitral stenosis. Indeed, the electrocardiogram showed signs consistent with left atrial enlargement, which was found at necropsy, and the pulmonary arterioles showed changes consistent with pulmonary hypertension.

Forward flow from the left ventricle would also be expected to be seriously compromised. The fatigue, pallor, and low blood pressure bespoke a low cardiac output.

Similar findings occur, of course, in many other conditions, including left ventricular aneurysm alone. In the presence of left ventricular aneurysm, a clue to the existence of mural thrombosis is throembolism, which occurs in almost two thirds of the cases. It is not always present, however, and it may also occur in the absence of clinical manifestations and in the absence of mural thrombosis.

Another clue to the presence of associated mural thrombosis is absence of pulsations of the left ventricular aneurysm. Pulsations of a left ventricular aneurysm are characteristically paradoxical. The involved wall of the left ventricle is fibrous and bulges with each ventricular systole. The presence of a mural thrombus would be anticipated to dampen such an outward thrust. The reliability of this sign in diagnosing associated mural thrombosis is questionable, however, for other conditions, like dilatation or pericardial effusion, may produce similar dampening. Also, to our knowledge, there has been no systematic study made of the pulsations of left ventricular aneurysm alone compared with those associated with mural thrombosis. Certain it is that pulsations do not exclude mural thrombosis. Other roentgenographic signs suggestive of left ventricular mural thrombosis include increased density and even calcification, but these are very rare.

For these reasons, clinical recognition is difficult. Nevertheless, knowledge of its occurrence can lead to a strong clinical suspicion of its presence which perhaps may be established with certainty by contrast roentgenography.

Summary and Conclusions

A case is recorded of a huge mural thrombus that encroached upon and almost obliterated the left ventricle. There was underlying occlusive coronary artery disease with extensive myocardial fibrosis and aneurysm formation. Clinical manifestations, other than

**Figure 2**

Electrocardiogram showing sinus rhythm and findings consistent with left atrial enlargement, extensive anterior myocardial fibrosis, probable aneurysm, and digitalis effect.

**Figure 3**

Gross sections of the liver (upper left), spleen (upper right), and heart viewed from above. There was chronic passive congestion of the liver and spleen, which were firm. In the section of heart, note the thin fibrous residue of the anterior wall and the huge, fibrous, layered thrombus arising from it and almost obliterating the left ventricle. The remaining cavity has been increased by distortion for photographic purposes. The subjacent muscle is the septal portion of the left ventricle.
those produced by a persistent left pleural effusion, were similar in effect to any condition producing severe restriction of left ventricular inflow and outflow. A clue to its presence was absence of left ventricular pulsations.

References

The very ingenious Dr. Hales writes me, that, having many years since, tied a ligature about a frog's neck, to prevent any effusion of blood, he cut off its head, and, thirty hours after, observed the blood circulating freely in the web of the foot: the frog also at this time moved its body when stimulated: but, on thrusting a needle down through the spinal marrow, the animal was strongly convulsed, and, immediately after, became motionless.—Robert Whytt. Physiological Essays. Edinburgh, 1755, p. 178.
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