Massive Thrombosis of the Left Ventricle

Case Report

By Ruben L. Shapiro, M.D., and Renzo G. Olivetti, M.D.

The purpose of this report is to describe the occurrence of a massive, organized thrombus in the left ventricle. The few similar cases described in the literature and reviewed by Zatuchni and Tan deal with thrombi associated with cardiac aneurysms. In our case the heart was enlarged and hypertrophied, and the site of extensive myocardial fibrosis due to coronary atheromatosis, but no aneurysm was demonstrated at autopsy. The space-occupying lesion in the ventricle had produced an increase in left atrial pressure with the functional effects of mitral stenosis and pulmonary hypertension. It is of interest that this patient was on anticoagulant drugs for 6 years prior to death.

Case Report

J.Z., a 43-year-old white linotype operator, was admitted to the Newington Veterans Hospital for the thirteenth time on December 12, 1962, complaining of anterior chest pain of 3 days' duration. He had been well until 1953, when he was found to have hypertension. He entered this hospital for the first time in January 1956, with a blood pressure of 210/120, grade-III hypertensive retinopathy, and increasing angina pectoris. Three months later he returned with a posterior wall myocardial infarction and was started on long-term anticoagulation.

The patient was readmitted again for three documented myocardial infarctions in February 1957, October 1959, and December 1959. After the last infarction, he developed left heart failure, controlled with digitalis. His course was complicated by thrombophlebitis of the left saphenous system, and repeated pulmonary emboli and infarctions of his lungs, for which the inferior vena cava was ligated. By July 1961 his blood pressure was normal, but the degree of failure continued to increase in spite of all therapeutic measures.

Oral anticoagulants were given from the first infarction in 1956 until the present admission in December 1962. A review of the available records of the prothrombin levels (fig. 1) shows that many values were out of the therapeutic range. Physical examinations on all 12 previous admissions had revealed progressive enlargement of the heart, dampening of heart tones, and persistent sinus tachycardia but no murmurs or thrills.

The physical examination at the final admission revealed a thin, slightly cyanotic, restless white man in acute distress with dyspnea and chest pain. The blood pressure was 118/100, the pulse was 120 and regular, and respirations were 34 per minute. Neck veins were engorged and filled from below. A few moist rales were heard at both bases, but the lungs were otherwise clear. The heart was greatly enlarged with the left border of cardiac dullness extending out to the anterior axillary line in the fifth intercostal space. Heart sounds were weak and there were no murmurs or thrills. The liver was enlarged and tender, extending five fingerbreadths beneath the right costal margin. There was 4+ pitting edema of both ankles extending up to the midcalf.

The admission prothrombin level was 62 per cent. The dosage of Panwarfin was adjusted thereafter to maintain prothrombin levels between 20 and 30 per cent. Chest x-ray demonstrated marked enlargement of the heart, especially the left ventricle. The lung fields were clear with no engorgement of the pulmonary vessels. An electrocardiogram revealed low voltage, rate of 120, residuals of the old anterior and posterior myocardial infarctions, and digitalis effect. The diagnosis on admission was myocardial ischemia with a possible new infarction and severe intractable cardiac failure. Morphine sulfate rapidly relieved the chest pain. The patient appeared comfortable for the next 2 days, but on the third hospital day he began to complain of increasing shortness of breath while at bed rest. The heart rate rose to 140, and hypotension developed. He failed to respond to vasopressors, oxygen, and sedation, and died on the fourth hospital day.

Autopsy Findings

The heart was enlarged and hypertrophied and weighed 590 Gm. The right ventricle was mod-

From the Cardiovascular Service and the Department of Pathology, Newington Veterans Hospital, Newington, Connecticut.
erately enlarged and measured 0.4 cm. in thickness midway between the valvular ring and apex. The left atrium was markedly dilated with smooth endocardium. The foramen ovale was closed. The left ventricle was tense and voluminous, but no circumscribed outpouchings were noted. On palpation there was an increase in consistency so that the left ventricle felt "sarcomatous."

The open heart revealed almost complete obliteration of the left ventricular cavity, which was occupied by an organized, pink-gray thrombus. This mass could not be detached from the light gray nacreous layer that represented the fibrosed parietal endocardium. The posterior papillary muscle was partially encased in the thrombus (fig. 2), which was constituted by a series of light gray concentric layers. No central canalization was observed and only a small (25 to 30 ml.) residue of the ventricular cavity just beneath the aortic and mitral leaflets could still be recognized. The myocardium exhibited extensive areas of fibrosis, involving the anterior and posterior left ventricular walls and the lower two thirds of the interventricular septum. No zones of recent myocardial necrosis were seen, no calcification was noted, and no alterations of valvular leaflets were encountered. Both coronary arteries and their major branches showed pronounced atheromatous changes. There was an occlusion of the anterior descending branch of the left coronary artery, and also of the main stem of the right coronary, 1.5 cm. from its origin.

Microscopically there was extensive fibrosis of the myocardium with replacement of the muscle by adult connective tissue. No recent infarcts were observed. Determinations for calcium from several sections of the myocardium and the thrombus were negative. It was not easy to differentiate the thickened endocardium and the fibrotic subendocardial muscle fibers from the adjacent organized thrombus. The latter was composed of dense collagen that was traversed haphazardly by vascularized bridges (fig. 3). These contained inflammatory cells with a predominance of plasma cells. Sections from the center of the thrombus revealed hyaline necrosis.

Several sections from the coronary arteries presented sclerotic occlusion and thrombosis with partial recanalization. The intima was irregularly thickened and the media was thinned owing to advanced atheromatosis. The septal branch of the left anterior descending artery was filled with a recent thrombus, and small hemorrhages were seen in the sclerotic intima of the vessels.

The aorta and large vessels exhibited moderate atheromatous changes. The lungs showed brown induration and chronic passive congestion. There were embioli in the spleen and right kidney and marked passive congestion in the liver and spleen.

**Discussion**

This case report was considered of interest for three reasons. Most of the left ventricle was occupied with a thrombus that did

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**Figure 1**

*Forty random prothrombin levels from October 1959 to December 1962.*

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**Figure 2**

*Section through the fibrosed wall of the left ventricle and through the laminated thrombus that occupies its cavity.*

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not allow adequate filling and caused intractable cardiac failure. Only a few cases of these large thrombi have been reported, and usually such hearts had aneurysms with the thrombi extending from them. This thrombus accumulated during prolonged anticoagulant therapy.

Bean\(^2\) was the first to present a case in which a massive antemortem thrombus had almost filled the entire left ventricular cavity. Since the introduction of anticoagulant therapy in the treatment of acute myocardial infarction, the incidence and size of mural thromboses have been significantly reduced.\(^3\)\(^4\)

Zatuchni and Tan\(^1\) described a patient whose left ventricle was so massively filled with an organized thrombus that the remaining cavity was estimated to be about 30 ml. The thrombus had originated from an aneurysm of the left ventricle, visualized by chest x-ray prior to death. This association is to be expected because the endocardial surface of a ventricular outpouching is frequently the seat of thrombosis. More interesting is the report of Adams and associates\(^5\) in which a large mural thrombus filling two thirds of the left ventricle was found. The patient was a 62-year-old man with “chronic myocarditis.” The heart (750 Gm.) demonstrated enlargement and hypertrophy of all chambers. The coronary arteries were “widely patent” and only “minimally” sclerotic, but an old healed posterior myocardial infarction was noted. Although the cause of death appeared to be massive pulmonary infarction (there were also multiple small thrombi in the right atrium and ventricle), the author states that the large left ventricular mural thrombus had impaired the cardiac output and contributed significantly to the terminal episode.

Our patient was a 43-year-old man who had been hypertensive for 9 years. He had suffered at least four myocardial infarctions, and the heart had demonstrated progressive enlargement without any x-ray evidence of aneurysm or thrombosis. Unless an aneurysm is present, the diagnosis of a thrombus within the left ventricle on routine chest x-ray is rarely made.\(^6\) When suspected, further diagnostic aids might include angiography, fluoroscopy, tomography, and cineangiography.

The natural history of large mural thrombi reveals that some remain in place even after the adjacent infarcted area has healed. They may undergo gradual central lysis due to proteolytic enzymatic action, and they may become covered by endothelium. Other mural thrombi are gradually organized, especially if a large part of the underlying myocardium survives. Bean\(^2\) described cases with mural thrombi that were adherent to 3-year-old scars of ventricular infarctions.

In our case the thrombus had continued to grow by successive accretions of collagen, and eventually encroached upon most of the left ventricular cavity. An ingrowth of newly formed capillaries was noted in several portions of the thrombus. This granulation tissue was rich in plasma cells and was found in all sections taken from the mass at different levels. The central core revealed complete hyalinization. It was not possible to identify histologically or clinically the infarction from which the thrombus originated, and we cannot discard a priori the hypothesis that the mass represented the coalescence of two (or more) mural thrombi, because of definite electrocardiographic evidence of septal, anterior, and posterior wall infarcts. From an

**Figure 3**

*Young granulation tissue with inflammatory cells (chiefly plasma cells) invading the thrombus. (Hematoxylin and eosin stain, \(×\) 100.)*

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anatomic viewpoint this assumption cannot be substantiated because of the lack of any central canal or endothelialized space in the thrombus and because of the uniform histologic appearance of the mass.

No conclusion as to the age of the thrombus can be obtained from the pathology findings or the clinical records, but we may consider the summer of 1961, 17 months prior to death, the time in which the thrombus had become so huge as to interfere seriously with ventricular inflow. Signs of right heart failure were first observed at that time by the family physician and soon afterwards enlarged liver, distended neck veins, cyanosis of the lips, basilar rales, and dyspnea were noted.

It is worthwhile to mention that the gross and histologic appearance of the lungs was that of impaired venous flow and increase of pulmonary capillary and arterial pressure.\textsuperscript{7-9} We can therefore accept the concept of a growing thrombus functionally obliterating the left ventricular cavity and causing gradual increase of left atrial pressure with secondary pulmonary vascular changes. Important here is the extremely low pulse pressure (118/ 100) observed during the last hospitalization. The mass had significantly compromised the forward flow of blood from the left ventricle.

The cardiac thrombus in our case and the ventricular thrombi originating from an aneurysmal sac probably have the same pathogenesis because the \textit{primum movens} of both lesions is represented by a myocardial infarction. Secondary dilatation of the infarcted wall with noncontractility and stasis is a very important mechanical factor. Other conditions, such as alternating states of hypocoagulability and hypercoagulability\textsuperscript{10} in patients on anticoagulants, may also play some role in the formation of massive intracardiac thrombi.

\textbf{Summary}

A case of massive thrombosis of the left ventricle is reported. The thrombus had caused a pronounced restriction of the ventricular inflow with the signs and symptoms of “functional” mitral stenosis.

\textbf{References}

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