Surgical Removal of an Intracavitary Left Ventricular Myxoma

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PRIMARY intracardiac tumors of the heart are rare. The commonest primary tumor is myxoma, found almost exclusively in the atria, 75 or more occurring in the left atrium. Mahaim1 reported 200 primary cardiac tumors in 1945, from the world literature up to that time. Strauss and Merliss2 reviewed 480,331 autopsies from the Los Angeles County Hospital and were able to report 8 cases of myxoma occurring in the left atrium.

Myxoma originating in the ventricles appears to be extremely rare. A well-documented pathologic study of a left ventricular myxoma with embolic occlusion of the abdominal aorta and renal arteries was published by Young and Hunter3 in 1957. The patient was a 10-year-old girl diagnosed as having rheumatic heart disease because of her cardiac disability and a rough mitral systolic murmur transmitted to the axilla and over the precordium. At autopsy a tumor was found practically filling the left ventricle and extending up to the aortic valve. The tumor was jelly-like, with multiple polypoid extensions. The tumor was attached to the endocardium below the insertion of the posterior papillary muscle of the mitral valve. We have found no report of a similar tumor in the recent literature.

The following is a case report of an intracavitary left ventricular myxomatous tumor, which was removed by the left transventricular route with the aid of cardiopulmonary bypass. This is apparently the first such tumor removed from the left ventricle.

Case Report

In October 1942, at the age of 15, the patient developed an embolus at the bifurcation of the aorta, and the following month the left leg was amputated below the knee. Thereafter, she had 3 children without incident. In April 1950, at the age of 23, she had a cerebral embolus with a left hemiplegia and shortly thereafter gave birth to her fourth child. The hemiplegia gradually improved until there was no residual disability. During this period of time there were multiple peripheral emboli despite long-term anticoagulant therapy with Dicumarol. From August 1955 to April 1959, 4 aortic emboli occurred that required surgical removal.* In 1956 a left thoracotomy was performed and at that time a multilobulated tumor was palpated in the left ventricular cavity. The mitral valve was normal to palpation.

The patient entered the St. Vincent's Hospital on February 10, 1959. There was a left ventricular thrill at the apex that had a peculiar double quality. There was also a palpable systolic click. A loud midsystolic sound was present that varied irregularly in intensity. No sounds or murmurs were heard in diastole. The second sound was normally split but not accentuated (fig. 1). The blood pressure in the left arm was 125/70. The right radial pulse was markedly diminished and both femoral pulsations were diminished. The lungs were clear, the liver was not enlarged, there was no edema, the venous pressure was not elevated, and no abnormal venous pulsations were detected.

The blood and urine examinations were normal. X-ray showed an unusual contour with prominence of the left ventricle and pulmonary artery. There was no evidence of enlargement of the left atrium. There was an adhesion to the pericardium, just above a segment along the left border, in the

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*By Alfred Wallner, M.D., of Kalispell, Mont.
region of the left ventricle, which had been noted on fluoroscopy to be noncontractile (fig. 2). An electrocardiogram revealed sinus rhythm at a rate of 75 per minute with evidence of left ventricular hypertrophy and digitalis effect (fig. 3).

Surgery was performed with the use of the Kay-Anderson heart-lung machine. A median sternotomy incision was made. The pericardial sac was opened. The heart was adherent to the pericardial sac due to prior cardiace exploration. The adhesions were freed by blunt and sharp dissection. Tapes were placed around the superior and inferior venae cavae. The left ulnar artery was isolated immediately distal to its origin from the brachial artery. The left femoral artery was exposed immediately distal to Poupart's ligament. The patient was given 3 mg. of heparin per Kg. of body weight. A catheter was inserted in the left ulnar artery for measuring pressures. A second catheter was inserted into the left femoral artery to return blood to the patient. The catheters inserted into the left ulnar artery and the left femoral artery met obstructions, which were thought to be due to previous emboli. The right atrial appendage was opened. No abnormality was palpated in the right atrium or the tricuspid valve. A catheter was inserted into the inferior vena cava through the incision in the right atrium and also another catheter was passed into the superior vena cava through the right atrial appendage. The patient was placed on cardiopulmonary bypass, the aorta was cross clamped approximately 5 cm. above the origin of the coronary arteries, and the heart was stopped by anoxia. The left ventricle was then opened wide. A very large tumor, approximately 7 cm. in diameter, was found to occupy the entire cavity. This tumor had many villous projections and apparently was arising from the lowermost portion of the ventricular septum as well as from the anterolateral portion of the wall of the left ventricle. Villous projections of the tumor protruded into the mitral valve as well as up into the aortic valve (fig. 4). The tumor was excised from its attachment to the septum and to the anterolateral wall of the left ventricle, a portion of the normal wall of the left ventricle being removed including 3 chordae tendineae in the midportion of the aortic leaflet of the mitral valve (fig. 5). To avoid incompetence of the valve, it was repaired with interrupted silk stitches (fig. 6). After the left ventricle was closed, ventricular fibrillation developed that was stopped with a countershock of 200 volts at 0.1 second. The cardiopulmonary bypass was removed after a total duration of 54 minutes. After 5 minutes the blood pressure fell to 30 or 40 mm. and a grade III or IV systolic thrill was felt in the left atrium. The cardiopulmonary bypass was begun and the left ventricular incision was reopened. The previously placed stitch in the aortic leaflet of the mitral valve was removed; this time the lateral third of the aortic leaflet of the mitral valve was sutured to the lateral third of the mural leaflet and this in turn to the lateral papillary muscle (fig. 6). This procedure narrowed the orifice of the mitral valve very slightly, if at all, but appeared to eliminate completely the mitral insufficiency. The left ventricle was again closed, the clamp was removed from the aorta, and the heart again developed ventricular fibrillation, which

![Fig. 1. Low-frequency phonocardiogram from the apex. A, atrial sound; 1, first sound; x, loud mid-systolic sound; 2, second heart sound; 3, third heart sound.](image)

![Fig. 2. X-ray showing unusual contour with prominence of left ventricle and pulmonary artery.](image)
Fig. 3. Preoperative electrocardiogram showing sinus rhythm at rate of 75 per minute with evidence of left ventricular hypertrophy and digitalis effect.

Fig. 4. Drawing of myxoma of left ventricle.
was stopped again with one electric shock of 220 volts at 0.1 second. The cardiopulmonary bypass was disconnected again after a period of use of 29 minutes but after 5 to 8 minutes the heart began to beat poorly. Again the heart-lung machine was started to give the heart a period of rest, and this time was kept going for 38 minutes. Just before removal of the cardiopulmonary bypass 7 ml. of 1:10,000 epinephrine were injected into the right ventricular cavity. Despite previous digitalization, 0.8 mg. of lanatoside-C was also given. Again after 10 minutes the blood pressure began to fall. Three milliliters of 1:10,000 epinephrine were injected into the right ventricular cavity and the heart improved, whereupon the surgical closure was completed.

The tumor measured 7 by 6 by 3 cm., weighed 60 Gm. (Figs. 7 and 8), and had a volume of 58 ml. Its peripheral portion was composed of smooth polyloid projections varying in size from 7 by 1 by 1 cm. to 1 by 0.3 by 0.3 cm. and in color from pale gray-tan to brown and to dark red. The periphery of the tumor was soft and jelly-like in consistency; near the base there was a stellate area of calcification. The base of attachment measured 3 by 2 by 0.4 cm.; here the soft tumor tissue was adherent to thickened white endocardium beneath which were 2 to 3 mm. of brown myocardium. Microscopically, the bulk of the tumor consisted of an amorphous nonfibrillary matrix, which stained pale pink with eosin, pale blue-green with Masson trichrome, blue-pink with phosphotungstic acid hematoxylin, very pale blue-pink with periodie acid fuchsin, and did not stain with Best's carmine. It varied in density from an

![Fig. 5. Photograph of tumor.](image)

![Fig. 6 Top. Drawing of first repair of aortic leaflet of mitral valve. Middle and Bottom. Drawing of final repair of aortic leaflet of mitral valve.](image)
REMOVAL OF MYXOMA

Fig. 7 Left. Photomicrograph of myxoma of left ventricle. X 400.

Fig. 8 Middle. Photomicrograph showing sharp transition between tumor tissue and underlying endocardium. X 100.

Fig. 9 Right. Photomicrograph of tumor embolus. X 400.

There was a sharp transition between the tumor tissue and the underlying endocardium, which was several times normal thickness, due to increase in both fibrous and elastic tissue. Neither fibrous or elastic fibrils entered the tumor from the endocardium.

The staining reactions exclude the possibility that the tumor matrix had its origin from either fibrin or fibrous tissue. The absence of a well-vascularized fibrous base between the endocardium and the tumor tissue further excluded the possibility that the lesion was an organized thrombus. The underlying myocardium contained no indication of infarction, old or recent. The diagnosis was made of myxoma of left ventricular endocardium (figs. 8 and 9).

Review of the sections from the tissue removed during previous embolectomies revealed the same tissue as was present inside the left ventricle. It was definitely myxomatous tissue rather than thrombus (fig. 9).

On the second postoperative day a transient nodal rhythm appeared. An electrocardiogram taken 16 days postoperatively showed some ectopic nodal beats and deep symmetrically inverted T waves in leads V3 through V6, which were thought to be due to reaction around the incision of the left ventricle (fig. 10). An electrocardio-
gram taken 3 weeks later showed less marked T-wave changes.

Ten days after operation the apical impulse was less thrusting and without the previous double quality. The heart sounds were normal, the loud variable systolic click having disappeared. There was a grade II systolic murmur at the apex.

The patient was discharged from the hospital 21 days after the operation and she returned to Montana by car 1 week later.

ADDENDUM

For 2 months after surgery the patient has been leading the normal life of a housewife.

It is a common error to think that the more a doctor sees the greater his experience and the more he knows. No one ever drew a more skilful distinction than Cowper in his oft-quoted lines, which I am never tired of repeating in a medical audience:

Knowledge and wisdom, far from being one,
Have oft-times no connexion. Knowledge dwells
In heads replete with thoughts of other men;
Wisdom in minds attentive to their own.
Knowledge is proud that he has learned so much;
Wisdom is humble that he knows no more.—WILLIAM OSLER, M.D.

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