CASE REPORTS

MYXOMA OF THE LEFT ATRIUM

BY

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In the past five years four patients with an atrial myxoma have been operated upon at the Toronto General Hospital. The clinical diagnosis in the first three cases (Aldridge and Greenwood, 1960) was mitral stenosis. In the fourth case, admitted in October, 1960, a correct clinical diagnosis was made.

Case Report

A woman, 40 years old, with no history of rheumatic fever, was well until 1957 when she developed an illness lasting six weeks that was probably pneumonia: recovery was complete. Before admission, she had noted fatigue for two years, exertional dyspnoea for one year, and faintness on stooping for six months. There was no history of hæmoptysis, nocturnal dyspnoea, arrhythmia, or embolism.

The patient was in no distress. The skin was warm and without cyanosis. The pulse rate was 70 a minute, and was normal in volume and rhythm. The blood pressure was 110/60. The jugular venous pulse showed *a* and *v* waves of equal height, 2 cm. above the sternal angle with the patient at 30°. The first sound was palpable at the apex. There was a slight impulse palpable at the left sternal border. The second sound and left ventricle were not felt. The first sound was moderately loud while the second was normally split with slight accentuation of the pulmonary component. A grade II/IV rumbling mid-diastolic murmur without presystolic accentuation was localized near the lower left sternal border. A grade II/IV blowing, early and mid-systolic murmur was maximal in the third left interspace, and was conducted downwards along the sternal border. Both murmurs increased in intensity with normal expiration. No opening snap was heard. Change in position did not alter these findings.

Phonocardiography demonstrated the systolic murmur, beginning with the first sound, but ending before the second. The diastolic murmur was not well recorded. No opening snap was seen.

The lung fields were clear. There was no hepatomegaly or peripheral oedema.

On radiological examination, there was no enlargement of the heart shadow. The hilar pulsations were normal. Moderate enlargement of the left atrium produced a double shadow at the right heart border and a prominence of the left heart border. No calcium was seen in either mitral or aortic valve areas. Kerley's lines were present. The cardio-thoracic ratio was 41 per cent.

The electrocardiogram showed sinus rhythm, a P-R interval of 0.2 sec., a broad P wave in lead II, and S-T segment depression in keeping with digitalis therapy. The QRS complex did not suggest dominance of either ventricle.

Several urine samples were normal. The hæmoglobin was 11.7 per 100 ml., white cell count 12,300; E.S.R. 44 mm./first hour; total and differential serum proteins by electrophoresis were normal.

At right heart catheterization, the mean pulmonary "wedge" pressure was elevated to 26 mm. Hg, the mean pulmonary arterial pressure was 34 mm., and the mean right atrial pressure was 3 mm. The pulmonary "wedge" tracing showed a relatively high *v* wave and a rapid *y* descent (Fig. 1). The cardiac output by direct Fick principle was 4.5 l./min. The pulmonary vascular resistance was 143 dynes/sec./cm.⁻⁵ (1.8 units).

At angiocardiology, after 50 ml. of 76 per cent renografin had been injected into the main pulmonary artery, a large filling defect was outlined in the lateral view by a thin rim of radio-opaque dye seen in an enlarged left atrium (Fig. 2).

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FIG. 1.—Pressure tracings obtained at right heart catheterization show a raised pulmonary wedge pressure with a dominant *v* wave and a rapid *y* descent. The pulmonary arterial and right ventricular pressures reflect the mild passive pulmonary hypertension.

FIG. 2.—Angiocardiogram, showing in the lateral projection a large filling defect in the left atrium due to the myxoma.

FIG. 3.—Surgical specimen of left atrial myxoma.

Operation. On November 9., 1960, Dr. W. G. Bigelow operated using the extra-corporeal circulation. A large tumour was found filling the left atrium, arising by a short stalk from the inter-atrial wall. The tumour was removed in one piece. The post-operative course was uneventful, and physical examination at this time showed a normal first sound and no murmurs.

The tumour (Fig. 3) measured 6.5 cm. $\times$ 5 cm., and was ovoid in shape. The microscopic sections were typical of a myxoma.

Discussion

Clinically left atrial tumours are commonly confused with mitral stenosis. Two points in this patient's history raised the suspicion of the presence of a tumour. The relatively short duration of symptoms was somewhat unusual for mitral stenosis in the absence of arrhythmia or active myocardial disease. The presence of faintness on stooping suggested the possibility of intermittent obstruction, although this symptom could not be reproduced by postural changes in hospital.

During the natural history of mitral stenosis, certain complications may occur that modify the clinical findings and make the diagnosis difficult. The most important of these are a low cardiac output, gross enlargement of the right ventricle, associated valve lesions, and calcification of the mitral valve. The low cardiac output may be due to tachycardia, pulmonary hypertension, or an inadequate myocardium. In this patient the skin was warm and the pulse volume normal, indicating at least an adequate output. The heart rate was normal, and none of the clinical findings of pulmonary hypertension were present. The presence of sinus rhythm excluded an important myocardial factor (Fleming and Wood, 1959). A large right ventricle, which is always associated with congestive failure or tricuspid regurgitation, may displace the mitral valve posteriorly: no evidence of either of these conditions was present. The associated valve lesions that are most confusing are tricuspid valve disease and aortic regurgitation: the normal pulse and venous pressure excluded significant disease at these valves. Calcification of the mitral valve tends to depress the first heart sound and opening snap by interfering with valve movement: careful fluoroscopic examination, however, failed to reveal any intra-cardiac calcium.

This patient had a loud first sound but no opening snap. A loud first sound is commonly caused by some factor that keeps the mitral valve leaflets wide open until after the onset of ventricular systole. In mitral stenosis it is due to prolonged left ventricular filling as a result of the high end diastolic gradient across the mitral valve. In the presence of sinus rhythm, this gradient is increased by atrial contraction and results in a presystolic murmur, but no such murmur was present in this patient. The normal P-R interval excluded late left atrial contraction as a cause of the loud first sound. The opening snap is related to the high early diastolic gradient in the presence of diseased, though pliable, valve cusps: the lack of calcium on fluoroscopy made it unlikely, therefore, that a rigid valve was responsible for its absence.

Both the systolic and diastolic murmurs were accentuated by normal expiration suggesting that they originated on the left side of the heart. The systolic murmur began with the first sound and was blowing in quality. In spite of its atypical location and short duration, it was in keeping with trivial mitral regurgitation. The diastolic murmur was of greater significance in that it was maximal near the tricuspid area and did not have presystolic accentuation. This atypical location was difficult, if not impossible, to explain on the basis of mitral stenosis. As noted above, in the presence of sinus rhythm, the absence of presystolic accentuation was incompatible with this diagnosis.

Radiological and electrocardiographic findings did not aid in the differential diagnosis, but confirmed the presence of obstruction in the region of the mitral valve. The broad P waves suggested left atrial enlargement, and this could be seen on X-ray. The presence of Kerley's lines indicated elevation of pulmonary venous pressure. However, there was no evidence of either right or left ventricular enlargement or hypertrophy.

On the basis of these findings a diagnosis could be made of a lesion that (1) was causing obstruction in the region of the mitral valve resulting in left atrial enlargement and a rise of pulmonary venous pressure, (2) was resulting in the mitral valve being held open in early ventricular systole

without evidence of a high end diastolic gradient across the valve or a short P-R interval, (3) was causing distortion of the valve during ventricular systole leading to trivial mitral regurgitation, (4) was resulting in an abnormal filling pathway in ventricular diastole and thus producing a medially placed inflow murmur, but (5) was not associated with the classical findings of mitral stenosis. This lesion could only be explained by a mass in the left atrium, and in the clinical setting a myxoma was much more likely than a ball valve thrombus.

Further investigation confirmed the clinical impression. At right heart catheterization the cardiac output, circulation time, and pulmonary vascular resistance were normal. The high *v* wave and the rapid *y* descent in the wedge pressure tracing were more in keeping with regurgitation than stenosis of the mitral valve. This suggested that the obstruction was not severe during the early phase of rapid left ventricular filling. A similar pulmonary wedge tracing was seen in Case 3 of Aldridge and Greenwood. The angiocardiogram confirmed the presence of a large filling defect in the left atrium.

Summary

A case of left atrial myxoma that simulated mitral stenosis is presented. The features that made a clinical diagnosis possible were (1) a relatively short history in the absence of arrhythmia or carditis, (2) symptoms suggestive of intermittent obstruction, (3) a loud first heart sound in the absence of an opening snap, (4) an atypical location of the diastolic murmur, (5) absence of presystolic accentuation of this murmur in the presence of sinus rhythm, and (6) absence of complications that would tend to depress the findings of mitral stenosis, such as low cardiac output, gross enlargement of the right ventricle, associated valve lesions, or calcification of the mitral valve. The diagnosis was confirmed by angiocardiography, and the tumour was successfully removed.

References

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