

Mitral incompetence associated with lipoma infiltrating the mitral valve

D R ANDERSON, M R GRAY*

From the Department of Cardiac Surgery, Guy's Hospital, London

SUMMARY A 54 year old man presented with presyncopal symptoms. Echocardiography and subsequently computed tomography showed a mass in the posterior mitral annulus causing incompetence of the valve. At operation a lipoma was found which could not be resected. Mitral incompetence was the result of chordal rupture where the lipoma had engulfed the papillary muscle. The valve was replaced and the patient made an uneventful recovery.

This case report shows a potential danger of an otherwise benign lesion.

Primary cardiac tumours are very rare and lipomas account for about 8% of these lesions. Approximately 50% are subendocardial, 25% are subepicardial, and 25% are subpericardial. The case presented here was unusual because the lipoma, though benign and arising in the wall of the ventricle, had infiltrated both the papillary muscle and the leaflets of the mitral valve and had caused incompetence of the valve.

Case report

A 54 year old white man presented with presyncopal symptoms which had lasted for two weeks and then spontaneously resolved. He was seen by his family doctor who referred him for cardiological assessment.

Further questioning did not elicit a history of rheumatic fever or other relevant cardiac history. He did, however, remark that more than 25 years before an Army Medical Officer had spent an unusually long time on auscultation of his heart and had called for a second opinion before passing him as fit for national service. No details of that examination are available.

Physical examination showed a fit looking man with blood pressure of 130/80 mm Hg when supine and normal jugular venous pressure. All peripheral pulses were present with a regular, normal wave form. The heart was impalpable. At auscultation, a soft late systolic murmur was best heard at the left sternal edge; there was no radiation. The remainder of the physical examination, including the respiratory system, was normal.

Requests for reprints to Mr D R Anderson, FRCS, Department of Cardiothoracic Surgery, St George's Hospital, Blackshaw Road, London SW17 0RE.

*Present address: Alderhey Hospital, Liverpool.

The erythrocyte sedimentation rate was 4 mm in the first hour and the full blood count was normal with a normal white cell differential count. Biochemical tests were also normal. The electrocardiogram showed sinus rhythm (90 beats per minute), a normal cardiac axis, no ischaemic features at rest, normal P wave, PR interval 0.16 s, and no evidence of right or left ventricular hypertrophy or strain. Chest x ray showed a normal cardiothoracic ratio with a normal cardiac silhouette and clear lung fields.

The echocardiogram showed an echodense intracardiac mass approximately 2 cm in diameter, which seemed to be pedunculated and was mobile within the left ventricle. It seemed to originate from between the posterior insertion of the mitral valve and the posterior wall of the left ventricle. All other aspects of the echocardiogram were normal.

Computed tomography (figure) of contiguous sections of the heart was performed before and after the administration of intravenous contrast. This showed a rounded area (2.5 cm in diameter) with the density of fat in the left ventricle which was still present after contrast, but inconsistently so, suggesting the possibility of a mobile mass.

Angiography of the left ventricle showed a filling defect close to the posterior mitral annulus that seemed to be spreading down the posterior wall of the left ventricle. There was grade 2 mitral regurgitation with normal coronary arteries.

At operation the pericardial sac contained no fluid and there were no adhesions. There was a lipomatous mass on the posterior aspect of the left ventricle in the region of the mitral annulus. The left atrium was explored under standard cardiopulmonary bypass and cold cardioplegic arrest. This showed that the fatty tumour on the epicardial surface of the heart

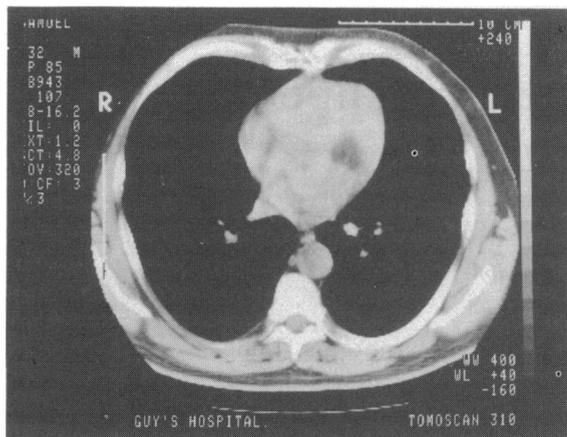


Figure Computed tomogram with contrast enhancement showing a filling defect (dark area) in the cavity of the left ventricle.

had penetrated through the posterolateral aspect of the left ventricular wall adjacent to the mitral annulus. It extended distally and subendocardially into the posterior ventricular myocardium and proximally through the mitral annulus into the floor of the left atrium. There was a patulous deformity of the lateral aspect of the mitral valve that was probably related to the anterior leaflet, though it was impossible to be certain about this because of the distortion produced by the fatty tumour at this point. In addition, there was rupture of some of the minor chordae attached to the anterior leaflet where the tumour seemed to have engulfed the anterior papillary muscle. It was impossible to excise the tumour so multiple biopsy specimens were taken. The mitral valve was excised and replaced with a bioprosthetic valve. The patient made a good recovery from the operation. When he was seen at follow up after three months he was well and ready to return to work. Repeat echocardiography showed a mass in the left ventricle as before, but reduced in size. He will be followed up by serial echocardiography.

Histology of the tissue showed a benign lipoma throughout all sections. There was myxoid change as well as infiltration by lipocytes into the anterior mitral leaflet and papillary muscle.

Discussion

Cardiac tumours are rare. The reported occurrence in two large necropsy series was 0.0017%¹ and 0.03%.² Nearly half of all cardiac tumours are myxomas. Rhabdomyoma, the next most common neoplasm, is the most common childhood tumour. Fibroma, lipoma, haemangioma, and lymphangioma

are rare mesodermal tumours. Lipomas must be distinguished from lipomatous hypertrophy of the interatrial septum, which is a more common finding. Lipomatous hypertrophy of the interatrial septum characterised by fetal fat cells, whereas lipomas contain mature lipocytes.³ Only about 25% of primary cardiac neoplasms are malignant and nearly all are sarcomas. But secondary deposits, which are 20–30 times more common than primary neoplasms, are the most common cardiac tumours.

Because cardiac tumours are rare their clinical diagnosis is difficult. Atrial myxomas are said to have characteristic features on auscultation that distinguish them from stenosis of mitral or tricuspid valves,⁴ but most workers found that these features were inconsistent. In the past many cardiac tumours were found unexpectedly at operation during closed mitral valvotomy for presumed mitral stenosis.

Modern cardiac imaging techniques such as angiography, cross sectional and M mode echocardiography,^{5,6} and, most recently, computed tomography,^{7–9} allow accurate diagnosis before operation. With computed tomography it is often possible to identify the tissue type in the observed mass. Lipomas have a very low attenuation coefficient, less than –50 Hounsfield units.^{8–10} There are many reports of atrial myxomas diagnosed by computed tomography but none mentions the attenuation values of the lesion.^{11–14} Tsuchiya *et al* reported that five of six cases had lesions that were less dense than blood and the sixth had a lesion with the same density as blood.¹⁴ They also reported consistent non-homogeneity of the masses and the presence of calcification in three. Nevertheless, some radiologists are sufficiently confident about the ability of computed tomography to identify fat that they are prepared to advise conservative management in the case of lipomas.⁸

The various published reports of cardiac lipomas provide no clear indication about the natural history of this lesion. In the patient described by Behnam *et al* a cardiac lipoma was first diagnosed at the age of two but the patient did not have an operation until she was 16, when multiple lipomas of the mitral valve and papillary muscle were found.¹⁵ Total excision was achieved by mitral valve replacement. Barberger-Gateau reported similar treatment of a fibrolipoma of the mitral valve.¹⁶ We have only found one other case where total removal was possible. Most workers agree with our observation that it was impossible to achieve total excision because of the tendency of the tumour to infiltrate throughout the myocardium.⁵ The case we report is the first to show such extensive infiltration, affecting the mitral valve leaflets and papillary muscles, and extension into the floor of the left atrium. Follow up of the cases treated

by incomplete excision has not been long enough to assess the behaviour of this tumour. None the less, we know that lipomas grow slowly at other sites and can safely be left alone. The patient described by Behnam *et al* lived for 14 years without any major problems except the development of mitral regurgitation, which was probably present when the patient was first seen at the age of two. The history of our patient suggests that the tumour may have been present 25 years earlier when he was examined for national service. If this is so cardiac lipomas may be regarded as being as benign as they are at other sites—except when they affect a valve or exert pressure on surrounding normal tissue. Unlike myxomas they do not seem to generate emboli, but until they can be diagnosed with certainty many will be explored to confirm the diagnosis. Computed tomography seems to offer the possibility of accurate tissue diagnosis and may avoid operation in some patients who are poor operative risks. But until the attenuation coefficient of myxoma tissues is confidently established a biopsy is the only way to be certain of the diagnosis.

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