

Calcified right atrial mass

Report of a case and discussion of the differential diagnosis

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This is the report of a case of a grossly calcified right atrial mass in a woman of 40 who also suffered from systemic hypertension, pyelonephritis, and hepatic cirrhosis. The differential diagnosis, surgical treatment, and necropsy findings are described along with a review of the literature.

Myxoma is the commonest intracardiac tumour and it is well known to present in bizarre ways, particularly when on the right side of the heart. The frequency of serious co-existent disease is striking and adds to the difficulty of diagnosis. Calcification of the tumour is uncommon, though probably more frequent in right than in left heart tumours; massive calcification is rare. Diagnosis can be followed by surgery with very satisfactory results.

The purpose of this paper is to present the case of an enormous, grossly calcified mass in the right atrium which extended down into the inferior vena cava and was associated with large calcified deposits in the main pulmonary artery and in the lungs. Although no recognizable myxoma tissue remained there is strong evidence that this was, in fact, a 'burnt-out' myxoma. The patient also had chronic pyelonephritis and hepatic cirrhosis.

The difficulty of diagnosis is discussed and the literature on calcified atrial myxomata is reviewed.

CASE HISTORY

Mrs. E. L. was aged 40 years when first seen in 1968.

In 1951 she had suffered from severe pre-eclamptic toxæmia leading to an abortion at five months. Her blood pressure was 190/120 mmHg and on that occasion pus cells, red blood cells, and granular casts were found in the urine. She was subsequently admitted for inevitable abortions in May and again in August 1955. These were at approximately three and two months' gestation respectively.

In June 1968 she was referred for treatment of menorrhagia of six months' duration. She was found to have a blood pressure of 220/160 mmHg and heavy albuminuria. The uterus was bulky and before a dilatation and curettage she was referred for a medical opinion to Dr. K. D. Allanby who made the following observations. She was breathless on climbing stairs. She slept on two pillows and there had been gross ankle oedema for six months. A rash and periorbital

oedema had appeared about one week previously and were attributed to strawberries. The venous pressure was above the angle of the jaw, oedema of the legs was well marked, and there was hepatomegaly. A coarse rub, probably of pericardial origin, was heard.

A diagnosis of cellulitis of the legs was made, and she was admitted to Peterborough Memorial Hospital immediately. A haemolytic streptococcus was isolated from the throat and she was therefore treated with penicillin. She was also treated with chlorothiazide and methyl dopa, with some improvement. However, at the end of one month she was still in congestive cardiac failure and grossly overweight. The treatment of her heart failure was intensified but the result was disappointing. The 'pericardial rub' varied in intensity but never disappeared. On occasions she complained of a feeling of tightness in the chest. The electrocardiogram showed changes which were thought to be compatible with myocardial infarction but these did not vary throughout the course of her illness. The tracings showed sinus rhythm with a long PR interval (0.28 sec), a partial right bundle-branch block with T-wave inversion from V1 to V5, and low-voltage ventricular complexes. The haemoglobin varied between 93% and 113%, and the ESR was 27 mm in one hour. The white cells, platelets, and bone marrow were normal. No LE cells were found on repeated search, and tests for antinuclear factor were negative. The blood urea was usually within normal limits but it rose to 67 mg/100 ml in August 1968; the electrolytes were normal. Protein electrophoresis showed a slight reduction in the albumin. The ASO-titre was 255 units. The serum levels of cholesterol, creatinine, phosphokinase, B12, folate, and iron, and the serum iron-binding capacity were all normal. The creatinine clearance was 90 ml/min and urinary VMA and 5 HIAA excretions were not elevated. The LDH was persistently raised, being 980 units in July and 1,980 units in August. The urine contained cellular casts on one occasion but most frequently showed only albuminuria.

She was admitted to Addenbrooke's Hospital on 1 November 1968. By then she had somewhat improved and her blood pressure was 140/100 mmHg.

The 'rub' remained. She was still in heart failure. Gross albuminuria persisted and there were a few red cells in the urine which was sterile. The following tests were normal or negative: full blood count, serum electrolytes, proteins and the electrophoretic pattern, liver function tests, serum calcium, phosphorus, cholesterol and iron, protein bound iodine, T3 resin uptake, urine VMA, bleeding time, kaolin-cephalin clotting time, direct Coombs test, LE cells, and the Rose-Waaler and Paul Bunnell reactions.

The blood urea was by now 57 mg/100 ml, and the creatinine clearance 30 ml/min, and a 24-hour urinary protein was 1.3 g.

RADIOLOGICAL FINDINGS The chest radiograph showed gross cardiac enlargement. Some irregular densities in the left hilar region were noted and thought to be due to calcification of the lymph nodes. It was not until she was being screened prior to a renal aortogram that Dr. Gregg noted patches of calcification in the heart and mentioned that these were in the region of the right ventricle and pulmonary artery. Tomographic cuts from 12 to 16 cm subsequently showed that the two chief areas of calcification were in the heart. A survey for metastatic calcification elsewhere in the body was negative.

During an infusion pyelogram it was noted that four gallstones were present. Both kidneys excreted equally but the contrast was not good. The right kidney measured approximately 10.8 cm in its long axis and

the left 12 cm. The examination was otherwise unremarkable. A renal arteriogram was carried out and this showed no evidence of renal artery stenosis. The intrarenal pattern on both sides suggested a nephrosclerotic type of vascular lesion. There was also shrinkage of the upper pole of the right kidney and some reduction of the cortical thickness. There was irregularity of the outline, suggesting that there might be pyelonephritis which appeared to be bilateral. There was a reduction in the arterial flow and also in the tubular and excretory phases.

Renal biopsy was carried out and the findings were consistent with chronic pyelonephritis.

Rectal biopsy was negative for amyloid.

At this stage we had a patient in gross congestive cardiac failure with systemic hypertension and a diagnosis of chronic pyelonephritis. There were areas of massive calcification within the heart shadow without any overt disturbance of calcium or phosphorus metabolism. The ESR was normal and there was no evidence of a systemic disorder. It was thought that the 'pericardial rub' was due to the mechanical effect of the calcium within the heart. The systolic and diastolic elements of this were confirmed by phonocardiography.

She was admitted to Papworth Hospital on 18 November 1968 for cardiac investigation. On screening the heart it was agreed that the inferior calcification moved into the right ventricle in early diastole and the superior calcification moved away from the ventricle in systole, i.e., paradoxical movement. On the

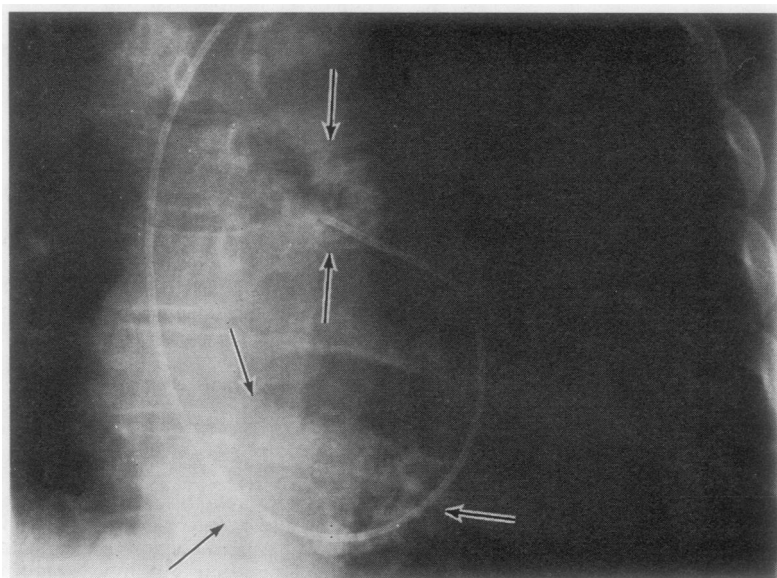


FIG. 1. Taken during cardiac catheterization. The tip of the catheter is engaged in the calcified mass in the pulmonary artery and the lower arrows indicate the larger mass passing through the tricuspid valve.

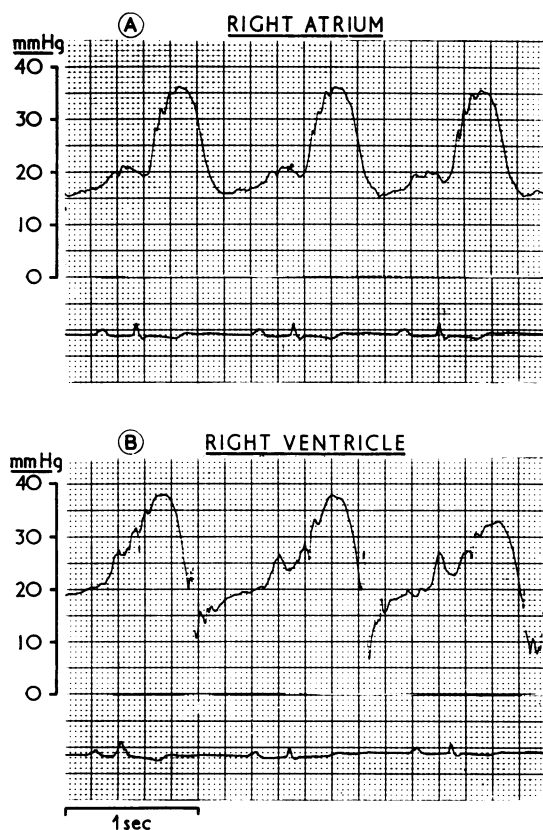


FIG. 2. A, Right atrial pressure pulse; B, right ventricular pressure pulse.

eve of cardiac catheterization and angiocardiography the diagnosis of calcified right atrial myxoma with calcified metastases in the pulmonary artery was made.

At cardiac catheterization the catheter was manipulated very gently into the main pulmonary artery where its further passage was interrupted by a calcified mass which could be felt on the tip of the catheter (Fig. 1). It was subsequently possible to pass the catheter past this into the distal pulmonary artery. The pressures were as follows:

Right atrium V wave at 36 mmHg (Fig. 2A)
 Right ventricular pressure 35/15 mmHg (Fig. 2B)
 Pulmonary artery 35/20, with a mean of 27 mmHg.
 Left brachial artery 185/110 mmHg.

The catheter was then replaced by a No. 9 NIH catheter and angiocardiography was carried out by injection of the contrast medium high into the right atrium. This showed that the right atrium and the right ventricle were grossly enlarged and the right atrium contained an irregular filling defect corresponding in position to the lower and larger calcified mass. This

defect was seen to enter the right ventricle during diastole. The right ventricular cavity appeared to be curiously striated, suggesting that there was a great increase in transverse trabeculation. The relationship of the superior smaller calcified mass to the pulmonary artery was confirmed.

Anticoagulant therapy with warfarin-sodium was instituted because of the possibility of thrombus occurring in the right atrial mass and giving rise to pulmonary embolism.

SURGERY On 17 December 1968 Mr. Milstein explored the heart under cardiopulmonary bypass. The right atrial mass was palpable when the heart was exposed. It could be felt extending into the tricuspid orifice and for some 4 cm down the inferior vena cava. A small mass could be felt in the main pulmonary artery extending down right and left main branches. Bypass was started and the right atrium was found to contain a heavily calcified polypoid mass measuring 7×5×5 cm (Fig. 3). This mass had no attachment to the right atrium or the atrial septum. Its only attachment was within the inferior vena cava. It extended through the destroyed tricuspid orifice into the right ventricle whence it was delivered by traction. With some difficulty the portion remaining in the inferior vena cava was removed by sharp dissection. It appeared to occlude the inferior vena cava almost completely and extended into the hepatic vein. The main pulmonary artery was opened with a vertical incision. A similar tumour was found to be firmly adherent to the walls of the bifurcation of the main pulmonary artery and its right and left branches. This was removed piecemeal with sharp dissection. Over an area of about 1 cm high on the atrial septum were a number of sessile, pearly nodules which were excised. The tricuspid valve had been completely

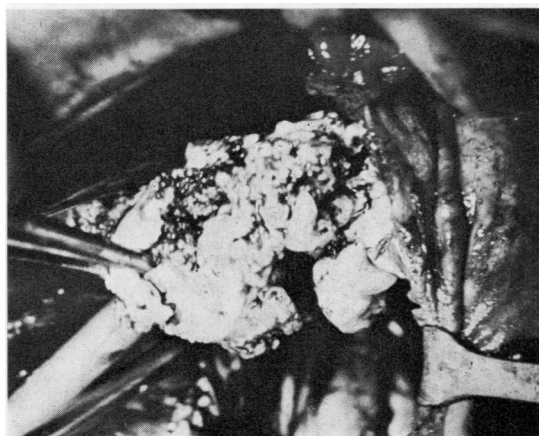


FIG. 3. Frame from cine taken at cardiotomy showing the polypoid calcified mass being delivered from the right ventricle through the atrial incision.

destroyed by the larger calcified mass. No cusps, chordae or papillary muscles were left. A 3M Starr-Edwards prosthesis was therefore sutured in the tricuspid orifice. Epicardial pacing wires were fixed at the end of the operation. Total bypass time was 2 hours 30 minutes.

Following surgery she remained hypotensive and unconscious with a low cardiac output. Despite treatment, her circulatory state and the ensuing oliguria failed to improve and after a series of cardiac arrests she died on the fourth postoperative day.

BIOPSY The surgical specimens came from varying sites between the venae cavae and the right pulmonary artery. The largest single specimen came from the right atrium and was a coiled mass with smooth-surfaced yellow and grey gyri and ulcerated calcium in the sulci (Fig. 4). The appearance suggested a coiled cylindrical structure, 1 to 1.3 cm diameter and about 13 cm in total length. There appeared to be no uncalcified tissue present and free iron could not be demonstrated.

Microscopically, in the right ventricle there was fibrous tissue on the eroded surface of muscle and small coronary artery branches. The intima and inner media of the pulmonary trunk were eroded and calcium was embedded in the damaged wall. The atrial septum showed endocardial fibrosis but no evidence of previous erosion of the underlying myocardium.

The calcified material looked very similar to the tissue of heavily calcified heart valves, being mostly fibrous tissue and calcium, with occasional foci of recent haemorrhage or fibrin, some areas with

thin-walled blood vessels and some with a few inflammatory cells. Bone formation and myxomatous tissue were absent.

NECROPSY The necropsy was performed three days after death. The endocardium of the inferior vena caval orifice and the lateral wall of the right atrium were thickened, rough, and white. There was a small paravalvular leak lateral to the Starr-Edwards tricuspid prosthesis. Except for the apical 2 cm and the conus, the right ventricular cavity was lined by a granular, opaque, thickened endocardium. There were no remnants of tricuspid papillary muscles. The pulmonary valves were normal but the endothelium of the medial and posterior surface of the main pulmonary trunk were roughened. Fragments of calcified material were present in the segmental pulmonary arteries without thrombosis on the fragments and without infarction of the lung.

The liver was cirrhotic and the gall bladder contained four calculi. The brain was macroscopically normal and there was an old haematoma about the left kidney.

Microscopically there was widespread endocardial thickening in the right heart. This was composed of laminated and nodular arrangements of fibroelastic tissue containing occasional thin-walled blood vessels and a few flecks of fibrin together with round and stellate and spindle cells, and occasional small foci of lymphocytes and eosinophils. For the most part the process appeared to have been a laying down on a previously intact endocardium but in the left ventricle there were foci of oedema and stellate and spindle cells suggestive of a subendocardial myxomatoid degeneration. There was some residual calcium still present in the endothelium of the inferior vena cava.

With alcian blue at pH 2.5 there were patches staining intense blue both superficially and deeper in the thickened endocardium. These areas of alcian blue staining were also the areas of most intense PAS staining, but the intensity of PAS staining was not as great as that of the alcian blue. The only exception was in the endocardium of the normal left atrium where the PAS staining was slightly but definitely stronger than the alcian blue. This indicates a predominant acidic mucopolysaccharide with a little neutral mucopolysaccharide ground substance consistent with immature reactive fibrous tissue and not necessarily implying an original myxoma. The nodal artery in the right atrium showed a considerable deposition of strongly alcian blue and PAS staining mucin in its adventitia.

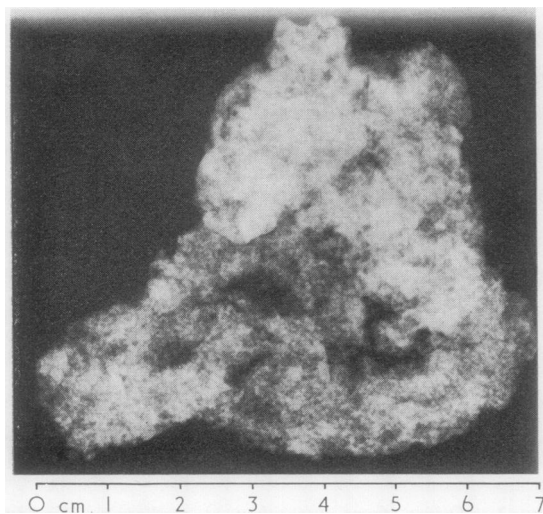


FIG. 4. Radiograph of the main mass of tumour removed at operation.

Stains for fibrin (Mallory's PTAH and Lendrum's MSB) showed a little fibrin incorporated in the thickened endocardium of the right ventricle and in a patchy thin film on the endocardium of the ventricles and right atrium with occasional Lambl excrescence-like structures having an acellular fibrous or fibroelastic core and a fibrin outer coat which are present in both ventricles. These lesions we would interpret as results of the abnormal turbulence around the calcified intracavitary mass.

In the lungs organized and recanalized old emboli were present in occasional pulmonary artery branches with a few calcific emboli. One calcific nodule in the right upper lobe apex appeared to have eroded through the arteriolar wall to reach the adventitia. Apart from the old and recent emboli the lungs showed some increase in medial thickness of pulmonary arteries. In none of the vessels was any recognizable myxoma tissue present.

Occasional calcific and sparsely cellular emboli up to 10 μ diameter were present in the coronary, cerebral, and adrenal arterioles.

The liver showed a fairly advanced portal type of cirrhosis rather than that which results from cardiac failure or hepatic venous occlusion of the Budd-Chiari type.

Patchy neuronal loss and subcortical demyelination were found in the cerebrum.

The kidney showed congestion with only minimal interlobular arterial intimal fibrosis.

DISCUSSION

PATHOLOGICAL The origin of the calcified mass in our patient is to some extent speculative as there was virtually nothing left of the active phase of the tumour. The two possibilities which have to be considered are myxoma and thrombus.

In favour of a myxoma is the presence of acidic mucopolysaccharides in the endocardium of the inferior vena cava, and the lateral wall of the right atrium and of the ventricles. It must be borne in mind that these areas had recently been the site of surgical trauma and this had probably been the cause of the increased acidic mucopolysaccharides in the subendocardial parts of the crests of some columnae carnae of the left ventricle. Similar mucoid changes are also seen in calcified aortic valves and in balloon degeneration of the mitral valve (Bittar and Soas, 1968), and these may indicate congenital abnormality in the valve structure but not necessarily a potential myxoma. Heath (1968) recommended alcian blue as the stain of choice for demonstrating myxomas. However, the

relative intensity of staining with alcian blue and PAS is not a reliable guide to myxoma (Fine, Morales, and Horn, 1968) nor is the presence or absence of elastic fibres. In neither the calcified mass nor in the heart was any vasoformative tissue seen, this being the other hall-mark of myxoma (Heath, 1968). Rodbard, Kihoshita, and Montes (1964), in an elegant experiment in dogs, produced apparently similar endocardial reactions by invaginating the left auricle over a firm Silastic ball.

In favour of a thrombotic origin is the twisted and coiled appearance of the larger pieces of excised calcified mass, the fact that apart from the embolic calcific fragments all other pulmonary vascular lesions were compatible with old thrombotic emboli, and the absence of myxoma tissue. It must be admitted, however, that the late results of myxomatous emboli are unknown. In the reported cases (Emmanuel and Lloyd, 1962; Heath and Mackinnon, 1964; Oliver and Missen, 1966; Fluck and Lopez-Bescos, 1968) only recent emboli were seen. The recent small calcific and fibrous and fibrinous systemic emboli in our patient were probably small fragments which were dislodged from the inferior vena cava and right atrium at the time of operation and passed through the filter in the Melrose pump oxygenator.

The observation made at the time of operation that there were two separate tumour masses attached respectively to the inferior vena cava and to the pulmonary artery could indicate a blood-borne or embolic spread but does not help to distinguish between a myxomatous or thrombotic origin for this patient's unusual tumour. It is possible that the tumour arose as a hepatic vein thrombus occurring at the time of the pregnancy toxæmia; the portal cirrhosis is consistent with past eclampsia (Popper and Schaffner, 1957). The presence of a little fresh fibrin on the surface of the clefts in the calcified mass is not unexpected and cannot be used as an argument in favour of a thrombotic origin for the calcified mass.

The few Lambl excrescence-like formations observed microscopically in the ventricles of this patient were similar to those noted in the heavily calcified myxoma of Oliver and Missen (1966). The minuteness and the infrequency of these excrescences would exclude the calcified mass being a unique degenerate papilliferous tumour of the heart. These rare papillary tumours are reported on the left side of the heart and are not calcified (Hudson, 1970).

Heath (1968) refers to calcification occurring in cardiac lipomata. There was no evidence of any

residual fatty tissue in our case and the calcification in lipomata is not a major feature when it occurs.

Dean, Pamukcoglu, and Roberts (1969) speculated that the calcified nodules in the right ventricle of their patient might be either the residua of mural thrombi or of *Histoplasma granulomata*. These authors did not consider myxoma and there appears to have been no myxoid change in the endocardium between the calcified endocardial nodules. Although not searched for there was no reason to suspect that this patient had had an histoplasma infection.

A study of the literature indicates that calcification is not infrequent in myxomata, especially those in the right atrium (Hudson, 1970), though it rarely completely replaces the tumour. Despite their much greater frequency, calcification is rare in thrombi in the atria and when it does occur it usually takes the form of a few plaques in the deeper endocardial part. It is essentially on this comparison with other less totally calcified myxomata that we venture to call this calcified mass a myxoma, even though its site of origin was the inferior vena cava and even though we have failed to find any convincing myxomatous tissue. We feel that there is a continuous gradation of cases from our totally calcified mass through the extensively calcified myxomata of Fluck and Lopez-Bescos (1968), the slightly less but still very extensively calcified myxoma of Oliver and Missen (1966), and the less calcified myxomata of Buenger, Paul, and Fell (1956) to the typical non-calcified myxoma.

CLINICAL Goodwin *et al.* (1962) emphasized that the general systemic effects of atrial myxoma are often of more diagnostic value than the classical but variable local signs. A useful evaluation of clinical and laboratory manifestations is made by Hattler, Fuchs, Cosson, and Sabiston (1970). The extensive literature on cardiac tumours in general and on myxomata in particular is well reviewed in the symposium published in the *American Journal of Cardiology* (Symposium on Cardiac Tumors, 1968), by Goodwin (1968), and by Hudson (1965, 1970).

Myxomata can occur anywhere in the heart and have even been described above the aortic valve and arising in the left ventricle (Danta and Williams, 1969). However, the commonest site of origin is from the atrial septum. More than three times as many tumours are found in the left atrium as in the right. Very occasional bilateral cases are recorded (Anderson *et al.*, 1970).

Our case is unusual for myxoma in that the 'tumour' was grossly calcified and arose in the

inferior vena cava, though Oliver and Missen's (1966) case, which was also calcified, arose close to the vena caval opening into the right atrium. In Fluck and Lopez-Bescos' (1968) case, a heavily calcified tumour arose in the fossa ovale and extended into the inferior vena cava. An inferior vena caval origin was also reported by Matsushita, Kuramochi, Kaneko, and Kuramoto (1968) for their haemorrhagic but not calcified myxoma.

The confusing presentation is in keeping with the general experience of myxomata and there is no doubt that right atrial myxoma can present as disease of any bodily system. Confusion is increased by the frequent co-existence of other disease, such as in our case where there was pyelonephritis with systemic hypertension and hepatic cirrhosis. Diagnosis can even be missed at cardiac catheterization; angiocardiology is obligatory for complete investigation. The diagnosis should be considered in any case of severe heart failure which does not respond to treatment.

The following review of some of the conditions that have been mimicked or falsely diagnosed illustrates these points. Because of the difficulty of making any reasonable classification, these are given in approximately chronological order. This by no means covers the whole literature. It will be noted that most of these cases were reported after 1960. An earlier review was made by Sannerstedt *et al.* (1962):

Sudden death from tumour embolization of the pulmonary artery

(Chiari, 1931, quoted by Prichard, 1951)

Recurrent attacks of heart failure for 43 years (Strouse, 1938) Carcinoma of rectum and marked atherosclerosis also present

Epilepsy and presumed bacterial endocarditis (Kendall and Symonds, 1952)

Ebstein's anomaly

(Coates and Drake, 1958)

Fatigue and intermittent oedema

(Campeti, Mahoney, and Yu, 1960)

Severe congestive failure and weight loss of 21 lb (Catt, Denborough, Grigg, and Sloman, 1962)

Polycythemia and raised platelet count regressing after removal of the tumour

(Levinson and Kincaid, 1961)

Constrictive pericarditis

(Sannerstedt *et al.*, 1962)

Short attacks of abdominal pain with extreme nausea, breathlessness and faintness, loss of appetite, and weight loss of 41 lb

(Barlow, Fuller, and Denny, 1962)

Pulmonary tuberculosis and later constrictive pericarditis

(Emmanuel and Lloyd, 1962)

Tricuspid stenosis, apparently confirmed by cardiac catheterization

(Morrissey, Campeti, Mahoney, and Yu, 1963)

Pulmonary stenosis with pressure gradient measured across the pulmonary valve

(Gottsegen, Wessely, Arvay, and Temesvari, 1963)

This tumour actually arose in the right ventricle

Recurrent pulmonary emboli with pulmonary hypertension

(Heath and Mackinnon, 1964)

Symptoms from the age of 26 years to the age of 63 years—thought to be tuberculous pericarditis

(Oliver and Missen, 1966) Grawitz tumour of the kidney also present

Diabetic, with Kimmelsteil Wilson syndrome

(Sterns, Eliot, Varco, and Edwards, 1966)

Joint pains, anaemia, disordered proteins, and heart antibodies diagnosed as rheumatic disorder.

(Currey, Mathews, and Robinson, 1967)

Steatorrhoea clearing after removal of tumour

(Webb-Peploe, Goodbody, Johnson, and McMillan, 1968)

Pericardial friction rub mimicking acute pericarditis

(Greenwood, 1968) Greenwood also mentions carcinoid and obstruction of the vena cava in the differential diagnosis.

Cyanosis and polycythaemia

(Miller, Paneth, and Gibson, 1968) This case had a patent foramen ovale.

Tricuspid incompetence

(Harvey, 1968) Harvey vividly describes the 'wrecking ball' action of the tumour so evident in our case.

Clubbing and cyanosis

(Talley, Baldwin, Symbas, and Nutter, 1970)

Diverse manifestations of inflammatory illness suggesting D.L.E., myocarditis, bacterial endocarditis or acute rheumatic fever

(Hattler *et al.*, 1970)

SYSTEMIC EFFECTS Fever, anaemia, raised ESR, and disordered plasma proteins were not found in our case, although these are frequently present in recorded reports. The mechanism of these changes is not clear, but it is possible that the completely burnt out appearance of our 'tumour' was consistent with its lack of reaction. The ESR was normal in the heavily calcified tumour of Fluck and Lopez-Bescos (1968) and was not recorded in Oliver and Missen's (1966) case. The very high LDH in our case was presumably due to continuing damage to the myocardium and liver.

AUSCULTATION The loud 'rub' was presumably due to the massive movement of the 'tumour' in and out of the ventricle which Harvey (1968) has so graphically described as the 'wrecking-ball' action. Matsushita *et al.* (1968) describe several systolic and diastolic clicks and murmurs, varying with posture in a non-calcified right atrial myxoma.

THE ELECTROCARDIOGRAM Sinus rhythm is remarkably common, considering the damage to the atrium. Atrial dysrhythmia is rarely mentioned and only one report of complete atrioventricular disassociation (Hattler *et al.*, 1970) has been discovered. Our patient consistently showed a first-degree heart block with a PR interval of 0.28 second. The low voltage of the ventricular complexes, together with the partial right bundle-branch block seen in our case, are relatively common features.

HAEMODYNAMICS The 'tumour' had completely destroyed the tricuspid valve and inflicted much damage within the right ventricle. This had resulted in the changes found in the Ebstein anomaly—so that the pressure pulses recorded in the superior vena cava, the right atrium, and the right ventricle were very similar to one another, with a systolic pressure of nearly 40 mmHg in all chambers (Fig. 2A and B). The notch on the upstroke of the right ventricular pressure trace has already been noted by Fluck and Lopez-Bescos (1968) in their case, and by others in cases of left heart tumours (Penny *et al.*, 1967; Pitt, Pitt, Schaefer, and Criley, 1967) and is attributed to the movement of the tumour in and out of the ventricle.

EMBOLI AND METASTASES The occurrence of pulmonary emboli and infarcts with atrial myxoma is frequently mentioned (Emmanuel and Lloyd, 1962; Heath and Mackinnon, 1964), and calcified emboli were found in Oliver and Missen's (1966) case. Heath and Mackinnon (1964) noted that at numerous sites the adventitia had been invaded and that there were also emboli in the bronchial arteries; they suggested that this demonstrated a limited invasiveness. Similar limited invasiveness had been recorded with emboli from left atrial myxoma in the mesenteric and brain vessels (Ringertz, 1942). In our case the main pulmonary artery was extensively scarred at the site of the large calcified deposits but there was no histological evidence to confirm invasion. The main pulmonary arteries must be an unusual site for large myxoma deposits to occur and we have found no report similar to this. There were, in addition, numerous calcified deposits in the pulmonary arteries, especially in the right lower lobe.

CALCIFICATION Degenerative changes are commonly reported in these tumours, but we have found no previous report of one which appeared to be so completely calcified. As is usual in degenerative areas, minor calcification is common and bone formation is occasionally recorded

(Gottsegen *et al.*, 1963). Strouse (1938) first reported calcification in a tumour of 43 years' duration, but gross calcification such as was seen in our case is rare and appears to be relatively more common in right atrial than in left atrial myxomata. Once seen, its radiological appearance is not easily forgotten—particularly the dramatic movement on fluoroscopy. Buenger *et al.* (1956) described calcification visible radiologically in a boy aged 16 years. Wight, McCall, and Wenger (1963) described two cases with calcified tumours, one in the right atrium and one in the left. Oliver and Missen (1966) described extensive microscopic calcification in a tumour removed from a man aged 63 whose symptoms dated from the age of 26, and in whom the calcified mass had been noted radiologically from the age of 26. Harvey (1968) also noted calcification in a woman aged 60. Greenwood (1968) says that gross calcification is rarely present in myxomata and records that it has been seen fluoroscopically in two right atrial and one left atrial myxomata. The case of Fluck and Lopez-Bescos (1968) seems to resemble ours in the grossness of the calcification as well as in the low ESR and the haemodynamic features.

TREATMENT Surgery has been successful in a number of cases (e.g., Fluck and Lopez-Bescos, 1968). In our case surgery was technically successful but the right ventricle was so massively damaged that it did not appear capable of adequate output to maintain life. The friable nature of the calcified tumour also proved a hazard not recognized at the time of operation. Multiple fragments, too fine to be arrested by the filter in the pump oxygenator, embolized small vessels in many of the organs examined. The long period on cardiopulmonary bypass necessary for the removal of the tumour probably also contributed. Detailed dissection of the tumour may have prolonged this time and produced a number of calcific emboli in the lungs. Although occasional tumour recurrences are reported after surgery (Bahl *et al.*, 1969; Baumann and Clavadetscher, 1969), the results of simple local excision in a number of series are excellent (Goodwin *et al.*, 1970). The calcified mass in the pulmonary artery was not causing obstruction and Mr. Milstein considers it would have been wiser to have left it and to have carried out a simple resection of the main mass prolapsing into the right ventricle with relief of the inferior vena caval obstruction.

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ADDENDUM

Since this paper was submitted for publication Waxler, Kawai, and Kasparian (1972) have reported another case of successful operation on a right atrial myxoma.

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