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Eosinophilic endomyocardial disease due to high grade chest wall sarcoma

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Abstract
Eosinophilic endomyocardial disease is characterised by persisting blood eosinophilia and acute endocardial lesions which progress to endomyocardial fibrosis. In most cases the cause is unknown but it has been described in association with malignant tumours. A fatal case is presented of a 64 year old woman with this disease due to a high grade sarcoma of the chest wall, an association not previously reported.

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Case report

A 64 year old housewife presented with a two month history of general malaise, anorexia, weight loss, non-productive cough, progressive effort dyspnoea causing breathlessness on minimal exertion, and a two week history of increasingly severe left upper posterior chest and shoulder pain. There was no history of haemoptysis or oedema. The patient was a current smoker of 40 pack-years with no relevant past medical history. Physical examination revealed evidence of recent weight loss, dyspnoea at rest with a respiratory rate of 24/minute, without cyanosis, lymphadenopathy or finger clubbing. She was afebrile with a regular heart rate of 90/minute, blood pressure 140/70 mm Hg, with a

third heart sound but no murmurs. Tenderness without palpable swelling was present over the left second and third ribs posteriorly. There was dullness to percussion at both bases with generalised expiratory wheezing on auscultation. Examination of the thyroid, abdomen and central nervous system showed no abnormality.

Laboratory investigations showed significant eosinophilia of $22 \times 10^9/l$ of a total white count of $46.1 \times 10^9/l$, haemoglobin 11.0 g/dl, ESR 50 mm/hour (Westergren). Serial chest radiographs with views of the left upper ribs showed a pleural-based opacity in the left upper zone peripherally associated with rapid bone destruction at the left second rib posteriorly and a pathological fracture of the left third rib. There were small bilateral pleural effusions present initially with subsequent consolidation of the right lower lobe after 10 days. The electrocardiogram showed sinus tachycardia with widespread T wave inversion. Echocardiography showed undilated chambers with significant asymmetrical thickening of the posterior wall of the left ventricle. On apical views there was dramatic thickening of the lateral wall of the left ventricle with binding down of the posterior mitral valve leaflet. Mitral regurgitation was demonstrated. Urinalysis, serum levels of urea, creatinine, electrolytes, and liver function tests were all normal. A fine needle aspiration of the pleural-based mass showed numerous large malignant cells with many surrounding eosinophils.

The patient required narcotic analgesia for pain relief and commenced oral prednisolone 40 mg daily resulting in rapid resolution of her eosinophilia, but she deteriorated progressively with dyspnoea due to heart failure which was unresponsive to treatment and died 21 days after admission.

Post mortem examination showed that the apex of the left upper lobe was tethered to thickened pleura by a 10 cm partially necrotic tumour mass that was eroding and partially destroying the second, third, and fourth ribs posteriorly. Histological examination of this tumour revealed a high grade sarcomatous neoplasm (figure) with moderate numbers of bizarre multinucleate giant cells. Epithelial markers were negative on immunohisto-

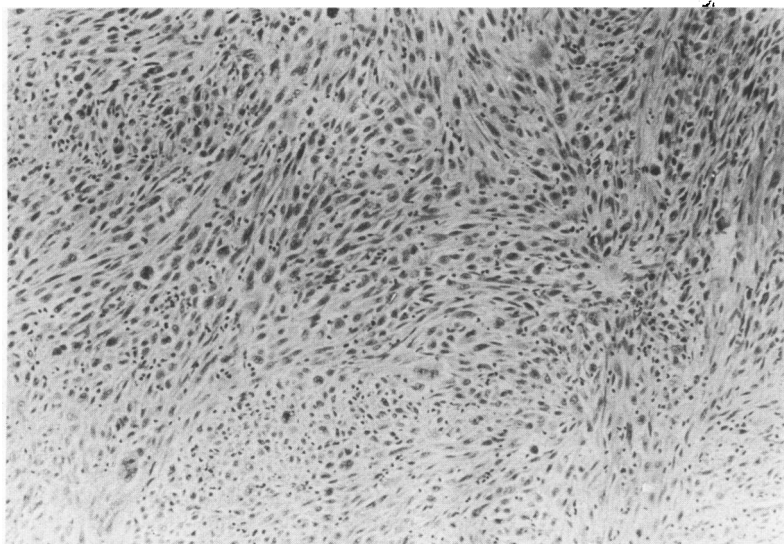
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Section of the chest wall tumour showing high grade sarcoma and several multinucleate tumour giant cells. Stain: haematoxylin and eosin, magnification $\times 100$ reduced to 62% in origination.

chemical analysis, but many of the tumour cells reacted with vimentin. Desmin 2-100, smooth muscle actin, and myoglobin were all negative. The heart was normal in size (290 g) with an adherent mural thrombus over the posterolateral aspect of the left ventricular wall and tethering of the posterior leaflet of the mitral valve. On sectioning the myocardium appeared to be pale and fibrotic with overlying thrombus with a mild degree of fibrous endocardial thickening and fibrous septa extending into the underlying myocardium. Large numbers of eosinophils were present both in the endocardium and myocardium, consistent with Löffler's endocarditis.

Discussion

Eosinophilic myocardial disease became well known after description of two patients in 1936 by Löffler.¹ It is characterised by persistent blood eosinophilia and acute endocardial lesions which progress to endomyocardial fibrosis. Olsen and Spry² proposed that the heart is involved in three stages. Initially, within six weeks, there is myocarditis and arteritis fol-

lowed by thrombosis within a year and then predominant fibrosis. The cardiac distribution varies with biventricular involvement in about half of cases, left ventricular alone in 38%, and right ventricle alone in 12%.³ In the case presented here the posterior and basal portions of the heart were predominantly involved with posterior mitral leaflet tethering, a distribution previously reported.⁴ Findings on echocardiography usually include septal or posterior wall hypertrophy, interference with mitral valve leaflet function, and ventricular thrombi. Ventricular function may be hyperdynamic.⁵ The pathology consists of endocardial thickening with strands extending into the adjacent myocardium and superimposed, sometimes calcified, thrombus is common. The eosinophil in eosinophilic states is often hypodense and granulated with low cationic protein levels and higher oxygen consumption⁶ when compared with normal eosinophils. These abnormal cells have the capacity to degranulate⁷ after appropriate stimulation to release toxic constituents such as eosinophil peroxidase and other basic proteins which may be important in the tissue injury. Eosinophilias seen with solid tumours are often the result of tumour-derived eosinophilopoietic activity, and have been reported for tumours as diverse as lung carcinomas, medullary carcinoma of the thyroid,⁸ and malignant fibrous histiocytoma, but the association of eosinophilic endomyocardial disease with sarcoma has not previously been reported.

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