Echocardiography in Löffler's endocarditis

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SUMMARY In a case of Löffler's endocarditis the main findings on M-mode and cross-sectional echocardiography were normal mitral leaflets, normal left ventricular dimensions, interventricular septal thickening, and abnormal echoes within the left and right ventricular cavities.

Löffler's eosinophilic endocarditis^{1,2} is a rare cardiomyopathy. Echocardiographic features of this disorder have been described³⁻⁵ but few illustrative echocardiograms have been published. This report details the M-mode and cross-sectional echocardiographic findings in a patient who had hyper-eosinophilia and a mitral systolic murmur and was considered to have Löffler's endocarditis.

Case report

A man aged 69 presented with a three day history of haemoptysis and dyspnoea at rest. He had sustained a hemiplegia five years previously and had a two year history of increasing exertional dyspnoea and occasional effort-related central chest pain. He had no rheumatic or allergic history and had never been outside the United Kingdom.

He was afebrile, tachypnoeic, and centrally cyanosed. He was in sinus rhythm (88 per min): blood pressure, initially 170/100 mmHg, settled within 24 hours to 140/80 mmHg. His apex beat was forceful and he had a fourth heart sound and a short, early mitral systolic murmur. Mid-zone and basal crepitations were present in both lung fields. The right ventricle was palpable, the jugular venous pressure was normal, there was no peripheral oedema, and there was no evidence of deep venous thrombosis. The old left hemiplegia was noted: there were no bruits over the extracranial cerebral arteries.

His haemoglobin was 12.6 g/dl and the ESR was 23 mm. The total white count was $11.9 \times 10^9/l$ with 42 per cent eosinophils ($5.3 \times 10^9/l$). Sputum culture and cytology were negative and no parasites or ova were seen in the stools. Immunoglobulins were normal and aspergillus precipitin and trichinella complement fixation tests were negative.

The electrocardiogram showed P wave notching and left ventricular hypertrophy with ST depression and T wave inversion in the anterolateral leads. On the chest x-ray there was left ventricular prominence (cardiothoracic ratio: 150/275), pulmonary venous congestion, and pulmonary oedema. A lung scan (seven days after admission) was normal.

MANAGEMENT

Left ventricular failure, which was initially unexplained, was treated with diuretics and, as additional pulmonary embolism seemed likely, the patient was anticoagulated. Löffler's endocarditis was diagnosed some weeks later on the basis of persisting eosinophilia (40%) and the mitral murmur; echocardiographic investigation was then undertaken.

ECHOCARDIOGRAPHY

M-mode echocardiograms (Fig 1) were recorded with a Picker 80C ultrasonoscope and a 2.25 MHz transducer.

The mitral diastolic closure rate was 50 mm/s (normal for our laboratory, 130 to 230 mm/s) but the mitral leaflets were thin and their motion was normally directed. Aortic and tricuspid valve echoes were normal. The maximal transverse dimension of the left atrium (3.0 cm) was normal as was the left ventricle (6.7 cm at end-diastole and 4.2 cm at end-systole). The left ventricular ejection fraction, calculated on the cubed assumption, was 76 per cent. Abnormal intracavitary echoes were recorded in the region of the left ventricular apex: they appeared granular and encroached on the mitral subvalvar apparatus and on the left ventricular aspect of the interventricular septum. The right ventricular outflow tract looked normal, but below this multiple predominantly linear echoes were recorded within

Fig. 1 (a) M-mode sweep from aortic root to left ventricular apex. (b) M-mode sweep from apex to aortic root.

the right ventricular cavity, obscuring the anterior surface of the septum. Left ventricular posterior wall thickness (1.4 cm) was normal. Close to the aortic root, the interventricular septum was clearly thickened (2.0 cm): it was impossible to determine its width below this level because endocardial detail was obscured. Septal motion was normally directed but pulsation appeared to be diminished particularly in its middle third.

Cross-sectional echocardiograms (Fig. 2) were recorded from a parasternal probe position using a prototype phased array. Long axis views (Fig. 2a) showed normal mitral leaflets, normal left ventricular dimensions, septal thickening, and abnormal echoes (arrowed) within the left ventricular cavity in the region of the posterior papillary muscle: similar, but less well-defined echoes were visible towards the apex of the right ventricle. Short axis scanning just below the mitral valve (Fig 2b) showed thickening of the interventricular septum

and a normal left ventricular cavity. Abnormal echoes were clearly defined within the left ventricular cavity on short axis scans closer to the apex (Fig. 2c).

Discussion

In view of our patient's age and prompt response to treatment, invasive investigation could not be justified. We thus have no histology or catheter data to substantiate the diagnosis of Löffler's endocarditis. We are, however, reasonably sure that it is correct—the persisting eosinophilia, the mitral murmur, the initially inexplicable left heart failure, and the history of possible pulmonary and systemic emboli are all in keeping.

The normal ejection fraction and, as evidenced by the mitral diastolic closure rate, the impaired diastolic function of the left ventricle, are consistent with previous echocardiographic^{3,5} and angiographic⁵ observations. Others have shown thicken-

Fig. 2 (a) Cross-sectional echocardiogram in left ventricular long axis (see text). (b) Cross-sectional echocardiogram in left ventricular short axis just below the mitral valve. (c) Cross-sectional echocardiogram in left ventricular short axis close to the apex.

ing of both the septum and the left ventricular posterior wall³; the present case indicates that posterior wall involvement may be minimal or absent.

The abnormal intracavitary echoes presumably reflect thrombosis superimposed on endocardial fibrosis; similar M-mode findings have been reported previously.⁴ Though the only clinical evidence of right-sided disease was the possible pulmonary embolus, the fibrothrombotic process clearly involved both ventricles. Cross-sectional echocardiography added little to the M-mode assessment but did define the site and extent of the obliterative process within the left ventricle. As this was shown to involve the region of the papillary muscles and to spare the mitral leaflets, it is reasonable to conclude that the mitral murmur was evidence of papillary muscle dysfunction.

These observations indicate that in Löffler's eosinophilic endocarditis, as in hypertrophic cardiomyopathy, echocardiography is a useful diagnostic tool.

References

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