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Rapid disappearance of left ventricular mass (presumed thrombus) in a patient with cardiomyopathy

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SUMMARY A man aged 41 years with a clinical diagnosis of cardiomyopathy and hepatic insufficiency was found, on angiocardiography, to have a mass, presumably a thrombus, within the left ventricle. A second angiocardiogram performed five days later showed that the thrombus had disappeared. The mechanism invoked was more probably an increase of spontaneous fibrinolysis as described in cirrhotic patients, perhaps helped by heparin, rather than ejection of the mass from the ventricle with silent embolisation somewhere in the systemic circulation.

The detection of a left ventricular mass in vivo may be made by angiocardiography. Two dimensional echocardiography is, however, the best and the most sensitive technique. A left ventricular mass is usually a thrombus but occasionally a tumour. We report a patient with congestive cardiomyopathy in whom a left ventricular mass apparently disappeared within five days of its detection by angiocardiography.

Case report

A 41-year-old man, a labourer, was admitted to hospital on 24 August 1979, because of congestive heart failure. There was a past history of heavy alcohol ingestion and laboratory data showed hepatic insufficiency (Table 1). The blood pressure was 110/70 mmHg, pulse regular at 116/min, and temperature normal. The erythrocyte sedimentation rate was 3 mm in one hour. There was venous distension and oedema of both legs. Basal râles were heard in the chest. There was a faint systolic murmur 1/6 at the apex. The heart was enlarged on chest radiography (cardiothoracic ratio 0·62). The electrocardiogram showed sinus tachycardia with left ventricular hypertrophy and left bundle-block branch. An M-mode echocardiogram recorded at the fourth intercostal space at the left sternal edge showed dilatation of the left ventricular cavity with an end-diastolic dimension of 6 cm and a very hypoactive interventricular septum. The patient was regarded as having congestive cardiomyopathy. He was given digitalis and diuretics and the result was good.

Cardiac catheterisation on 31 August 1979 (Table 2) showed an increase of both right and left ventricular end-diastolic pressures. The cardiac index was 2·4 l/min per m^2, left ventricular end-diastolic volume was 182 ml/m^2, and the ejection fraction was 0·21.

Selective left ventricular cineangiography in the right anterior oblique projection disclosed dilatation and considerable hypokinesia of the left ventricle. In addition, angiography showed a mobile left ventricular filling defect at the apex during diastole moving

Table 2 *Cardiac catheterisation data*

	Pressure (mmHg) Systolic/diastolic	*Mean*
Right atrium	17/9	13
Right ventricle	55/16	—
Main pulmonary artery	55/32	40
Wedge pressure	38/23	30
Left ventricle	120/35	—
Aorta	120/90	100

Table 1 *Laboratory data*

Albumin (g/l)	*Alkaline phosphatase*	*AST* (U/ml)*	*ALT* (U/ml)*	*γGT* (U/ml)*	*BSP* retention (%)*	*Prothrombin time (%)*	*Total cholesterol (mmol/l)†*	*Bilirubin (μmol/l)†*
36	12	41	62	74	100	60	2·7	6·84

*Abbreviations: AST, serum aspartate aminotransferase; ALT, serum alanine aminotransferase; γGT, gamma glutamyl transferase (normal value <40); BSP, bromsulphophthalein.
†Conversion from SI units to traditional units: cholesterol 1 mmol/l ≈ 38·6 mg/100 ml; bilirubin 1μmol/l ≈ 0·058 mg/100 ml.

toward the aortic valve in systole (Fig. 1). The size of the defect was 2·5 × 1·5 cm. The patient was considered to have a left ventricular thrombus rather than a left ventricular tumour. Heparin was given intravenously at the rate of 40 mg (4000 IU) every three hours and surgical treatment was planned. Before this, however, a further cineangiogram was taken on 5 September 1979. It was surprising to see that the left ventricular filling defect had disappeared (Fig. 2). The patient had not shown peripheral or cerebral emboli. Selective coronary arteriography was normal. The patient was discharged home on 9 September 1979. He was seen four, eight, and 12 months later and was reasonably well, taking anticoagulant therapy, digitalis, and diuretics.

Discussion

The left ventricular mass was considered to be a thrombus particularly because the underlying disease was a cardiomyopathy. Left ventricular thrombi occur frequently in cardiomyopathies, with an incidence of 25 to 50% of necropsied patients, according to Perrin *et al.*[1] and Reeder *et al.*[2]

The disappearance of this mass, recognised five days after anticoagulant treatment, can be explained

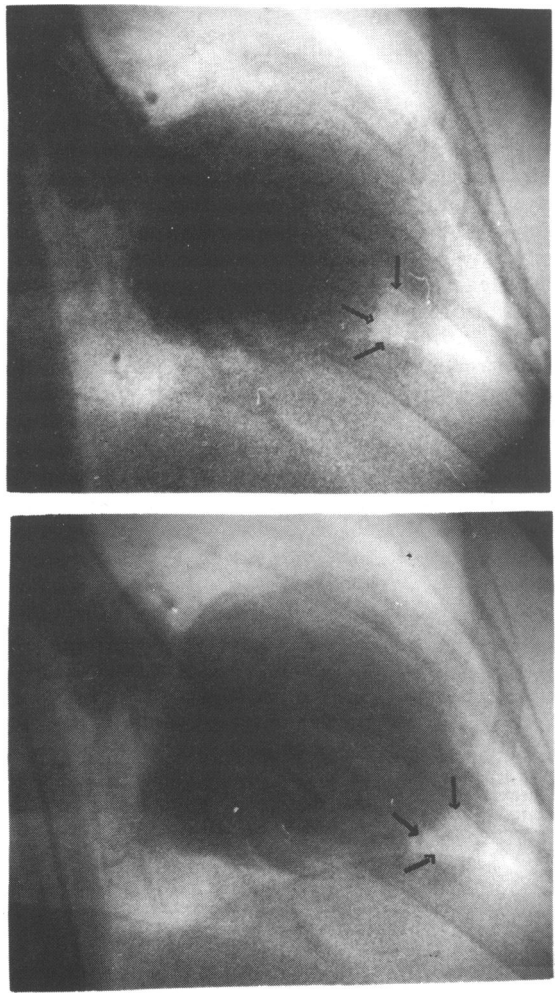

Fig. 1 *First cineangiography performed on 31 August 1979. Left ventriculogram in the right anterior oblique projection, in systole (top) and diastole (bottom). Note the filling defect at the apex of left ventricle.*

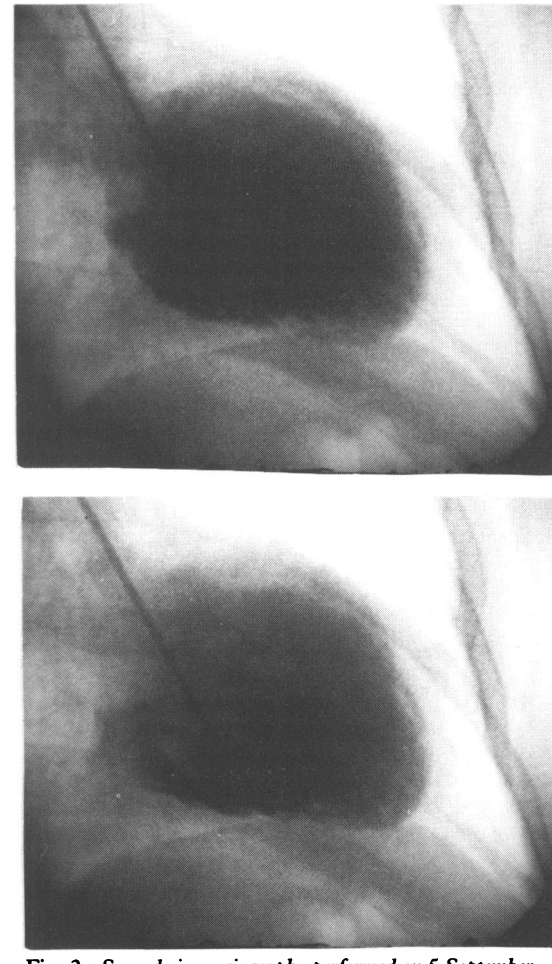

Fig. 2 *Second cineangiography performed on 5 September 1979. Left ventriculogram in systole (top) and diastole (bottom). Note the disappearance of the filling defect. The similar appearance of the left ventricular cavity in systole and diastole indicates severe generalised hypokinesia, presumably a result of cardiomyopathy.*

in one of two ways. The first is expulsion from the ventricle to produce silent embolisation somewhere in the systemic circulation. Miller *et al.*[3] have reported several cases of silent emboli, especially involving the spleen or kidneys in acute and in healed myocardial infarction. The second explanation which seems the more probable is lysis of the thrombus. According to Fletcher *et al.*[4] and to Pises *et al.*,[5] disappearance of the thrombus could be explained by increased spontaneous fibrinolysis. Our patient had severe hepatic insufficiency and these authors[4 5] showed abnormally active fibrinolysis in cirrhotic patients, probably the result of failure of hepatic clearance mechanisms for plasminogen activator.

Other cases of disappearance of left ventricular thrombi have been previously reported.[6 7] Mikell *et al.*[6] have followed 15 patients, eight with chronic cardiomyopathy and seven with recent myocardial infarction, in whom left ventricular thrombi were discovered. They performed serial two dimensional echocardiograms, 33 to 240 days after initial examination. In the nine patients who received anticoagulation for at least 30 days, left ventricular thrombi resolved in five cases, decreased in size in two cases, and remained unchanged in two cases. No patient had clinically apparent emboli. In the six patients who did not receive anticoagulant treatment, the left ventricular thrombi remained apparently unchanged in five cases, and the size of the thrombus decreased in one case; two patients had emboli. Meltzer *et al.*,[7] also using serial two dimensional echocardiograms, found a reduction in size of left ventricular thrombi at 10-month follow-up in two patients, and in one of them the mass had disappeared after 19 months.

According to these reports and in view of our own case we suggest that when a left ventricular mass is discovered in patients with cardiomyopathy, anticoagulant treatment should be given and surgical treatment may be delayed. Serial two dimensional echocardiograms are advised to follow the size of the intraventricular mass. If echocardiography does not show the mass, then a second angiocardiogram may be considered before surgical intervention is advised.

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