Transience of left ventricular thrombosis

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SUMMARY A left ventricular mural thrombus with a mobile appendix of $0.5 \times 0.5 \times 3$ cm was shown by two dimensional echocardiography in a 31 year old man who had dilated congestive myocardial disease. Serial echocardiograms showed formation of the thrombus within three weeks and uncomplicated disappearance of the mobile clot within less than two days. Heparin and phenprocoumon were given in doses generally not accepted to affect left ventricular thrombosis adequately. Such transience of left ventricular thrombosis may be worth considering when deciding whether to treat patients with thrombi accidentally demonstrated by echocardiography; it also might explain some of the many negative examinations in the echocardiographic screening of patients with embolism.

The ability to visualise intracardiac thrombi by means of echocardiography¹⁻³ has not so far led to clear therapeutic guidelines. This is mainly because of incomplete information concerning the natural history, the incidence of embolism, and the effect of various anticoagulants. It is therefore important to gather information from series of patients with intracavitary thrombi⁴ but isolated cases may also contribute to knowledge of the behaviour of cardiac thrombi. The present paper reports a pedunculated left ventricular thrombus with a dramatic appearance of threatening imminent embolism which disappeared in less than two days without embolism occurring.

Case report

A 31 year old previously healthy man was admitted with heart failure which had developed over one week. The electrocardiogram showed sinus rhythm, left anterior hemiblock, and negative T waves in the precordial leads V2-V6. Several attacks of ventricular tachycardia lasting for hours were recorded. Cardiomegaly and pulmonary congestion were apparent from the chest x-ray examination. Echocardiography showed diffuse cavity dilatation and normal myocardial thickness. Left ventricular wall motion was uniformly depressed with a fractional shortening of 20% and a left ventricular end-diastolic diameter of 6.6 cm. The clinical condition stabilised on treatment with digoxin, frusemide, and amiodarone, and the patient was discharged. Because we suspected myocarditis as the underlying lesion the patient was readmitted three weeks later for further examination.

Right and left heart catheterisation with left ventricular angiography were performed and endomyocardial biopsy was carried out guided by fluoroscopy and two dimensional echocardiography. Angiography confirmed the presence of globally reduced wall motion with an ejection fraction of 31%. In addition, it suggested an apical filling defect in the contrast medium. Echocardiography performed in the catheterisation room after introduction of the biopptome into the left ventricle and immediately before left ventricular biopsy showed a flat apical mural mass with an extremely mobile appendix ($0.5 \times 0.5 \times 3$ cm) protruding into the left ventricular cavity (Fig.). To avoid embolism the biopptome was gently removed from the left ventricle and only right ventricular biopsies were subsequently carried out. Later, the biopsies showed non-specific increased fibrosis in the myocardium without signs of inflammation. Anticoagulation with phenprocoumon and low dose heparin (5000 U administered subcutaneously b.i.d.) was initiated. Echocardiography after six hours showed no change, but at repeat examination 48 hours after catheterisation it was impossible to visualise the previously conspicuous mobile process despite careful examination from multiple parasternal, apical, and subxiphoid views. The flat mural mass and the congestive myopathic left ventricle appeared to be unchanged. The prothrombin value at that time was 104% as opposed to 125% immediately before starting treatment with phenprocoumon. The patient had not experienced any unusual symptoms and neurological examination and examination of peripheral pulses, skin, eyes, and mucuous membrane of the mouth showed no signs of embolism.

Fig. Short axis view obtained apically to the papillary muscles. Visualisation of the thrombus required a slightly oblique direction compared with standard short axis views (central drawing). A semilunar mural thrombus (MT) is seen along the posterior wall (PW). The finger shape, floating projection (FT) disappeared during less than two days without clinical embolism, despite an alarming swirling motion which is shown in the left and right panels and indicated by the arrows on the central drawing. R and L, right and left side of the body.

Discussion

An intracardiac mass of the type illustrated is compatible with left ventricular clot as seen in some patients with reduced left ventricular function of various causes.¹⁻⁴ In the present patient, the history and findings indicated congestive cardiomyopathy or myocarditis as the underlying disease. According to the serial echocardiograms, the left ventricular thrombosis had developed within three weeks, but in theory the occurrence of the floating appendix may have been provoked by manipulation of the catheters in the left ventricle. This, however, implies very rapid clot formation. The disappearance of the mobile clot, the most rapid evanescence of cardiac thrombosis so far reported, may have been caused by fragmentation and subclinical microembolisation or by undetectable adherence of the clot to the surrounding ventricular wall. If resolution of the clot took place it must have been spontaneously, since low dose heparin treatment is known not to protect against formation of left ventricular clots.⁴ Though some uncertainty remains regarding the definitive fate of the thrombus, it seems worth noting that a clot with an alarming appearance of threatening imminent embolisation could disappear without clinical symptoms in less than two days and without intensive medical anticoagulation or thrombolysis.

The transience of intracardiac thrombosis, as here reported, is worth consideration in deciding what treatment should be given to patients who, by chance,

have a thrombus demonstrated. It should also be borne in mind in the echocardiographic screening of patients who have experienced an embolic attack, since an ephemeral clot might explain some of the many negative examinations in this setting.^{5,6}

References

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