Left ventricular thrombosis in acute transmural myocardial infarction

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Summary: To determine the incidence and natural history of left ventricular thrombosis in acute transmural myocardial infarction we performed serial two-dimensional echocardiography in 51 patients. Seventeen patients had inferior infarcts. None of these developed left ventricular thrombosis. The remaining 34 patients had anterior infarcts. Ten of these developed left ventricular thrombus at an average of 4 ± 2 days after admission. All patients with left ventricular thrombosis had apical akinesia or dyskinesia.

Patients with anterior myocardial infarction and akinesia or dyskinesia of the apex are at high risk of developing left ventricular thrombosis. Peak aspartate aminotransferase and lactate dehydrogenase enzyme activity were of little value in identifying this high risk group.

Introduction

Mural thrombi represent an important potentially catastrophic complication of acute myocardial infarction and have been detected at autopsy in 20–60% of such patients.¹ We used two-dimensional echocardiography, a reliable non-invasive technique for the detection of left ventricular thrombi,² to determine the incidence and natural history of mural thrombosis in acute transmural myocardial infarction.

Materials and methods

Fifty-one patients admitted to the Heart Care Unit with first acute transmural myocardial infarction were studied prospectively. A diagnosis of myocardial infarction was made when all 3 of the following criteria were met: (a) chest pain for more than 30 minutes; (b) electrocardiographic changes diagnostic of acute transmural myocardial infarction; (c) a serial cardiac enzyme pattern typical of myocardial infarction.

A 12 lead electrocardiogram was performed on admission and daily for the first 3 days as were serum aspartate aminotransferase enzyme activity and total LDH activity. Two-dimensional echocardiography (using a Hewlett Packard 77020A

phased array scanner) was performed within 72 hours of admission and serially during the first week of admission. Left ventricular thrombus was defined as an echo-dense mass in the left ventricular cavity adjacent to asynergic myocardium and distinct from the endocardial surface. The mass was identified in at least 2 planes at different angles. All the management decisions were made by the patients' physicians and none of the study patients received therapeutic anticoagulants before the demonstration of a left ventricular thrombus on echocardiogram. Student-*t* and Fisher exact tests were used for statistical analysis.

Results

Of the 51 patients studied, 17 had inferior infarcts. None of these developed left ventricular thrombosis. The remaining 34 patients had anterior infarcts. Twenty-eight of those were given subcutaneous heparin prophylactically and seven of these went on to develop mural thrombosis. Of the six patients not given heparin, three developed mural thrombi. Left ventricular thrombus was detected at an average of 4 ± 2 days (range 1 to 7 days) after admission. All thrombi detected were localized at the apex. They were broad based, protruding into the lumen and not mobile. Apical akinesia or dyskinesia was present in all patients with left ventricular thrombosis. Full anticoagulation with intravenous heparin followed by an oral

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anticoagulant was given to 8 out of the 10 patients who developed mural thrombosis. Two of these eight patients died in hospital with ventricular arrhythmias. Five of the six remaining patients who received therapeutic anticoagulants showed resolution of their thrombi, and anticoagulants were discontinued at that stage (range 3 to 6 months). The 6th patient still showed evidence of left ventricular thrombosis 13 months later. Two patients did not receive anticoagulants and both had evidence of mural thrombus at 12 months follow-up.

Emboic phenomena occurred in 2 patients with anterior infarcts within the first 48 hours after admission before echocardiography had been performed. At subsequent echocardiography neither of these showed left ventricular thrombosis. No other patients developed embolization during the follow-up period (minimum of 6 months). A comparison of various clinical and laboratory indices (Table I) showed that there was no significant difference between those who developed thrombi, and those who did not, other than the site of infarction.

Discussion

The results of this study are in keeping with others which have shown that left ventricular thrombosis occurs relatively frequently (29% in our study and 28–34% in other studies)^{1,3,5} in patients with

anterior myocardial infarction, particularly those with severe apical dyskinesia or akinesia on two-dimensional echocardiography. Patients with inferior myocardial infarction are at low risk of developing thrombosis in the left ventricle. The incidence of embolic phenomena was 4% of the total group which is consistent with Johannessen's³ findings of an incidence of 5.5%; however, no patient with left ventricular thrombosis who received therapeutic anticoagulants had an embolic episode in our series. Previously reported data have demonstrated a marked reduction of systemic embolization in patients with left ventricular thrombosis who received anticoagulants following myocardial infarction.⁴ These findings support the view⁵ that patients at high risk of developing left ventricular thrombosis cannot be identified by commonly measurable clinical variables other than the electrocardiographic location of the site of infarct. Routine anticoagulation with heparin followed by oral anticoagulants for 1–3 months should, therefore, be considered in patients with acute transmural anterior myocardial infarction as has been recently recommended by the American College of Chest Physicians and the National Heart, Lung and Blood Institute Conference.⁶

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Table I Clinical characteristics of patients with acute infarction

Characteristic	Inferior infarct	Anterior infarct without left ventricular thrombus	Anterior infarct with left ventricular thrombus
Number of patients	17	24	10
Average age (in years)	62 ± 8 (42–71)	60 ± 9 (38–75)	58 ± 8 (49–74)
Sex: male/female	12/5	22/2	8/2
Average Killip class	1.4 ± 0.8	1.3 ± 0.7	1.5 ± 0.5
Number of patients with			
(i) Cardiomegaly	2 (12%)	3 (13%)	2 (20%)
(ii) Heart failure	6 (35%)	11 (46%)	4 (40%)
(iii) Ventricular tachycardia or ventricular fibrillation	5 (29%)	5 (21%)	4 (40%)
Average peak AST (IU/l)	308 ± 156	302 ± 180	307 ± 198
Average peak LDH (IU/l)	878 ± 423	859 ± 405	929 ± 466

AST = aspartate aminotransferase; LDH = lactate dehydrogenase.

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