

Detection of left ventricular thrombi by computerised tomography

A preliminary report

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SUMMARY In order to investigate the potential of computerised tomography in the detection of left ventricular mural thrombi, 16 patients suspected of having left ventricular mural thrombi were studied. All patients had suffered transmural myocardial infarction. Fifteen patients had a ventricular aneurysm. One had had systemic emboli. The mean length of time between the myocardial infarction and the study was 14.8 months, with a range of one month to 79 months. All patients underwent computerised tomography of the heart, M-mode echocardiography (M-mode), and two-dimensional echocardiography (2-D). Eight patients underwent left ventricular cineangiography. Five patients had surgical confirmation. Computerised tomography, two-dimensional, and M-mode echocardiography predicted left ventricular mural thrombi in 10, eight, and one of the 16 patients, respectively. Left ventricular cineangiography predicted left ventricular mural thrombi in four of the eight patients. Both computerised tomography and left ventricular cineangiography correctly predicted the presence or absence of left ventricular thrombi in all five patients who underwent operation. In the same group, however, two-dimensional and M-mode echocardiography failed to predict the presence of thrombi in one and three patients, respectively. Among the 11 patients without surgical confirmation, one, in whom no left ventricular thrombi were shown by M-mode and two-dimensional echocardiography, was found to have thrombi on computerised tomography. In another, two-dimensional echocardiography was positive but this finding was not confirmed either by computerised tomography or by left ventricular angiography. Thus, computerised tomography appears to be a promising non-invasive technique and further studies of larger numbers of patients shown to have left ventricular thrombi at operation are indicated.

At necropsy, the reported incidence of left ventricular mural thrombi is between 20 and 60 per cent.¹⁻⁴ Eighty to 90 per cent of systemic emboli originate in the heart.⁵⁻⁷ With a 3 to 5 per cent incidence of systemic emboli, left ventricular mural thrombi are an important complication of acute myocardial infarction.^{4, 8, 9} A reliable, preferably non-invasive, method of diagnosis would be valuable. Except in a few cases, M-mode echocardiography has proved unsatisfactory.¹⁰⁻¹⁴ It was hoped that two-dimensional echocardiography, with its ability to visualise the entire left ventricle including the cardiac apex, would provide a reliable

method of diagnosis,¹⁵⁻¹⁸ and a number of reports have appeared.¹⁹⁻²² Unfortunately, early expectations have not been confirmed. In a series reported by Silverman and Schiller,¹⁸ two-dimensional echocardiography clearly identified only 50 per cent of cases angiographically and surgically proven. In another series, Meltzer *et al.*²¹ studied 16 patients; their results disclosed two false-negatives and one false-positive.

Computerised tomography is a relatively new diagnostic tool which has been rapidly gaining in popularity in the diagnosis of disorders of the brain and other organs. Computerised tomography gives a complete cross-sectional display without overlap of superimposed structures. The quality of the image is usually excellent because of the lack of

scattered radiation. Very subtle differences in attenuation of x-radiation (and hence density) of structures can be identified. As yet, the technique has not been applied extensively to the heart because of the necessary long scan time enforced by cardiac motion. In the past few years more rapid scanners have been developed. These have brought the era of cardiac computerised tomography closer to realisation. This study reports our early experience in detecting left ventricular mural thrombi using this technique.

Patients and methods

Sixteen patients were studied (Table 1). All had suffered transmural myocardial infarctions (12 anterior and four inferior). The mean length of time between infarction and study was 14.8 months, with a range of one month to 79 months. Fifteen patients were suspected of having left ventricular mural thrombi because of ventricular aneurysms. The remaining patient was suspected of having a mural thrombus because he had had systemic

Table 1 *Clinical, endocardiographic, computerised tomographic, angiographic, and surgical findings*

Case no.	Age/sex	Presentation	Time and type of MI	M-mode echo	Two-dimensional echo	Computerised tomography
1	49/M	Chest pain	3/79—Anterior	Decreased septal excursion; decreased LVF; no thrombus	Anteroapical aneurysm with thrombus	Apical filling defect
2	57/M	Chest pain	9/79—Anterior	Decreased LVF with septal excursion; no thrombus	Anteroapical aneurysm with apical thrombus	Apical filling defect
3	67/M	Chest pain, CHF	8/79—Anterior	LVE with possible apical aneurysm; no thrombus	Anteroapical aneurysm; no thrombus	Apical filling defect
4	51/M	Chest pain	10/79—Anterior	LVE with decreased LVF; no thrombus	Anteroapical aneurysm; no thrombus	No filling defect
5	53/M	Chest pain	10/79—Anterior	LVE with decreased LVF; no thrombus	Anteroapical aneurysm; no thrombus	No filling defect
6	37/M	Chest pain	6/76—Anterior	LVE with possible apical aneurysm; no thrombus	Anteroapical aneurysm with apical thrombus	Apical filling defect
7	45/M	Chest pain	3/77—Anterior	LVE with decreased LVF; no thrombus	Anteroapical aneurysm; no thrombus	No filling defect
8	70/M	CHF, ventricular arrhythmia	10/79—Anterior	LVE with decreased LVF; no thrombus	LVE with diffusely decreased contractility; apical thrombus	No filling defect
9	66/F	CHF	4/73—Anterior	LVE with decreased LVF; thrombus attached to septum	LVE with decreased LVF; apical thrombus attached to septum	Apical filling defect with septal continuity
10	77/M	Cerebral embolus	10/79—Anterior	Decreased septal excursion; no thrombus	Anteroapical aneurysm with apical thrombus extending to septum	Apical filling defects in continuity with septum
11	71/F	CHF, ventricular arrhythmia	10/74—Anterior	LVE with decreased LVF; no thrombus	Anteroapical aneurysm with thrombus	Apical filling defect
12	54/M	CHF, ventricular arrhythmia	11/79—Infero-lateral	LVE with decreased LVF; no thrombus	LVE with diffusely decreased contractility; apical thrombus	Apical filling defect
13	49/M	Chest pain	11/79—Infero-posterior	Decreased posterior wall excursion; no thrombus	Inferior aneurysm; no thrombus	Filling defect over the inferobasal area of left ventricle
14	63/M	Chest pain	11/79—Inferior	Decreased posterior wall excursion	Inferior aneurysm; no thrombus	Filling defect over inferobasal area
15	60/M	Chest pain	11/79—Inferior	Normal findings; no thrombus	Inferior aneurysm; no thrombus	No filling defect
16	78/F	Chest pain	10/79—Inferior	Decreased posterior wall excursion	Inferior aneurysm; no thrombus	No filling defect

CHF, congestive heart failure; Echo, echocardiography; LVE, left ventricular enlargement; LVF, left ventricular function.

started approximately 20 seconds after the injection of contrast media was begun. Six contiguous 1 cm thick slices were obtained through the region of the heart as rapidly as possible. In most cases this took approximately 150 seconds. The injection was finished just before the start of the next to last scan. The images were reconstructed and displayed on a 320×320 matrix. The cardiac structures and mural thrombi were better delineated at the window level of zero with a window width of 150 to 300 (-500 to $+500$ scale). The images were recorded on film by a multifram camera.

Results

The clinical, echocardiographic, cineangiographic, and computerised tomographic features of all patients are summarised in Table 1.

ECHOCARDIOGRAPHY

Technically, optimal M-mode echocardiographic sweeps from apex to the base of the left ventricle were obtained in all patients. Only case 9, however, had multiple, fine echoes in continuity with the interventricular septum thought to be suggestive of mural thrombus. Technically optimal two-dimensional echocardiograms of the left ventricle in various projections were obtained in all patients, though the apex of the left ventricle was not completely visualised in the long and short axis projections. In only two patients could a thrombus be identified in the long axis view. No patient showed evidence of a thrombus in the short axis view. The left ventricular apex was well seen in all patients in the four and two chamber views. These views showed that eight of the 16 patients had mural thrombi. There was septal continuity of the thrombi in two patients. All eight patients were

found to have absent or paradoxical systolic motion of the anteroapical areas.

CINEANGIOGRAPHY

Eight patients underwent cardiac catheterisation. All had technically satisfactory left ventricular cineangiography. Four patients showed apical filling defects consistent with mural thrombi. Septal continuity of thrombi could not be identified in any patient.

SURGERY

Three of the five patients who underwent left ventricular aneurysmectomy had large left ventricular thrombi. All thrombi were apical. In two patients septal continuity was seen. All thrombi were organised with intermingled fresh thrombotic material.

COMPUTERISED TOMOGRAPHY (Fig. 1)

Satisfactory computerised recordings were obtained on the initial attempt from all 16 patients. In 10 patients left ventricular filling defects consistent with mural thrombi were demonstrated. Six had apical defects. Two more had apical defects with septal continuity. Two had inferobasal defects. The presence or absence of left ventricular aneurysms was not well defined.

CORRELATION OF FINDINGS

From Table 2, it is apparent that both cine left ventriculography and computerised tomography correctly predicted the presence or absence of left ventricular mural thrombi in all patients who underwent surgery. In the same group, however, two-dimensional echocardiography failed to predict the presence of a thrombus in one patient (case 3 and Fig. 2), and M-mode echocardiography failed

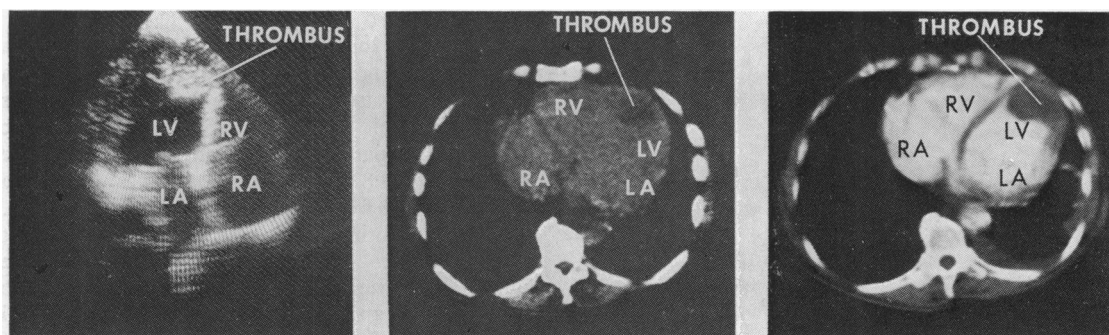


Fig. 1 Two-dimensional echocardiogram (left) and computerised tomogram with (right) and without (middle) contrast enhancement showing left ventricular apical thrombus with septal continuity. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Table 2 Summary of results

Case no.	M-mode echocardiography	Two-dimensional echocardiography	Computerised tomography	Cineangiography	Surgery
1	-	+	+	+	+
2	-	+	+	+	+
3	-	-	+	+	+
4	-	-	-	-	-
5	-	-	-	-	-
6	-	+	+	+	-
7	-	-	-	-	-
8	-	+	-	-	-
9	+	+	+	-	-
10	-	+	+	-	-
11	-	+	+	-	-
12	-	+	+	-	-
13	-	-	+	-	-
14	-	-	+	-	-
15	-	-	-	-	-
16	-	-	-	-	-

+, thrombus detected; -, thrombus not detected.

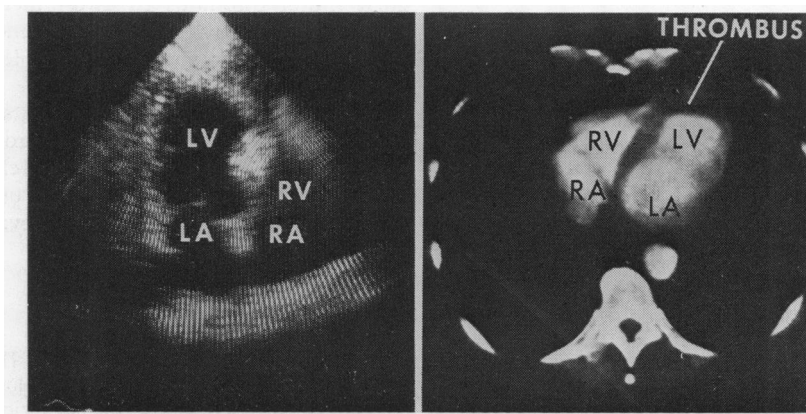


Fig. 2 Two-dimensional echocardiogram (left) and contrast enhanced computerised tomogram (right) of case 3. A distinct left ventricular apical filling defect seen in the computerised tomogram was confirmed by left ventricular cineangiogram and surgery. Two-dimensional echocardiogram failed to show apical thrombus. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

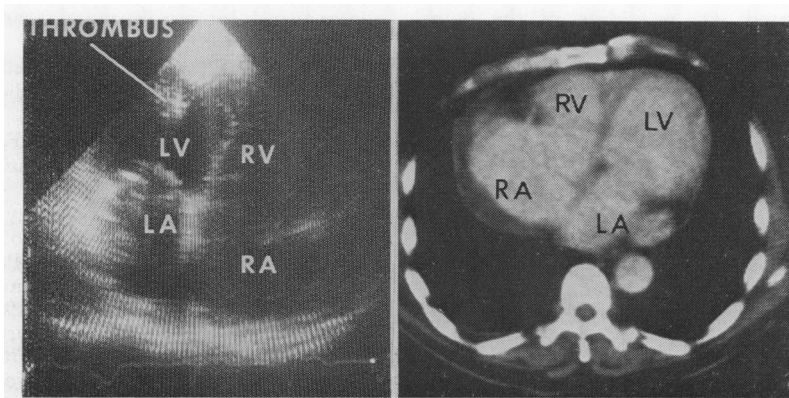


Fig. 3 Two-dimensional echocardiogram (left) and contrast enhanced computerised tomogram (right) of case 8. Note the left ventricular apical mass seen in two-dimensional echocardiogram which could not be confirmed by computerised tomography or left ventricular cineangiography. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

to predict the presence of thrombi in three patients (cases 1, 2, and 3). Among the 11 patients who did not undergo surgery, two (cases 13 and 14) were shown to have left ventricular mural thrombi by computerised tomography but were not identified by either M-mode or two-dimensional echocardiography. Though it is possible that these were false-positive results, it is unlikely since both filling defects were clearly identified by computerised tomography. Two-dimensional echocardiography suggested that one patient who did not undergo surgery (case 8) had a left ventricular mural thrombus but this was not confirmed by either cineangiography or computerised tomography (Fig. 3). Computerised tomographic scanning delineated the filling defects more clearly than either M-mode echocardiography or two-dimensional echocardiography.

Discussion

The sensitive capacity of computerised tomography to resolve small differences in x-ray attenuation makes it potentially attractive as a means of imaging the heart.^{24 25} It has a high degree of reproducibility and requires only a minimal amount of technical skill. The study can be performed rapidly (total time approximately 25 minutes). Initially, the primary problem encountered in *in vivo* imaging of the heart by this technique was the pronounced degradation of the reconstructed image resulting from a three minute scan time and from cardiac motion. In order to obtain high resolution reconstruction, each point of the object being imaged must remain in a fixed position during rotation of the scanner. To avoid problems related to cardiac motion, three solutions have been suggested: (1) reduction of the data collection time per slice to milliseconds; (2) synchronisation of the data collection so that it occurs only at fixed points in the cardiac cycle; and (3) synchronisation of the data with the cardiac cycle after data acquisition. The last method has undergone most development²⁶ and holds the greatest promise. Several investigators^{27 28} have reported studies using this technique but, to our knowledge, none has reported the imaging of left ventricular mural thrombi. Our preliminary study, the results of which are reported here, compared computerised tomography with M-mode echocardiography and two-dimensional echocardiography.

From our results, and those of others, it appears that M-mode echocardiography is insensitive in detecting left ventricular mural thrombi. Though some authors have been able to show that two-

dimensional echocardiography is more useful,^{20 21} not all agree. Ports *et al.*¹⁹ recently reported that they were able to identify only 50 per cent of ventricular thrombi using this technique. In our study, two-dimensional echocardiography identified eight of the 10 patients shown to have left ventricular mural thrombi by computerised tomography. It failed, however, to identify left ventricular mural thrombi in three patients with clearly defined left ventricular filling defects by computerised tomography. One of these patients who underwent surgery was proved to have thrombus. Though it is possible that the results of computerised tomography in the other two patients were false-positives, this is unlikely, since, in both cases, the filling defects were very clearly identified. Finally, one patient was identified as having a left ventricular mural thrombus by two-dimensional echocardiography which was not found by either computerised tomography (Fig. 3) or left ventricular cineangiography.

Hence, this preliminary study suggests that computerised tomography is a promising non-invasive technique for diagnosing left ventricular mural thrombi. A larger study using surgical confirmation will be necessary, however, to validate its usefulness.

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