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Availability of ^{111}In-labeled platelet scintigraphy in patients with postinfarction left ventricular aneurysm

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Eighteen patients with postinfarction left ventricular aneurysms (LVAs) were examined with Indium-111-labeled autologous platelet scintigraphy to identify intracardiac thrombi and to investigate the effect of antithrombotic agents on thrombogenesity within their LVAs. Left ventriculography (LVG), and two-dimensional echocardiography were also carried out to assess the diagnostic ability of the platelet imaging.

Indium-111-platelet scintigraphy for the detection of LVA mural thrombi had a sensitivity of 60% and a specificity of 100%. Four of six patients with false-negative scintigraphic studies had been under antiplatelet therapy. Eight of the nine patients who had showed active platelet deposition on initial examination had not received antiplatelet therapy. Thereafter, five of these nine were treated with tichlopidine (300 mg/day) for 29.8±5.0 days. On the second platelet study, two had resolution and the other three had interruption of intra-aneurysmal deposition, which remained positive. In only one patient of the three, the third platelet study was performed after warfarin therapy. It took two weeks after beginning the therapy to completely interrupt platelet deposition within the LVA in this patient.

ECG gated radionuclide ventriculography and Thallium-201-myocardial scintigraphy were also performed to assess left ventricular wall motion of left ventricular ejection fraction (LVEF) and myocardial blood perfusion.

Thallium-201-SPECT showed apical or anteroapical perfusion defects and the radionuclide ventriculography correctly identified all 18 apical and anteroseptal aneurysms which were confirmed by LVG methods.

The comparison between the thrombus positive group and the thrombus negative group was carried out on both the LVEF and the period from the last myocardial infarction to the initial platelet scanning study. There were no statistical differences in the LVEF and the interval (34.5±12.5% vs 37.3±14.6%, 39.6±52.6 days vs 89.6±108.3 days) between the two groups.

These results suggest that Indium-111-labeled platelet scintigraphy can be a reliable method for the identification of active left ventricular mural thrombi and a practical method of judging antiplatelet and anticoagulant therapy.

Key words: Left ventricular aneurysm (LVA), Indium-111-platelet scintigraphy, Antithrombotic therapy

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INTRODUCTION

VENTRICULAR ANEURYSM is a common complication of transmural myocardial infarction. Autopsy data show an incidence of ventricular aneurysm in pa-

Table 1 Clinical features of patients with left ventricular aneurysms

Case	Name	Sex	Age (Years)	CAG (>75% stenosis)	LVEF (%)	Interval since Last infarct (Days)	Aneurysm site (LVG, Tl-201-SPECT)
1	K.K.	M	62	LAD	51.0	26	Anteroapical
2	K.T.	M	55	LAD	33.3	55	Anteroapical
3	K.S.	M	51	LAD, RCA	55.6	51	Apical
4	Y.S.	M	53	LAD, LCX	36.4	21	Anteroapical
5	T.W.	M	73	LAD, LCX	25.2	24	Anteroapical
6	T.F.	M	47	LAD	26.4	6	Anteroapical
7	M.O.	M	42	LAD, RCX	25.2	165	Apical
8	K.T.	M	57	LAD	22.7	5	Anteroapical
9	M.S.	M	56	LAD	30.0	17	Anteroapical
10	T.W.	M	47	LAD	21.0	140	Anteroapical
11	M.M.	M	62	LAD, RCA	48.9	143	Apical
12	K.T.	M	57	LAD	46.2	33	Anteroapical
13	A.A.	M	27	LAD	8.7	389	Anteroapical
14	K.S.	M	59	LAD	51.4	15	Apical
15	M.M.	M	55	LAD, RCA	36.3	128	Anteroapical
16	C.T.	M	64	LAD, LCX	38.1	40	Apical
17	N.Y.	M	57	LAD, RCA	40.1	39	Apical
18	K.I.	M	57	LAD, RCA	45.2	18	Apical

Age: 54.4±10.1 (years)
LVEF: 35.9±13.0 (%)
One vessel disease: 9 cases
Two vessel disease: 9 cases

tients with myocardial infarction varing from 3.5 to 25%.[1,2] The complications of left ventricular aneurysm (LVA) are chronic congestive heart failure, ventricular tachyarrhythmia, arterial embolization and mural thrombus formation which are found during surgery or autopsy.[2-6] Systemic emboli occur in 6 to 13% of patients with LVA in clinical studies,[5-7] and the intraaneurysmal mural thrombi significantly increases the incidence of thromboembolic phenomena.[1]

To identify the intraventricular thrombi, contrast ventriculography, two-dimensional echocardiography and contrast enhanced X-ray CT have been used successfully. However, contrast ventriculography is invasive and therefore not suitable for repeated studies when seeking information concerning thrombus activity and the efficacy of antiplatelet therapy. Cross-sectional echocardiography is limited by the necessity to differentiate artifacts from true lesions. Furthermore, these methods are not specific for the assessment of thrombus activity. For scintigraphic examination, fibrinogen labeled with Iodine-125 or Iodine-131, and labeled antibodies to fibrinogen or platelets have been developed and applied clinically. But the physical properties of Iodine-125 (low energy) and Iodine-131 (very high energy) severely limit their clinical applicability. Antibodies attached to fibrinogen and platelets carry the risk of reaction to foreign protein, especially when given intravenously.

The labeling method using Indium-111 chelated with oxine, which was first introduced by Thakur et al.,[8] has been modified by a number of investigators to achieve high labeling efficacy without loss of platelet viability. Indium-111-platelet labeling has become widely accepted during the last several years for both scintigraphic imaging and *in vivo* kinetic studies.[8-11]

However, only a few studies have been reported on the clinical availability of Indium-111-platelet scintigraphy in patients with LVAs.[12-14] In this study, the diagnostic availability of Indium-111-platelet scintigraphy was compared with that of other examinations, and its effectiveness in the assessment of thrombus activity was evaluated during the anticoagulant therapy in patients with LVA mural thrombus.

MATERIALS AND METHODS

Patient population (Table 1):
Eighteen patients with LVA were studied, all males, mean age 54±10.1 years (±SD) (range 27–73 years). All underwent routine diagnostic coronary arteriography. Each patient had 75% or more lumina diameter stenosis of at least one major coronary artery. Nine patients had single vessel disease, the other nine had double vessel disease. All had left ventricular aneurysm that showed paradoxical wall motion in the apical or anteroapical wall of the left ventricle. The diagnosis of aneurysm was made with contrast ventriculography, Thallium-201-myocardial SPECT[15] and nuclear ventriculography.[16-19] In 14 patients, persistent ST-segment elevation (defined as

1 mm of ST-segment elevation for 30 days or more after the last myocardial infarction) was present. The interval from the last myocardial infarction to the Indium-111-platelet scintigraphic study varied from 5 days to 389 days. None had clinical evidence of systemic emboli. Among the 18 patients, the presence or absence of LV thrombi was determined by means of contrast ventriculography, cross-sectional echocardiography, and/or contrast enhanced CT study. The LVEF was calculated from the left ventricular time-activity curve constructed from the sequential equilibrium cardiac pool images.

Preparation of Indium-111-labeled-platelet (Fig. 1): With the consent of the patients, 43 m*l* of their whole blood was drawn into a syringe containing 7 m*l* of a solution of acid citrate dextrose (ACD-A) with a 19 guage butterfly needle. The blood was transferred to a sterile 50 m*l* conical plastic centrifuge tube and centrifuged at 200 g for 10 minutes. The upper platelet-rich plasma was transferred with sterile plastic disposable pipettes into 50 m*l* conical plastic tubes, and was acidified to pH 6.5 with ACD-A solution and centrifuged at 2,000 g for 10 minutes. The resultant platelet-poor plasma was saved and the platelet pellet was suspended in ACD-S solution (a solution of ACD-A and saline (1 : 7)) and centrifuged at 2,000 g for 5 minutes. The pH of the ACD-S solution was adjusted to 6.5 with 0.1 N NaOH solution. The supernatant was decanted, and the platelets resuspended in the ACD-S solution were incubated with Indium-111-oxine at 37°C for 3 minutes. The resultant platelet-poor plasma was transferred with sterile plastic disposable pipettes into a sterile conical tube with the labeled platelet suspension. The platelet precipitate was finally resuspended in 10 m*l* of platelet-poor citrated plasma. The platelet labeling efficacy, which was estimated from the activity of the non-cell-bound and cell-bound citrated plasma, was higher than 85% in every patient. The platelet suspension was injected intravenously with a 19-gauge needle. An Indium-111-labeled platelet image was obtained 48 hours after intravenous injection of the platelet suspension. In all patients, over 200,000 count images were obtained. Imaging was performed on a large-field of view gamma camera which was fitted with a medium energy collimator and set on both photopeaks (173 keV 89%, 245 keV 94%) of Indium-111 with a 20% window. All images were interpreted by two radiologists without knowledge of the clinical and laboratory data.

Venous blood 43 m*l*
ACD-A solution 7 m*l*
↓ Centrifugation at 200 g for 10 min
PPP—PRP (platelet rich plasma)
↓ Add 1/20 volume of ACD-A solution
↓ Centrifugation at 1,000 g for 10 min
Platelet pellet
↓ Suspend by 3 m*l* of ACD-S solution
↓ Centrifugation at 1,000 g for 10 min
Platelet pellet
↓ Resuspend by 3 m*l* of ACD-S solution
Add Indium-111 oxine (1 mCi)
↓ Incubate at 37°C for 3 min
↓ Add PPP 6 m*l*
↓ Centrifugation at 1,000 g for 10 min
Indium-111-labeled platelet pellet
↓ Calculation of labeling efficiency
↓ Resuspend in PPP 10 m*l*
Intravenous injection

‡ACD-A: Acid citric dextrose-A
ACD-S: Acid citric dextrosesaline
PPP: Platelet poor plasma
PRP: Platelet rich plasma

Fig. 1 Autologous platelets labeling procedure using 111-Indium chelated with oxine.

Left ventriculography (LVG):
The LVG was carried out in 30 degree right anterior oblique and 60 degree left anterior oblique projections. The criterion for the diagnosis of intracardiac thrombus was the presence of a contrast filling defect in relationship to the aneurysm, usually in both views. A ventricular aneurysm was defined as a protrusion of a localized portion of the external aspect of the ventricle during both diastolic and systolic phases, showing paradoxical wall motion.

Echocardiographic examination:
All patients underwent echocardiogram to check for the presence of mural thrombi. Examination was performed with the patients in the supine or the 30–45 degree left lateral position. Particular attention was directed to areas of dyskinesia. The transducer was placed at the apex of the heart, and the ultrasonic

Table 2 Result of diagnostic examinations from patients with LVA

Case	Diagnosis of thrombi (Initial study)			Therapy (Initial platelet study)
	Platelet imaging	Echocardiography	Left ventriculography	Anticoagulants
1	+		−	Aspirin (300–600 mg/day)
2	+	+	+	ND
3	+	+	+	ND
4	+	+	+	ND
5	+	+	−	ND
6	+	+	+	ND
7	+	+	+	ND
8	+	+	+	ND
9	+	+	+	ND
10	−	+	−	Aspirin (300 mg/day)
11	−	+	+	ND
12	−	−	−	ND
13	−	−	−	ND
14	−	−	−	Dipyridamole (150 mg/day)
15	−	+	−	Tichlopidine (300 mg/day)
16	−	+	+	ND
17	−	+	−	Tichlopidine (300 mg/day)
18	−	+	+	Dipyridamole (150 mg/day)

LVA: Left ventricular aneurysm ND: Not done +: Positive −: Negative ±: Possible

beam was directed toward the right scapula. By this means, an apical four-chamber view (AP4CV) was recorded. With the transducer in the same position, the ultrasonic beam was directed parallel to the intraventricular septum, producing a left anterior oblique equivalent view (APLAO). Only technically adequate echocardiograms were used for diagnostic purposes. Results were then categorized as either positive, possible, or negative.

ECG gated cardiac pool scintigraphy:
Gated cardiac pool scintigraphy was performed by the method previously described.[3,4] Stannous pyrophosphate was administered intravenously and 20 mCi of Technetium-99m-pertechnetate injected to achieve *in vivo* labeling of the red blood cells. Images were obtained in the LAO projection with a gamma camera using a low energy all purpose collimator. LVEF was calculated from the left ventricular volume curve. ECG gated pool SPECT was also performed in order to evaluate regional wall motion three-dimensionally.[17–19]

Myocardial SPECT:
Myocardial SPECT images were acquired after intravenous injection of 4.0 mCi of Thallium-201-chrolide. The acquisition arc was 180 degrees around the patient, and generally 36 images were obtained in an arc extending from 45 degree right anterior oblique to 45 degree left posterior oblique projections, each projection being acquired for 30 seconds. Filtered back projection was then performed with a Shepp-Logan's filter to construct the transaxial tomograms each of 1 pixel thickness (approximately 6 mm). The tomograms were then aligned to the long axis of the heart, and oblique-angle tomograms parallel to the long- and short-axis of the left ventricle were generated from the original transaxial tomograms using a coordinate transformation with commercially available algorithms.[19]

RESULTS

The results of the diagnostic examinations of the 18 patients are summarized in Table 2. Compared with contrast ventriculography and two-dimensional echocardiography for the identification of ventricular thrombi, Indium-111-platelet scintigraphy had a sensitivity of 60 percent (9/15) and a specificity of 100 percent (3/3).

Several typical cases are shown in Figs. 2, 3 and 4. Fig. 2 shows an Indium-111-platelet scintigram, multigated Technetium-99m blood pool ventriculogram and Thallium-201-myocardial scintigram of a 43-year-old male with anteroseptal myocardial infarction and apical aneurysm. In sagittal and four chamber projections, Thallium-201-myocardial SPECT images showed a perfusion defect of the apical wall and divergence of the left ventricular walls approaching the apex. The amplitude and phase images of the first Fourier coefficient calculated from the Technetium-99m blood pool image data

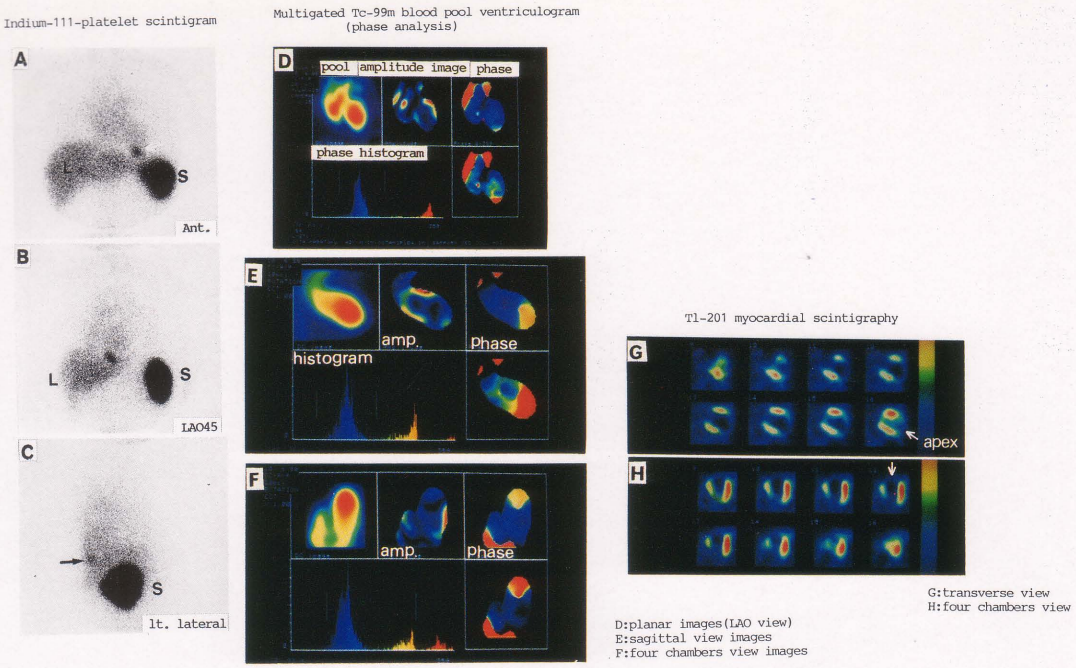

Fig. 2 Anteroseptal infarction, apical aneurysm (M.O. 43-years-old male).
Indium-111 scintigram (A, B, C) shows active mural thrombus in the apical region of the left ventricle. In this case which had 95% luminal diameter stenosis of the left descending artery and 75% luminal diameter narrowing of the right coronary artery on arteriography, Tl-201 myocardial SPECT scans (G, H) show perfusion defect of apical wall and divergence of the LV walls. The amplitude image and phase image of the first Fourier coefficient calculated from the Tc-99m blood pool image data show marked LV enlargement and dyskinesis in the infarct apical wall (D, E, F).

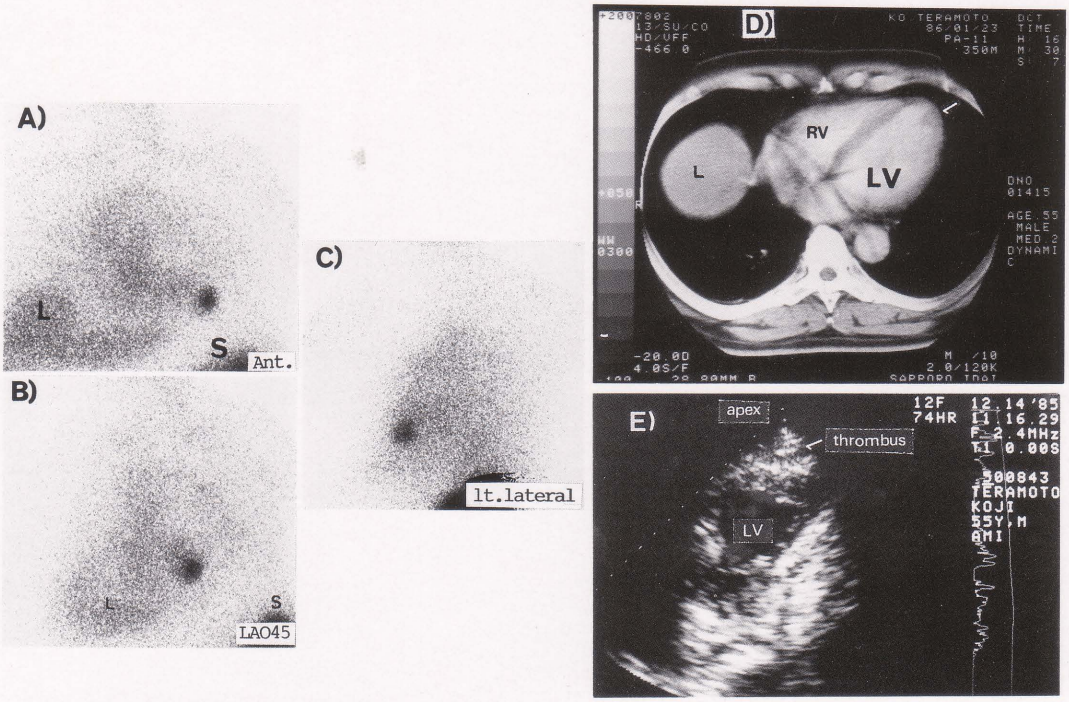

Fig. 3 Broad anterior infarction, apical aneurysm (K.T. 55-year-old male).
Indium-111-labeled scintiphoto, CT and echocardiography reveal mural thrombus in the apical region of the LV (Indium-111 platelet scan: A; Anterior view, B; Left anterior oblique view, C; Left lateral view). After these examinations, antiplatelet therapy (Tichlopidine; 300 mg/day) was performed.

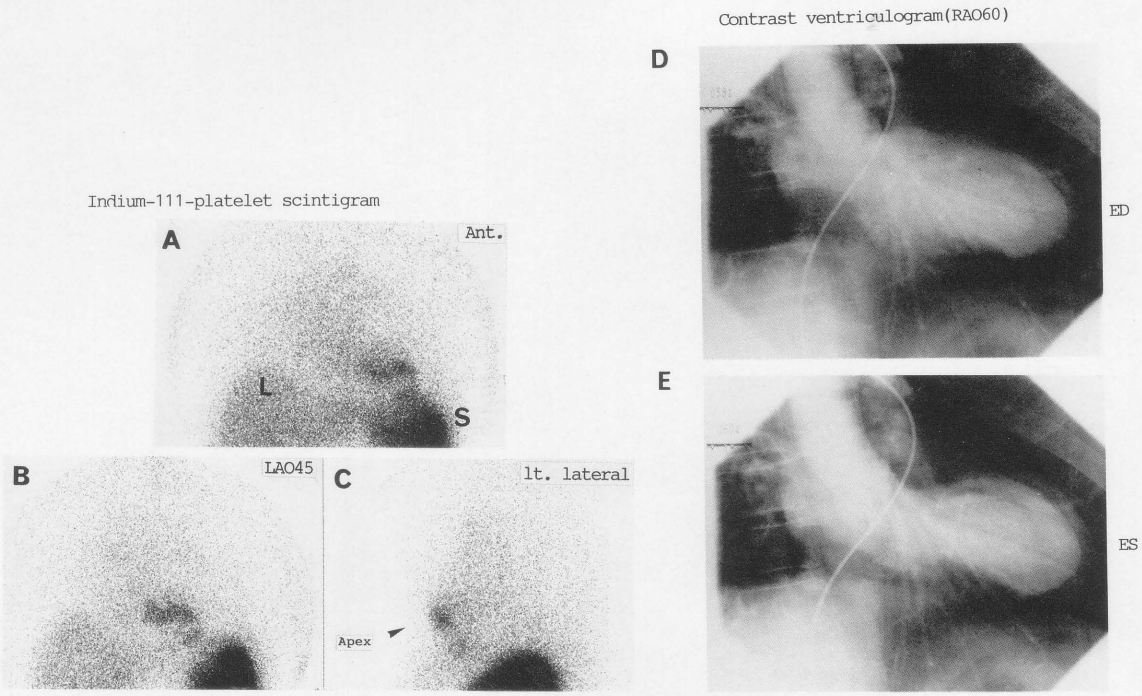

Fig. 4 Broad anterior infarction, left ventricular aneurysm (K.T. 57-year-old male). Indium-111 scintigram obtained 48 hours after injection of the labeled platelet suspension shows active thrombus in a large apical aneurysm (panels A, B and C). Contrast ventriculogram (D: End-diastolic phase, E: End-systolic phase) shows apical aneurysm and mural thrombus, L: Liver, S: Spleen.

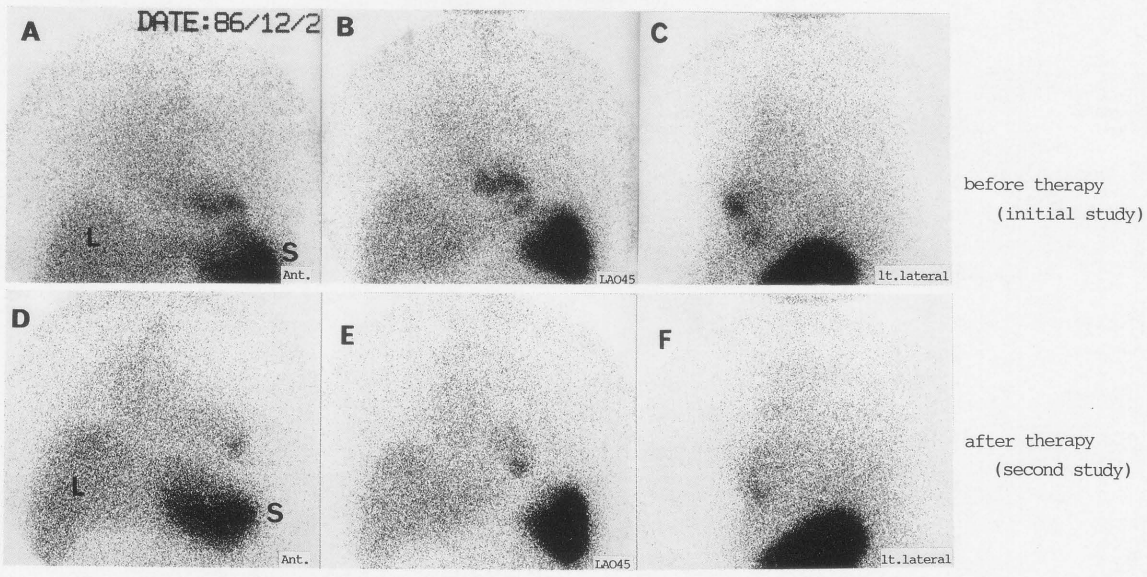

Fig. 5 Indium-111-labeled platelet scintigram obtained 48 hours after injection of the platelet suspension. After anticoagulant therapy (Tichlopidine; 300 mg/day), indium-111 activity in a large anteroapical LVA was significantly reduced (panels: D, E and F) compared with that of scintiphotos (frames A, B and C) before the therapy. L: Liver, S: Spleen (K.T. 57-year-old male)

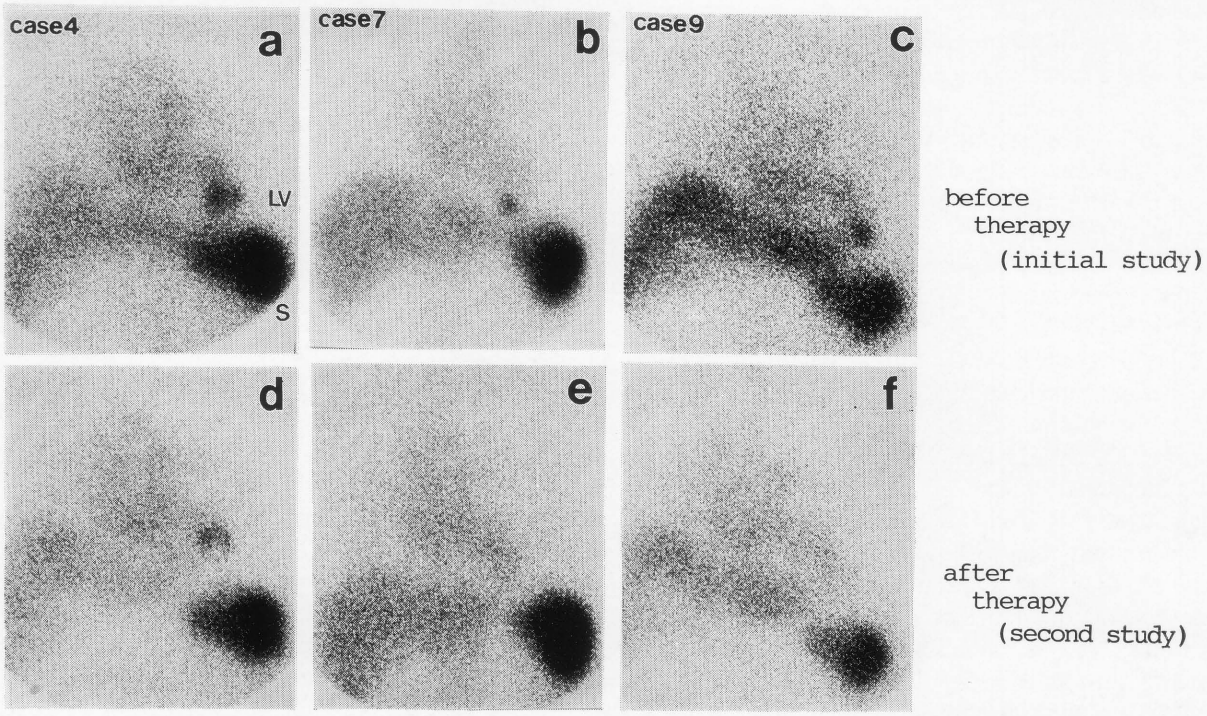

Fig. 6 Indium-111-labeled scintigrams (Cases of 4, 7 and 9). After antiplatelet therapy, indium-111 activity within LVA (panels: a, b and c) showed a marked decrease compared with that of scintigrams (panels: d, e and f) obtained before the therapy. L: Liver, S: Spleen, LV: Left ventricle.

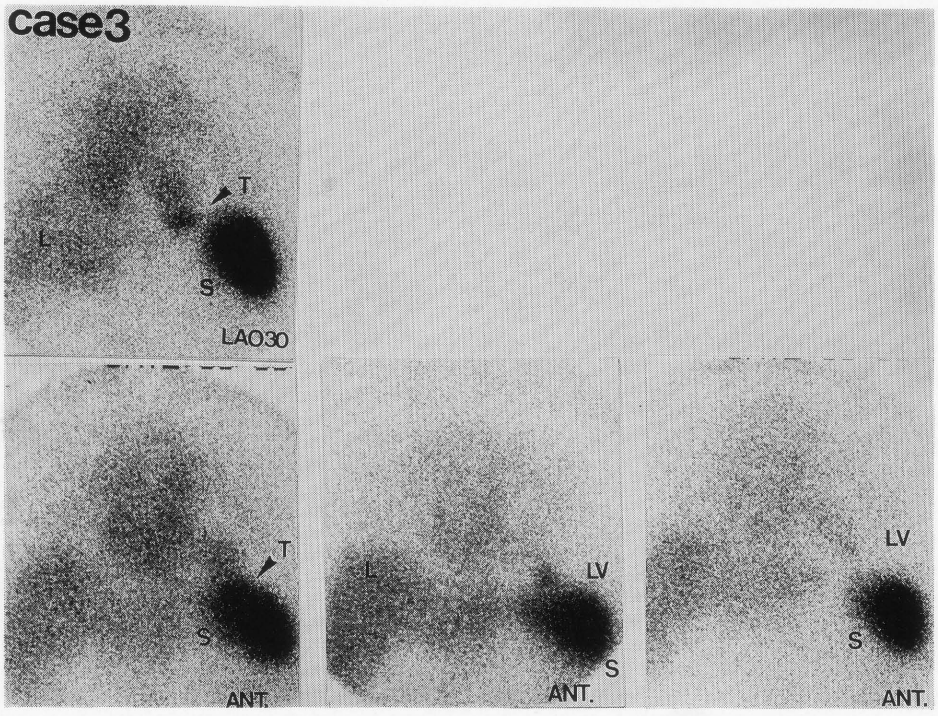

Fig. 7 During tichlopidine therapy (300 mg/day), platelet deposition in an apical thrombus continued positive. The study, 2 weeks after beginning warfarin (5 mg/day), showed complete inhibition of platelet deposition (case 3).

showed marked left ventricular enlargement and a phase delay in the apical area of the left ventricle. The blood pool tomographic study delineated the extent of the aneurysm better than the planar study. Indium-111 scintigrams (Ant., LAO45 and left lateral views) revealed active mural thrombus in the apex.

In the case of broad anterior infarction, Indium-111-platelet scintigraphy, contrast enhancement CT and two-dimensional echocardiography revealed a mural thrombus in the apical region of the left ventricle (Fig. 3).

The highest platelet deposition was observed within the left ventricular aneurysm shown on contrast ventriculography. After antiplatelet therapy (tichlopidine: 300 mg/day for 38 days), Indium-111 activity in a large anteroapical left ventricular aneurysm was significantly reduced (Fig. 4, 5).

In six patients with false-negative scintigraphic studies, four had been under antiplatelet therapy (aspirin, dipyridamole or tichlopidine). To examine the effect of antithrombotic therapy on intraaneurysmal thrombogenesity in a qualitative manner, Indium-111-platelet studies were carried out before and after the therapy. In the five patients studied on tichlopidine (300 mg/day for 29±27 days), three platelet scans became negative and in two cases intraaneurysmal deposition was decreased (Figs. 5, 6 and 7).

Echocardiography showed that thrombus size was unchanged in these patients. In the one patient (case 3) whose platelet scan continued positive after tichlopidine therapy (for 3 weeks), it became negative at 2 weeks after beginning warfarin (5 mg/day) (Fig. 7).

LVEF and the intervals from the last infarct to the initial platelet study were compared in the thrombus-positive and thrombus-negative groups in platelet-imaging. LVEF and the intervals were 34.5± 12.5% and 39.6±52.6 days for thrombus-positive patients vs 37.3±14.6% and 89.6±108.3 days for thrombus-negative patients, respectively. There are no significant differences between these two groups (results mean±SD) (Table 3).

Table 3 Platelet imaging in 18 patients with LVA

Platelet imaging	LVEF (ECG gated blood pool scan) (%)	Internal from infarct to platelet study (days) (Initial study)
Positive (9 cases)	34.5±12.5	39.6±52.6
	N.S.	N.S.
Negative (9 cases)	37.3±14.6	89.6±108.3

LVA: Left ventricular aneurysm

DISCUSSION

Left ventricular thrombi are a prime source of systemic embolization in patients with transmural myocardial infarction. Intraaneurysmal thrombosis is common, and the incidence of systemic embolism is greater than that in all patients with healed myocardial infarction. Emboli may cause significant morbidity and even death.[21] Treatment should be based on the prevention of systemic thromboembolism, so identifying mural thrombi is clinically very important.[22] Venous thrombi are red thrombi in their mature stages and consist primarily of a red fibrin network. On the other hand, arterial thrombi ordinarily are white thrombi consisting chiefly of platelets and a small amount of fibrin network and red cells. Since the initial report by Thakur et al.[18] held that human platelets could be labeled with a lipid soluble complex of the radioactive indium without impairment of platelet function, arterial thrombi can be visualized noninvasively.

Ezekowitz et al.[9,11] reported that left ventricular thrombi frequently demonstrated sufficient uptake of Indium-111-platelet to permit *in vivo* imaging and that the availability of this approach was subsequently compared to two-dimensional echocardiography. They also examined whether left ventricular thrombi were present or absent during surgery or autopsy among 34 patients with left ventricular aneurysm. Stratton et al.[10] reported similar results in patients with cardiomyopathy or transmural myocardial infarction. From these studies, they showed that active left ventricular thrombi could be readily identified by Indium-111-labeled platelet scintigraphy and this technique might not only define the location of the thrombus, but also reflect its activity. Ezekowitz et al. studied 11 patients with intracardiac thrombi 5 patients were treated with aspirin, in doses of 300–2,400 mg/day, and platelets continued to be incorporated into these thrombi. Vreeken et al.[13] reported that there was no apparent change in platelet deposition in ventricular thrombi upon medication with anticoagulant.

These findings suggest that aspirin alone does not completely interrupt the platelet accumulation within left ventricles. Theoretically, because the increased Indium-111 activity reflects an active thrombus surface and not the mass of the thrombus, it is predicted that patients with positive results on platelet scintigraphy will have a higher incidence of embolism. In follow-up studies, a marked decrease in platelet deposition within their aneurysms was observed in 5 patients (case 3, 4, 7, 8, 9) after treatment with an oral anticoagulant (300 mg/day of tichlopidine). A serial study was performed in one (case 3) of these

five patients after the drug was stopped. The uptake of labeled platelet become negative 2 weeks after beginning warfarin in this patient. The effects of warfarin on patients with left ventricular aneurysm developed after myocardial infarction have scarcely been reported. Since warfarin acts predominantly on the inhibition of fibrin polymerization, it is suggested that the formation of a thrombus within a left ventricular aneurysm is partially fibrin dependent.

LVEF and the interval from the last infarct to the initial platelet study were investigated in patients with positive or negative platelet scans, in order to study the factors affecting thrombus formation within LVAs. There was no significant difference in LVEF or the interval between two groups. No absolute conclusion was reached on the importance of these two thrombogenic factors, but the thrombus positive group had lower LVEF and a shorter interval since the last infarct than those in the thrombus negative group. These findings suggest that the thrombi of patients with remote myocardial infarction are chronic, and that the more rapid changes in thrombogenesity within LVAs may cause thrombi in patients with acute myocardial infarction. Because systemic emboli occur in 6 to 13% of patients with LVA, the relationship between the results of platelet scintigraphy and clinically important systemic emboli should be studied in a larger series. In addition, there is neither morbidity nor any known harmful biologic effect resulting from this procedure. This technique provided the most reliable information regarding to the thrombi in the patients with left ventricular aneurysms and the effect of anticoagulant therapy.

CONCLUSION

1. The overall accuracy of Indium-111-platelet scintigrams for the detection of left ventricular thrombi was 66.7% compared with cross-sectional echocardiography. There were six false-negative images, and five of them were treated with aspirin, tichlopidine or dipyridamole. These techniques are complement each other and represent the best examination procedure currently available for the diagnosis of LVA thrombi.

2. Intraaneurysmal thrombosis is common and the tendency toward systemic thromboembolism is great. In this study, 9 of 18 cases (50%) had positive findings of thrombi within their aneurysms on Indium-111-platelet imaging but a history of systemic embolism had occurred in these patients under treatment with oral anticoagulants. Presumably, inhibition of activity on platelet imaging may be associated with decreased embolic risk.

3. Indium-111-platelet studies before and after the therapy were carried out in 5 patients. Tichlopidine and warfarin inhibited platelet deposition in these patients. To prevent systemic thrombo-embolism, follow-up studies to identify mural thrombi within left ventricular aneurysm by Indium-111-platelet scintigraphy will be widely used in the future.

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