Indium-111-Antimyosin Antibody Imaging for Detecting Different Stages of Myocardial Infarction: Comparison with Technetium-99m-Pyrophosphate Imaging

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The diagnostic value of ^{111}In-antimyosin (AM) imaging for identifying myocardial infarction was evaluated in comparison with ^{99m}Tc-pyrophosphate (PPi) imaging. Twenty-four patients with various stages of myocardial infarction, ranging from three days to nine months after the onset of infarction, underwent both AM and PPi scans. Of 26 infarct lesions AM scan identified 22 (85%), while PPi scans detected 10 (38%) ($p < 0.01$). When less than a week had passed since the onset both scans demonstrated all infarct lesions. For seven subacute lesions studied within one to two weeks of onset, AM scans detected (100%), while PPi scans identified only 2 (29%). Furthermore, AM scans showed discrete myocardial uptake in 7 (64%) of those studied more than two weeks after onset. The intensity of AM uptake in the infarcts studied more than seven days after onset was less than that in acute infarcts studied within seven days of onset ($p < 0.05$). These preliminary data indicate that the abnormal myocardial uptake of AM persists beyond the first two weeks when PPi no longer accumulates. Thus, AM scans can be considered to provide a sensitive diagnosis of subacute as well as acute myocardial necrosis.

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Infarct avid scintigraphy may provide valuable diagnostic and prognostic information for the management of patients with acute myocardial infarction. Acute infarction can be identified with ^{99m}Tc-stannous pyrophosphate imaging (*1–5*) or antimyosin antibody imaging (*6–11*). Since these radiopharmaceuticals localize in myocardial necrosis by different mechanisms, the uptake pattern in myocardial infarction may be substantially different. Khaw et al. compared these tracers in animals with acute infarction (*12*), as well as in patients (*8*). Johnson et al. (*10*) in a multicenter trial described the clinical values of ^{111}In-antimyosin for detecting myocardial necrosis. All of these studies were undertaken in acute infarcts within 72–96 hr after infarction. To determine if sites of acute necrosis can still be identified days to weeks after the acute episode with ^{99m}Tc-pyrophosphate and ^{111}In-antimyosin, we injected these radiopharmaceuticals on the same day to assess both tracer distributions in patients at various times after myocardial infarction.

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MATERIALS AND METHODS

Patient Population

Twenty-four consecutive patients with myocardial infarction who received both ^{99m}Tc-pyrophosphate and ^{111}In-antimyosin on the same day were selected for this study. The study group included 20 men and 2 women ranging from 36 to 80 yr old (mean: 59 yr old). All patients gave written informed consent approved by the hospital human study committee. Myocardial infarction was diagnosed based on precordial chest pain of at least 30-min duration, ST segment elevation in two or more leads of an ECG and significant elevation of creatine kinase. Eight patients were imaged within the first week after chest pain, seven within 1–2 weeks and the remaining nine patients after the first two weeks of onset. None of them had recurrent chest pain or elevation of cardiac enzymes between the acute episode and the radionuclide study. There were no reinfarct patients. Ten of them had received successful PTCA therapy in the acute stage of myocardial infarction.

Radiopharmaceuticals

Each patient was injected intravenously with 555–740 MBq (15–20 mCi) of ^{99m}Tc-pyrophosphate. All patients had an intradermal skin test with 0.05 mg of diethyltriamine pentaacetic acid (DTPA)-antimyosin prior to intravenous admin-

istration of ^{111}In-antimyosin. There were no wheal and flare reactions to the skin test. Antimyosin Fab (R11D10, Centocor) 0.5 mg, conjugated to DTPA, and labeled with 74 MBq (2 mCi) of ^{111}In- was injected intravenously.

Protocol

Three hours after injection of ^{99m}Tc-pyrophosphate, planar images were obtained in the anterior, 45° and 70° left anterior oblique, and left lateral views for 5–7 minutes. Three-hundred to five-hundred kilo counts using a low energy high resolution collimator were collected. Immediately after the pyrophosphate scan, ^{111}In-antimyosin was administered slowly over 30–60 sec. An antimyosin scan was performed 48 hr later (Fig. 1). Planar images were obtained with the same projections as described for the pyrophosphate scan. Each acquisition lasted 7 min. collecting 300–500 kilo counts with use of both photo peaks of ^{111}In- (174 and 247 KeV) and a medium-energy general-purpose collimator.

These procedures permitted injection of both tracers on the same day after the onset of infarction and images were obtained without significant cross talk from the two radionuclides. Thus, there was no demonstrable interference of ^{111}In to ^{99m}Tc and vice versa.

Image Analysis

Technetium-99m-pyrophosphate and ^{111}In-antimyosin images were displayed on a computer with 256 by 256 matrices without background subtraction and independently interpreted by two experienced observers without knowledge of the clinical data. The intensity of the tracer concentration in the myocardium was graded from zero to 4+. Zero – no definite uptake in the heart; 1+ – diffuse faint uptake in the heart; 2+ – discrete myocardial uptake with less intensity than that of liver (antimyosin) or bone (pyrophosphate); 3+ – discrete myocardial uptake with similar intensity as that of liver or bone; 4+ – discrete myocardial uptake with higher intensity than that of liver or bone (*2,10*). The uptake grades 2+, 3+ or 4+ were considered positive, while the grades zero or 1+ were considered negative in this study. When the interpretation of the two observers were discordant, a consensus reached after discussion was used as the final interpretation.

Statistical Analysis

The chi-square test or Fisher's exact test were used to determine the significance of difference in rates of occurrence.

RESULTS

The interval from the onset of myocardial infarction to the tracer injection ranged from three days to nine months. Of the 24 patients with myocardial infarction, ^{111}In-antimyosin showed positive uptake in the infarcted area in 22 patients (92%) and ^{99m}Tc-pyrophosphate showed positive uptake in 10 patients (42%) ($p < 0.01$) (Table 1). The two patients showing negative antimyosin uptake had their acute infarct one month and two months before the scan, respectively. The 13 patients showing negative pyrophosphate uptake had their myocardial infarct more than eight days prior to the scan. There were 12 patients showing positive anti-

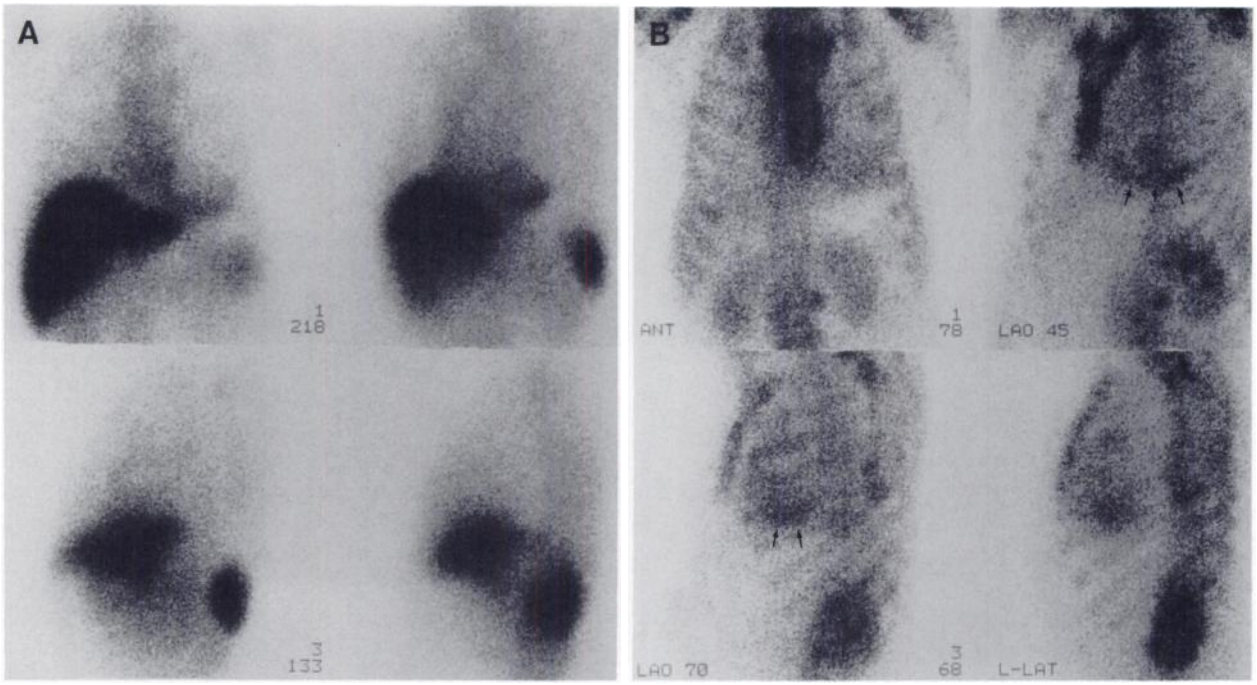

FIGURE 1
Indium-11-antimyosin (A) and ^{99m}Tc pyrophosphate (B) images of a patient seven days after the onset of inferior infarction. Discrete intense uptake in inferior wall is observed on antimyosin scan (3+), while pyrophosphate scan showed faint but discrete uptake in the same area (2+) (arrows). Note that the area of antimyosin uptake is slightly smaller than that of pyrophosphate.

TABLE 1

Results of ^{111}In-Antimyosin (AM) and ^{99m}Tc-Pyrophosphate (PPi) Imaging in 24 Patients with Myocardial Infarction

No	Age/Sex	ECG location	CAG finding	Onset-injection	AM uptake	PPi uptake
1	55/F	anterior	LAD	2 days	2+	2+
2	52/M	inferior	LAD, CX (post PTCA)	3 days	3+	3+
3	60/M	anterior	LAD (post PTCA)	4 days	3+	4+
4	68/M	anterior	not done	4 days	3+	2+
5	60/M	inferior	RCA	5 days	3+	3+
6	61/M	inferior	RCA	6 days	3+	2+
7	47/M	inferior	CX (post PTCA)	7 days	3+	2+
8	80/F	lateral	not done	7 days	2+	2+
9	36/M	anterior	LAD	8 days	2+	2+
10	64/M	inferior	RCA, CX (post PTCA)	8 days	2+	(1+)
11	59/M	inferior	CX (post PTCA)	10 days	2+	2+
12	63/M	anterior	LAD	10 days	2+	(1+)
13	63/M	inferior	RCA (post PTCA)	11 days	2+	(0)
14	65/M	lateral	LAD, CX	11 days	2+	(0)
15	72/M	inferior	RCA, CX (post PTCA)	12 days	2+	(0)
16	66/M	lateral	CX	22 days	2+	(0)
17	59/M	anterior	LAD	24 days	2+	(0)
18	60/M	anterior	LAD	1 mo	(1+)	(1+)
19	55/M	inferior	RCA, LAD	1 mo	2+	(1+)
		anterior		8.5 yr	(0)	(1+)
20	51/M	anterior	LAD	2 mo	2+	(1+)
21	43/M	inferior	normal	2 mo	(0)	(0)
22	59/M	anterior	LAD (post PTCA)	3 mo	2+	(0)
23	51/M	anterior	LAD (post PTCA)	4 mo	2+	(0)
		lateral	CX (post PTCA)	8 mo	(0)	(0)
24	59/M	anterior	LAD (post PTCA)	9 mo	2+	(1+)

RCA = right coronary artery; LAD = left anterior descending artery; CX = circumflex artery.

myosin uptake but no pyrophosphate uptake in the infarcted myocardium. On the other hand, there were no patients showing negative antimyosin uptake with positive pyrophosphate uptake in the infarcted myocardium (Table 1).

Two patients with negative antimyosin uptake had two different infarcted lesions. Among 26 lesions, ^{111}In-antimyosin scan identified 22 infarcted lesions (85%), while ^{99m}Tc-pyrophosphate scan detected 10 (38%) ($p < 0.001$). Ten of them received successful PTCA in their acute stage of infarction.

When the infarcted lesions were classified into three groups in the course of myocardial infarction (Table 2), both antimyosin and pyrophosphate scans showed positive uptake in the infarcted lesions within the first week after onset, as illustrated in Figure 1. Of seven infarcted lesions with one to two weeks after onset, the antimyosin scan showed positive uptake in all of them (100%), while the pyrophosphate scan demonstrated positive uptake only in two of them (29%) ($p < 0.01$) (Fig. 2). Of 11 infarcted lesions beyond the first two weeks, the antimyosin scan remained positive in 7 (64%) but the pyrophosphate scan did not show positive uptake in any of them (0%) ($p < 0.005$) (Fig. 3). There were four patients studied with two months or more after onset who showed persistent uptake of antimyosin. All four had anterior wall myocardial infarction with coronary stenosis of 90% or less in diameter. Three of them received successful PTCA therapy. A patient with a 9-mo old infarction also showed faint but discrete myocardial uptake of antimyosin in the infarcted area (2+) (Fig. 4). This patient had PTCA in the left anterior descending artery in the acute stage of infarction. He did not have any recurrent chest pain after the onset.

High intensity uptake (3+ or 4+) of the tracer concentration was observed in six infarcted lesions on the antimyosin scan and three lesions on the pyrophosphate scan (Table 3). All of these patients had been imaged within one week of onset of infarction. Those with a subacute stage of infarction, beyond the first week, showed relatively less uptake (2+). Thus high intensity of antimyosin uptake was observed in the acute stage of infarction more often (75%) than in subacute stage of infarction (0%) ($p < 0.05$) (Table 3).

TABLE 2
Sensitivity for Detecting Infarcted Myocardium by ^{111}In-Antimyosin (AM) and ^{99m}Tc-Pyrophosphate (PPi) in Relation to the Interval from the Onset of Infarction

Interval	AM	PPi	Significance
0–1 wk	8/8 (100%)	8/8 (100%)	ns
1–2 wk	7/7 (100%)	2/7 (29%)	$p < 0.01$
2 wk	7/11 (64%)	0/11 (0%)	$p < 0.005$
Total	22/26 (85%)	10/26 (38%)	$p < 0.001$

DISCUSSION

These data indicate that ^{111}In-antimyosin imaging can be used to detect not only the acute infarcts within the first week after the onset but also subacute lesions, although antimyosin intensity in infarcts in this stage was not as high as that in the acute stage. This persistent uptake of antimyosin enhances the value of this scan both in the early detection of myocardial infarction and the evaluation of the patients who present late in the course of disease. In addition, the antimyosin scan can be performed in later stages of infarction to detect and localize infarcted myocardium when the patient becomes more stable. Thus, it is useful for retrospective evaluation of myocardial infarction. On the other hand, positive uptake of antimyosin does not necessarily indicate the presence of an acute episode of myocardial infarction.

Indium-111-antimyosin has been considered a sensitive and specific method for detecting and localizing acute myocardial infarction (*6–12*). All the experimental and clinical studies have been undertaken in the acute stage of myocardial infarction (*6–12*). A previous comparative study of antimyosin and pyrophosphate for detecting myocardial infarction also was limited to cases in the acute stage (*8,13*).

The persistence of antimyosin uptake in the infarcted myocardium may reflect prolonged breakdown of myosin in the myofilaments and the myosin persistently exposed to extracellular fluid. The previous experimental study suggested persistence of extractable myosin from infarcted tissue up to 30 days after the onset of infarction (*14*). The study of radioimmunoassay for human cardiac myosin light chain showed relatively slow peak (2–4 days) in its serum concentration after the onset of myocardial infarction and remained elevated particularly in patients with large myocardial infarction (*15,16*). Serum levels of a ventricular heavy chain also remained elevated for as long as one week after myocardial infarction (*17*). These in vitro studies support our in vivo results indicating a prolonged breakdown of myosin after myocardial infarction. In addition, an experimental study of viral myocarditis showed high and persistent uptake of antimyosin in the myocardium for 2–4 wk, while pyrophosphate uptake was observed for no more than one week after inoculation. These studies suggest that a prolonged breakdown of membrane integrity and retention of myosin in extracellular fluid occurs after myocardial damage. The ongoing necrosis may possibly cause persistent uptake of antimyosin in the infarcted myocardium. However, it

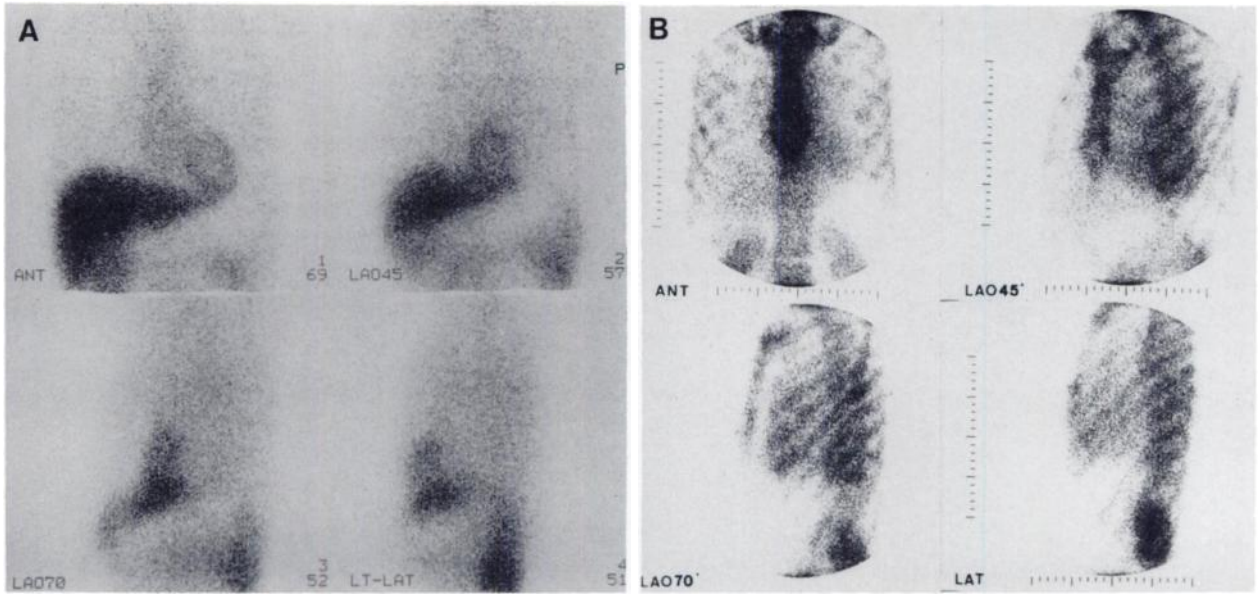

FIGURE 2
Indium-111-antimyosin (A) and ^{99m}Tc-pyrophosphate (B) images of a patient 10 days after the onset of anterior infarction. Discrete uptake of antimyosin (2+) is observed in anterior and septal regions, while pyrophosphate scan shows only diffuse faint uptake in the cardiac region (1+).

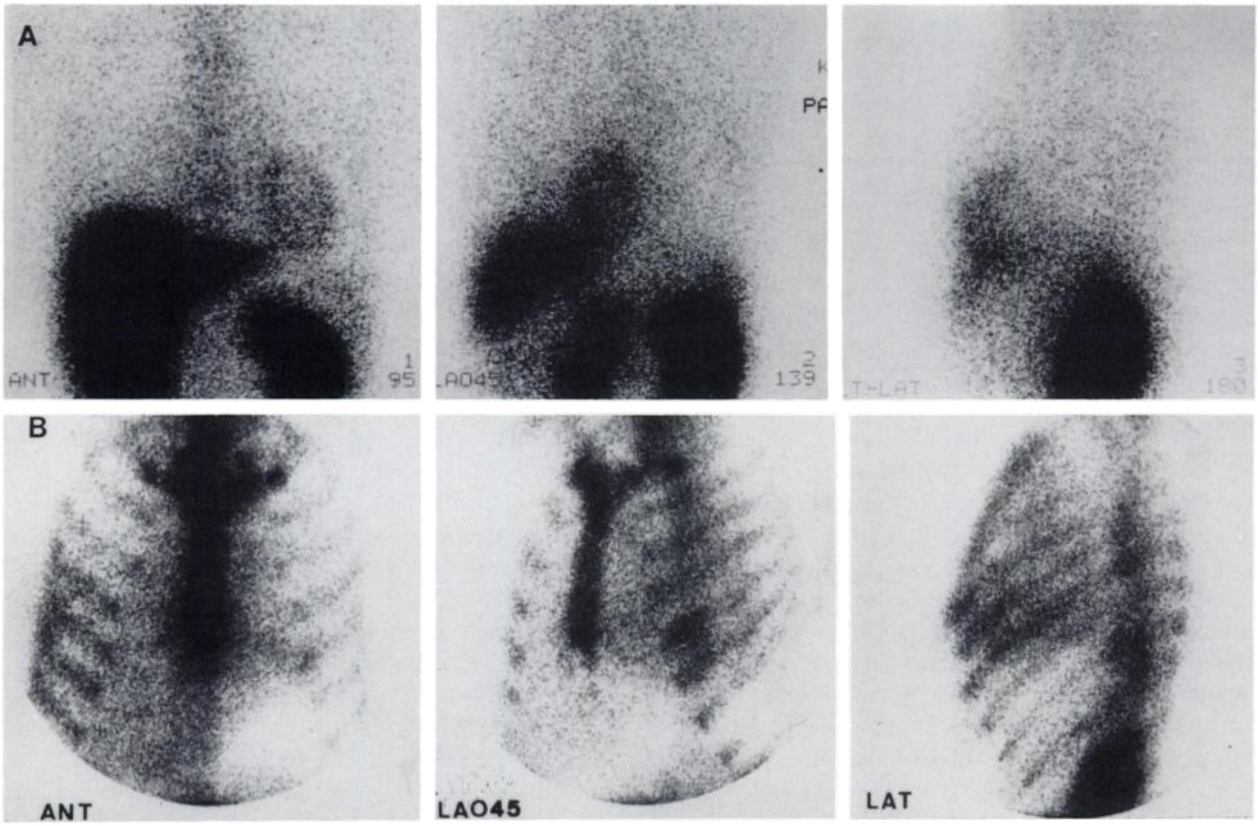

FIGURE 3
Indium-111-antimyosin (A) and ^{99m}Tc-pyrophosphate (B) images of a patient 2 mo after the onset of anterior infarction. Discrete uptake of antimyosin (2+) is observed in anterior and septal regions where pyrophosphate scan does not show any discrete myocardial uptake (1+).

may be less likely since no patients showing persistent uptake had prolonged elevation of cardiac enzymes or complained of persistent precordial symptoms.

Radiolabeled antimyosin and pyrophosphate are two infarct localizing agents with apparently different mechanisms of localization. Antimyosin monoclonal antibody binds to myosin exposed to extracellular fluid by virtue of loss of membrane integrity in infarcted myocardium (*6,7*). Pyrophosphate is considered to coprecipitate with calcium complex in the damaged myocardium, particularly in the irreversibly damaged mitochondria (*1,3*). The localization of pyrophosphate in the infarct begins 12–16 hr following infarction, persists for 4–6 days, and disappears by 2 wk (*2,3*). Our data were consistent with the previous reports. The early disappearance of myocardial uptake may limit the value of pyrophosphate scan to the identification of the infarct. However, Olson et al. reported several patients with persistently positive uptake of pyrophosphate following acute myocardial infarction (*20*). The majority of patients with a persistent positive scan had poor prognosis with fulminant clinical course. In addition,

FIGURE 4
Indium-111-antimyosin images in anterior (left) and LAO (right) projections of a patient 9 mo after the onset of anterior wall infarction. Faint but discrete uptake (2+) is noted in anterolateral wall.

TABLE 3
Intensity of ^{111}In-Antimyosin (AM) and ^{99m}Tc-Pyrophosphate (PPi) Uptake in the Infarcted Myocardium in Relation to the Interval from the Onset of Infarction.

	AM			PPi		
Interval	2+	3+	4+	2+	3+	4+
0–1 wk	2	6	0	5	2	1
1–2 wk	7	0	0	2	0	0
2 wk	7	0	0	0	0	0
Total	16	6	0	7	2	1

pyrophosphate uptake is observed in injured but viable myocardium as well (*21–24*). In their experimental and clinical studies, Khaw et al. demonstrated overestimation of infarct size by pyrophosphate in comparison with areas of antimyosin uptake or histochemical infarct size (*8,12*). Since there were only 10 patients showing positive uptake of both antimyosin and pyrophosphate in this study, the extent of uptake was not precisely assessed. However, a few cases demonstrated a smaller area of antimyosin uptake than that of pyrophosphate uptake, as shown in Figure 1, supporting the previous reports (*8,12*).

The intensity of antimyosin uptake in the infarcted myocardium may reflect the amount of antimyosin in the tissue. Johnson et al. in a clinical study of acute myocardial infarction demonstrated that intensity correlated with infarct location and the presence or absence of collateral vessels (*10*). In our study, all patients with subacute stage of myocardial infarction beyond the first week after onset had less intense uptake of antimyosin as compared to those with acute infarction, indicating gradual decrease in antimyosin binding sites in the course of myocardial infarction.

Four patients studied with two months or more after the onset of infarction showed persistent uptake of antimyosin. The precise mechanism remained unknown. None of these four had a coronary occlusion of his angiogram. These patients had large anterior wall myocardial infarction but showed relatively faint uptake (2+), indicating a relatively small number of binding sites in the extracellular fluid. To verify this preliminary finding and to assess its clinical and prognostic significance, a greater number of patients with subacute and chronic stages of myocardial infarction should be investigated with ^{111}In-antimyosin. In addition, sequential follow-up studies of the same patients in the course of myocardial infarction may be warranted.

In our research a dual radionuclide study was undertaken for comparison with injection of these tracers on the same day. There was no significant cross talk for the imaging from each other, since antimyosin was administered immediately after pyrophosphate scan and antimyosin images were obtained 48 hr later. Therefore, diagnostic value for detecting infarcted myocardium by two imaging methods in the course of myocardial infraction can be compared without any bias. The physiological uptake in the liver and bone may sometimes cause difficulty in interpretation of antimyosin and pyrophosphate scans, respectively. In this respect, single-photon emission tomography may be useful for the separation of myocardial uptake from the physiological uptake (*25,26*) and for infarct sizing (*8,26–30*).

In conclusion, while both ^{111}In-antimyosin and ^{99m}Tc-pyrophosphate accumulate in the acute stage of myocardial infarction, persistent uptake of antimyosin has also been observed in subacute infarcts beyond the first two weeks when pyrophosphate no longer accumulates. Thus antimyosin scans appear to be valuable for the early identification of acute myocardial infarction, and for the retrospective evaluation of infarction in the course of myocardial infarction.

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