CLINICAL EVALUATION OF SERIAL THALLIUM-201 MYOCARDIAL SCINTIGRAPHY IN SPECIAL REFERENCE TO LEFT VENTRICULAR FUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

YOSHIHARU NAKASHIMA, KAZUMI MAEDA, YASUMASA TOKI,
HIROYUKI KUROGANE, KATSUYA KOBAYASHI, HISASHI FUKUZAKI

In 20 patients with acute myocardial infarction (AMI), serial Tl-201 myocardial scintigraphy was performed and scintigraphic infarct size (SIS) was determined 1 (or less), 2 and 8 weeks after AMI. Hemodynamic indices were determined with a Swan-Ganz catheter at a mean of 27.7 hours after AMI. Left ventricular ejection fraction (EF) was determined with Tc-99m HSA cardiac blood pool scintigram at 8 weeks after AMI. Mapping ECG was also observed serially.

Of 20 patients, SIS decreased in 12, was unchanged in 2 and increased in 5. No image defect was observed on the initial scintigram in one case. When the patients were classified into two groups, i.e. those with initial SIS more than 25% and those with initial SIS less than 25%, hemodynamic indices were demonstrated to be more markedly impaired in the former than in the latter. However, when all patients were pooled, correlations between the initial SIS and hemodynamic indices (CI, SI, SWI, PADP) were poor. There was a good correlation between SIS and EF both determined 8 weeks after AMI.

When the patients were classified into the improved and unimproved groups according to the per cent change of SIS (CIS), both hemodynamic indices on admission and EF at 8 weeks were better in the improved than in the unimproved group, and especially significant correlation was found between CIS and each of SI and PADP.

There was a good correlation between maximum Σ ST in mapping ECG and SIS at 1 week after AMI.

From these results, it is suggested that a decrease in SIS in serial Tl-201 myocardial scintigrams represents the presence of transiently ischemic area and that serial Tl-201 myocardial scintigraphy performed from the initial stage after AMI may provide a useful information for evaluating the effect of therapeutic intervention and predicting the prognosis.

Key Words:

Serial Thallium-201 myocardial scintigraphy
Gated cardiac blood pool scintigraphy
Scintigraphic infarct size
Precordial mapping ECG

DETERMINATION of infarct size and its change with time is expected to provide very useful information for assessing the effect of therapeutic intervention and predicting the prognosis in acute myocardial infarction (AMI). In clinical practice, mapping electrocardiography and analysis of serum creatine phosphokinase

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The First Department of Internal Medicine, School of Medicine, Kobe University

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(CRK) level have been used for estimating infarct size. However, neither of these methods is completely satisfactory because they provide only indirect estimation of infarct size.

Recently myocardial scintigraphy has been introduced as a means of determining infarct size. For this purpose, thallium-201 (Tl-201) and technetium-99m pyrophosphate (Tc-99m PYP) are widely used. With Tl-201, the infarcted area is depicted as an image defect, whereas with Tc-99mPYP, the necrotic area is visualized as a positive image. Wackers, et al.¹ reported good agreement between Tl-201 myocardial scintigram and autopsy finding in terms of location and extent of myocardial infarction. Since the radioactivity of Tl-201 in the myocardium is dependent on the blood flow in the myocardium, a transiently ischemic area as well as a necrotic area is visualized as an image defect. Wacker, et al.² and Smitherman, et al.³ recently reported that, in serial Tl-201 scintigrams, changes in image defect were observed. Smitherman, et al. suggested that such changes might be useful in delineating ischemic from infarcted myocardium in the postinfarction period.

In the present study, the authors attempted to determine the infarct size by serial Tl-201 myocardial scintigraphy in patients with acute myocardial infarction and examined its correlation with (1) hemodynamic changes determined by cardiac catheterization, (2) left ventricular ejection fraction determined by Tc-99mHSA cardiac blood pool scintigraphy, (3) infarct size estimated by mapping electrocardiography.

MATERIAL AND METHODS

The subjects were 20 patients (15 men and 5 women) admitted to the coronary care unit at our university hospital with a clinical diagnosis of acute myocardial infarction (AMI) during the period between December, 1976 and July, 1978. The age range was 45 to 77 years (mean 58.4). All patients had a typical attack of anterior chest pain and showed significant elevation of serum enzyme activities. On ECG, 19 cases showed ST-elevation and/or abnormal Q wave. The remaining one case (Case 14) showed only ST-segment depression in all 12 leads. In one case (Case 13) reinfarction was suspected from ECG. (The patient had previous anterior infarction in 1971). Based on history, ECG and serum enzymes, 19 cases were given the final diagnosis of transmural myocardial infarction and the remaining one case

was diagnosed as subendocardial myocardial infarction. The patients were admitted 1 to 4 days (mean 27.7 hours) after the onset of acute myocardial infarction. Upon admission, a balloon-tipped Swan-Ganz catheter was inserted through the antecubital vein and various hemodynamic parameters were monitored. Cardiac output was determined with dyedilution method. Hemodynamic indices were calculated as follows: Left ventricular stroke work index ($\text{g}\cdot\text{m}/\text{m}^2$) = [Mean systolic pressure (mmHg) - Pulmonary arterial diastolic pressure (mmHg)] $\times$ 0.0136 $\times$ Stroke index. Mean systolic pressure (mmHg) = $0.8 \times$ [Systolic pressure (mmHg) - Diastolic pressure (mmHg)] + Diastolic pressure (mmHg).

Tl-201 myocardial scintigraphy was performed serially, i.e., one week or less (mean 5.6 days), 2 and 8 weeks after the onset of AMI. In two cases (Cases 7 and 12), the initial scintigram (1 and 2 weeks after AMI, respectively) did not show any image defect. Therefore, no further scintigraphy was performed in these two cases. In four cases (Cases 13, 15, 18 and 19), scintigraphy 2 weeks after AMI was not performed. A gamma camera (Toshiba GCA 101) with a pin-hole collimator was used. Energy was selected at 80 KeV photons with a 25 per cent window. Ten minutes after intravenous injection of 2mCi of thallium chloride, myocardial scintigram was taken in anterior, 45° left anterior oblique and left lateral position to obtain images of 200,000 counts. The data were stored in and processed by a computer (TOSBAC 40, DAP 5000 system). On the images thus obtained, the ratio of the area of the image defect to the whole area of the left ventricular myocardium was determined with planimetry. The ratio (%) named "Scintigraphic Infarct Size (SIS)" was expressed as a mean of the anterior, 45° left anterior oblique and left lateral views. Depending on the SIS, the patients were classified into the large (SIS $\geq$ 25%) and small (SIS < 25%) infarct groups. In 19 patients except one (Case 12) who had no image defect, change in infarct size was discussed. Changes in infarct size (CIS) calculated as (SIS % at 8 week - SIS % at 1 week / SIS % at 1 week) $\times$ 100 (%) were judged as decreased, unchanged and increased, and accordingly the patients were classified into improved (CIS < 0) and unimproved (CIS $\geq$ 0) group. In two cases (Case 10, 14) who died between 2 and 3 weeks after AMI and one case (Case 7) who had no image defect at 2 weeks and favorable clinical course in chronic stage, change in infarct size was calculated by SIS at 1 and 2

TABLE I SUMMARY OF SCINTIGRAPHIC, HEMODYNAMIC AND CLINICAL DATA

Patient	Age & Sex	Diagnosis	Days of 1st Scintigraphy after Attack	SIS (%)			Change of SIS (%)	Gated Cardiac Blood Pool Scintigraphy EF (%)	CI (L/min/m ²)	SI (ml/beat/m ²)	SWI (g.m/m ²)	PADP (mmHg)	ΣST (mm)
				1w	2w	8w							
1. K.S.	51 M	I	7	18	11	3	-83	56	3.7	44	50	20	-
2. K.T.	66 M	A	10	22	3	2	-91	-	4.6	48	60	13	71
3. H.T.	77 M	A	8	33	25	24	-27	46	-	-	-	-	-
4. T.I.	56 M	A	8	14	16	5	-64	68	7.1	103	210	12	70
5. I.T.	60 M	A	8	26	30	22	-15	39	2.1	22	30	17	168
6. Y.F.	68 F	A	6	22	10	13	-41	49	4.7	72	111	11	-
7. O.A.	50 M	A	3	8	0	-	-100	55	2.4	46	96	10	44
8. I.K.	53 M	A	14	-	18	24	+33	32	3.3	33	58	23	-
9. K.K.	55 M	A	7	21	21	15	-29	39	3.5	53	59	11	115
10. I.H.t	76 F	A	3	31	33	-	+ 6	29	-	-	-	40	101
11. N.K.	66 M	A	8	10	2	0	-100	69	3.5	46	62	14	66
12. O.S.	65 M	A	6	0	-	-	-	-	3.6	41	59	11	38
13. I.Y.	60 F	I+A*	9	20	-	20	0	36	2.0	21	26	19	-
14. S.T.t	62 F	S	3	3	12	-	+300	37	1.0	17	23	6	-
15. H.H.t	49 M	I+A*	1	28	-	30	+ 7	35	1.6	14	13	30	-
16. G.Y.	45 M	I	5	30	21	17	-43	47	2.6	28	51	15	-
17. N.K.	45 M	A	2	25	20	25	0	36	2.3	28	35	11	244
18. Y.T.	71 F	A	1	36	-	33	- 8	42	2.3	26	44	17	-
19. K.Y.	52 M	I	6	19	-	9	-53	57	3.1	36	59	9	-
20. F.S.	41 M	A	13	-	33	37	+12	41	2.6	26	30	24	195

Abbreviations: † = patients who died; * = re-infarction; A = anterior; I = inferior; S = subendocardial MI; SIS = scintigraphic infarct size; + = increase; - = decrease

Fig. 1. Serial changes in scintigraphic infarct size on serial Tl-201 myocardial scintigrams in patients with AMI.

weeks in these cases. Cardiac output could not be calculated in case 10 using dyedilution method, because the case had left to right shunt caused by the rupture of interventricular septum.

Eight weeks after acute myocardial infarction, 10 mCi of technetium-99m-labeled human serum albumin was administered intravenously and gated cardiac blood pool scintigrams were obtained in the 30° right anterior oblique view using the gamma camera with a parallel 4000-

hole collimator. In each cardiac cycle, exposure was made for 100 msec starting at the peak of R or T waves of ECG and end-systolic images and end-diastolic images of 400,000 counts were obtained. From these images, left ventricular ejection fraction (EF) was determined using the method described by Green, et al.⁴ The same procedure was done at 2 weeks in two cases (Case 10, 14) who died between 2 and 3 weeks after AMI and the result in Case 10 was used for statistical analysis. Mapping ECG was performed in 10 patients with anterior wall infarction using the method described by Maroko, et al.⁵ ECG was recorded from 48 points on the anterior chest wall. Shift of the ST segment was measured at the point 0.06 sec after the J-point. Elevation of 0.2 mV or more was judged as positive for ST-segment elevation and Σ ST was calculated. Initial mapping ECG was performed immediately after admission (mean 37.7 hours after the onset of AMI) and the same procedure was repeated every 24 hours for 7 days. From the data thus obtained, the maximum Σ ST was determined.

The patient (Case 14) with subendocardial myocardial infarction was excluded for statistical analysis because of the difficulty delineating the subendocardial infarct using Tl-201 myocardial scintigraphy. Statistical analysis of the data was

Fig. 2. Tl-201 myocardial scintigrams of a case with acute anterior wall myocardial infarction who showed a reduction of scintigraphic infarct size (left side; original images, right side; processed images).

Fig.3. Serial changes in scintigraphic infarct size in improved and unimproved groups (mean $\pm$ SEM).

performed using unpaired t test to assess differences between groups, and the significance of correlations between parameters was determined by using linear regression analysis.

RESULTS

1. Serial myocardial scintigram

Table I and Figure 1 show the scintigraphic infarct size (SIS) obtained serially at each period of one week or less, 2 weeks and 8 weeks after the onset of AMI. Out of 20 patients, SIS decreased in 12, did not change in 2, and increased in 5. In one case (Case 12), no image defect was observed on the scintigram within one week after AMI. In this case, myocardial infarction of a small size was suspected based on serum enzyme values and ECG changes. Out of 12

patients who showed a decrease in SIS, myocardial scintigram 8 weeks after AMI was almost normal in 4 cases (Cases 1, 2, 4 and 11) and no perfusion defect on scintigram was observed at 2 weeks after AMI in one case (Case 7). In these 5 cases, the mean SIS one week after AMI was 14%, indicating that the infarct was of a small size. The clinical course of these 5 cases were favorable.

Figure 2 shows the scintigrams of one of the cases who showed improvement. Of 20 patients, 7 (Case 8, 10, 13, 14, 15, 17 and 20) showed an increase or no change in SIS. Many of these 7 patients developed serious complications and three died. Case 14 was suspected of myocardial infarction which had developed from unstable angina. Although the SIS was small (3% at 1 week and 12% at 2 weeks), the patient died of left cardiac failure. At autopsy, an extensive subendocardial infarction involving the whole circumference of the left ventricle was found.

Figure 3 shows the serial changes in SIS in patients who showed a decrease in SIS (the improved group) and those who showed an increase or no change in SIS (the unimproved group). In the improved group, the mean SIS, was $21.6 \pm 8.66\%$ one week after the onset of AMI and it was decreased significantly to $13 \pm 10.4\%$ 8 weeks after the onset of AMI ($p < 0.05$). In the unimproved group, the mean SIS was $26 \pm 4.69\%$ at 1 week and $27.2 \pm 6.53\%$ at 8 weeks after the onset of AMI. There was no obvious difference in SIS between the improved and

Fig.4. Relationship between hemodynamic parameters in patients with large infarct and small infarct determined by scintigraphic infarct size.

TABLE II CORRELATION BETWEEN HEMODYNAMIC PARAMETERS AND SCINTIGRAPHIC INFARCT SIZE, AND ITS PERCENT CHANGES

	CI	SI	SWI	PADP	EF
SIS	-0.35	-0.40	-0.37	0.49	-0.82***
Percent Change of SIS	0.45	0.51*	0.44	-0.56**	0.88***

All figures represent correlation coefficients (*r* values)

* $P < 0.05$, ** $P < 0.02$, *** $P < 0.001$

Abbreviations: SIS = scintigraphic infarct size; CI = cardiac index; SI = stroke index; SWI = stroke work index; PADP = pulmonary artery diastolic pressure; EF = ejection fraction at 8 weeks after AMI

unimproved groups at 1 week. Difference was noted between the two groups at 2 weeks and there was a significant difference at 8 weeks ($p < 0.02$).

2. Relationship between scintigraphic infarct size and left ventricular function

According to SIS at one week after AMI, the patients were divided into the large (mean SIS $30.3 \pm 3.77\%$) and small (mean SIS $17.2 \pm 4.94\%$) infarct groups. As shown in figure 4, various hemodynamic indices on admission were compared between two groups. In the large infarct group, cardiac index (CI), stroke index (SI) and stroke work index (SWI) were 2.3 ± 0.4 L/min/m², 24 ± 5.4 ml/beat/m² and 34 ± 13 g.m/m², respectively. In the small infarct group, CI, SI and SWI were 3.8 ± 1.4 L/min/m², 49 ± 22 ml/beat/m², and 77 ± 49 g.m/m², respectively. CI and SI were significantly lower in the large infarct group than in the small infarct group ($p <$

0.02). In contrast, pulmonary arterial diastolic pressure (PADP) was 22 ± 10.1 mmHg in large infarct group and 13.9 ± 4.6 mmHg in small infarct group, respectively and the former was significantly higher than the latter ($p < 0.05$).

Left ventricular ejection fraction determined by gated cardiac blood pool scintigrams 8 weeks after AMI was $36.6 \pm 5.22\%$ in the large infarct group and was significantly lower ($p < 0.05$) than in the small infarct group ($49.4 \pm 12\%$).

Figure 5 shows hemodynamic indices determined on admission for the improved and unimproved groups. In the improved group, CI, SI, and SWI were 3.6 ± 1.5 L/min/m², 48 ± 23 ml/beat/m² and 76 ± 50 g.m/m², respectively. In the unimproved group, CI, SI, and SWI were 2.4 ± 0.6 L/min/m², 24 ± 7 ml/beat/m², and 32 ± 17 g.m/m², respectively. SI was significantly higher in the improved group than in the unimproved group. Although statistically not significant, CI and SWI were also higher in the improved group

Fig.5. Relationship between hemodynamic parameters in improved and unimproved groups determined by percent change in scintigraphic infarct size.

Fig. 6. Relationship between scintigraphic infarct size and maximum ΣST_{max} calculated from mapping ECG in patients with AMI.

than in the unimproved group. PADP in the improved group was 14 ± 3.4 mmHg and was significantly lower than that in the unimproved group which was 25 ± 5.9 mmHg ($p < 0.001$).

Left ventricular ejection fraction as determined by gated cardiac blood pool scintigrams 8 weeks after AMI was $52 \pm 11\%$ in the improved group and $35 \pm 4.1\%$ in the unimproved group and the former was significantly higher than the latter ($p < 0.005$).

Table II shows the correlation between SIS or its per cent change and hemodynamic indices (CI, SI, SWI, PADP, EF). Hemodynamic indices determined in the early stage after the onset of AMI showed better correlation with per cent decrease of SIS than with SIS at one week after AMI. Particularly there was a significant correlation between per cent decrease of SIS and SI, PADP. Ejection fraction determined 8 weeks after AMI showed a significant correlation both with SIS and its per cent decrease.

3. Scintigraphic infarct size and precordial mapping ECG

Figure 6 shows correlation between the maximum ΣST and SIS at one week after the onset of attack in patients with anterior wall myocardial infarction. There was a good correlation between these two parameters ($r = 0.73$; $p < 0.02$).

DISCUSSION

Wacker, et al² performed repeated myocardial scintigraphy within 24 hours after acute myocardial infarction and found a decrease in infarct size in many cases. They demonstrated that the defect size tended to decrease owing to regression of ischemia.

In the present study, serial Tl-201 myocardial scintigraphy was performed at each period of 1 week or less, 2 and 8 weeks after myocardial infarction. Of 20 patients observed, scintigraphic infarct size decreased in 12, was unchanged in 2 and increased in 5. Of the 12 cases who showed a decrease in scintigraphic infarct size, 5 showed almost normal myocardial scintigrams 2 and 8 weeks after infarction. The decrease in scintigraphic infarct size seems to suggest the recovery of injured myocardium including the transiently ischemic area or shrinkage of the infarct area due to fibrotic changes of area necrotic myocardium.⁶

Although there is no definite clinical evidence that the observed scintigraphic changes were induced by the transiently ischemic area, the two cases whose myocardial scintigrams showed disappearance of image defect also showed disappearance of abnormal Q waves on ECG. This finding suggests that serial Tl-201 myocardial scintigrams can demonstrate the changes of infarcted area including the transiently ischemic area.

In the present study, the correlation between the initial scintigraphic size or the rate of its subsequent change and left ventricular function was also examined. When patients were divided into the improved group (with a decrease in infarct size) and the unimproved group (with no decrease in infarct size), there was no marked difference in the initial SIS between the two groups (Fig. 3). However, there was an apparent difference in SIS between the two groups at 2 weeks and there was a significant difference at 8 weeks after myocardial infarction. This suggests that, although the initial infarct size was very similar, there was a difference in recovery process between two groups. This result suggests that some factors other than the initial scintigraphic infarct size play a role in the recovery of the infarcted area. Collateral circulation⁷ and therapeutic intervention may be just a few examples of the factors which may affect the recovery of the infarcted area.

It is interesting to note that, although there was no marked difference in initial scintigraphic infarct size, left ventricular function in early stage of the improved group was significantly better than that in the unimproved group. This is assumed to reflect the difference of myocardial function not only in the non-infarcted area, but also the transiently ischemic area.

Scintigraphic infarct size 8 weeks after myocardial infarction in the improved group was

significantly smaller than that in the unimproved group. Left ventricular ejection fraction determined by gated cardiac blood pool scintigrams was also significantly higher in the improved group than in the unimproved group. Thus, in the unimproved group, many cases showed impairment of the left ventricular pump function in the early stage after myocardial infarction and three cases died. In contrast, the improved group showed better left ventricular pump function, less complications and more favorable clinical course. It was indicated that there was a close relationship between scintigraphic infarct size or per cent change of SIS and left ventricular function. These results indicate the potential usefulness of per cent change of SIS in predicting the prognosis of patients with acute myocardial infarction.

Infarct size has correlated with various degrees of impairment of ventricular function^{8,9}. Norman Sharpe, et al¹⁰ reported good correlation between infarct size determined by Tc-99mPYP and left ventricular function. In the present study, the large infarct group showed more marked impairment of the left ventricular pump function at one week after myocardial infarction than the small infarct group. The same tendency was observed also in terms of left ventricular ejection fraction determined by gated cardiac blood pool scintigrams 8 weeks after myocardial infarction.

However, one week after AMI, there was not necessarily a close relationship between scintigraphic infarct size and various parameters of left ventricular function. In contrast, there was a close correlation between SIS and left ventricular ejection fraction both determined 8 weeks after AMI. This is assumed to be due to inclusion of the transiently ischemic area in the Tl-201 image defect in the acute stage of myocardial infarction. The scintigraphic infarct size 8 weeks after AMI seemed to represent the true infarct area and, therefore, showed a close correlation with left ventricular ejection fraction.

There was a good correlation between the per cent change of SIS and left ventricular function. This seems due to the fact that per cent change of SIS reflected recovery of the transiently ischemic area.

In the present study, a relationship between the scintigraphic infarct size and the infarct size determined by other methods was also examined. Determination of the extent of myocardial injury by means of mapping ECG proposed by Maroko⁵

and Muller et al¹¹ is widely used in actual clinical setting. In this method, the extent of myocardial injury is expressed with Σ ST. As is shown in Figure 6, there was good correlation between SIS one week after AMI and the maximum Σ ST ($r = 0.73$). Similar good correlation was reported by other authors^{12,13} between the infarct size determined by Tc-99mPYP scintigram and precordial mapping ECG.

Selwyn, et al¹⁴ and Willerson, et al¹³ reported that the maximum ST elevation occurred within 24 hours after AMI. Since in the present study, initial mapping ECG was conducted a mean of 37.7 hours after AMI, there is a possibility that in some cases the maximum Σ ST was underestimated. Nevertheless the correlation between the scintigraphic infarct size and the maximum Σ ST in the present study ($r = 0.73$) was better than that reported by Willerson, et al¹³ using Tc-99mPYP ($r = 0.39$). These result may suggest that the infarct size estimated by mapping ECG is better correlated with that estimated by Tl-201 myocardial scintigraphy than that estimated by Tc-99mPYP.

From these facts, it may be suggested that positive image in Tc-99mPYP scintigram demonstrates only the necrotic myocardium whereas both image defect in Tl-201 myocardial scintigram and ST-segment elevation in mapping ECG represent the transiently ischemic area as well as the necrotic myocardium.

There were two cases in which myocardial infarct was not detected properly by myocardial scintigraphy. Case 14 showed a small image defect of 3–12% at the apex but died of marked left ventricular pump failure. At autopsy, infarct at the apex and extensive subendocardial infarct surrounding the whole circumference of the left ventricle were confirmed. Case 12 did not show obvious image defect on myocardial scintigram but was diagnosed as small myocardial infarction based on mapping ECG and serum enzymes. The time which elapsed between the onset of myocardial infarction and myocardial scintigraphy may have been the reason for the false negative myocardial scintigram.

Wacker, et al² reported that the rate of successful diagnosis was higher when myocardial scintigraphy was performed within 24 hours after myocardial infarction than when it was performed later. In the present study, initial myocardial scintigraphy was performed a mean of 6.5 days after myocardial infarction, and 19 cases out of 20 showed image defect. This might ascribe to

the fact that most of the cases in the present study had larger infarct size than in Wacker's. If myocardial scintigraphy was conducted at an earlier period after the onset of myocardial infarction, the rate of successful diagnosis would have more increased. This may be particularly true in cases with small infarct.

There are certain limitations to myocardial scintigraphy. In low output state, myocardial scintigraphic image is often not clear enough and is difficult to read. Determination of infarct size is often difficult because the image is too small and boundary of the infarct area is not well defined. In the present study, a pinhole collimator was used to magnify the images and the images were processed with a computer in order to determine the boundary. A pinhole collimator increases sensitivity but causes distortion of the image. From the results so far obtained in our phantom study, the pinhole collimator was placed at a distance of 1.5 cm from the anterior chest wall in order to make the distortion of the image minimum.

The problem of quantification of the two-dimensional imaging remains unsolved. Computer processing methods in the future seem to be developing and will no doubt play a part in the quantification of infarct size with Tl-201 image.

The minimum size of infarct that can be diagnosed by Tl-201 myocardial scintigraphy was reported to be 4.9 grams in weight¹⁸ and 3.5 cm in diameter.¹⁹

Recently positron emission transaxial tomography²⁰ has been proposed as a method to supplement the limitations inherent to the two dimensional imaging.

However, when these limitations are well understood and taken into account, Tl-201 myocardial scintigraphy is considered to be a useful non-invasive method for diagnosis and predicting the prognosis of ischemic heart disease.

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