Myocardial Perfusion at Fatal Infarction: Location and Size of Scintigraphic Defects

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In a consecutive study of myocardial scintigraphy in acute ischemic syndrome, four patients had ^{99m}Tc-hexamibi injected intravenously before they developed fatal cardiogenic shock. Planar scintigraphy was performed after death. Slices of the hearts after autopsy were analyzed for scintigraphic and pathoanatomic abnormalities. Location of perfusion defects in planar views of the heart was in good agreement with the scintigraphied, sliced sections. The extent of infarction judged from inspection and formasan staining was much smaller (7%–40% and 6%–43% of the total slice area) than found at scintigraphy, where 83%–92% of the myocardium showed ischemia as defined by a ^{99m}Tc-hexamibi uptake below an arbitrary limit on half maximum uptake. Myocardial hypoperfusion might thus aggravate the functional impairment at myocardial infarction and lead to cardiogenic shock.

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Myocardial scintigraphy after intravenous injection of ^{201}Tl or of ^{99m}Tc-hexamibi has been proposed to delineate myocardial areas at risk following coronary arterial occlusion (*1–4*). Binding to myocardium after the initial uptake of ^{99m}Tc-hexamibi (*5*) furthermore gives the possibility of obtaining a "frozen" image of myocardial perfusion distribution several hours after the acute intravenous injection (*4*). This technique was used on a consecutive series of patients admitted to our county hospital for suspicion of acute ischemic syndrome of the heart. As part of the clinical study, we intended to compare clinical, scintigraphic and pathoanatomical signs of acute myocardial infarction in patients who died of cardiogenic shock within 12 hr after radioactive labeling of the myocardium. The aim was to validate the acute scintigraphic picture with ^{99m}Tc-hexamibi of myocardial ischemia as performed earlier by Wackers et al. (*3*) using ^{201}Tl in fatal cases.

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METHODS

Over a 4-mo period (February to June 1990), all patients admitted to the coronary care unit with suspicion of an acute ischemic syndrome (myocardial infarction or unstable angina) were immediately injected with ^{99m}Tc-hexamibi (duPont, Germany). The radioactive dose was adjusted to result in a dose of 300 MBq at the time of scintigraphy, which was performed up to 18 hr after injection.

Six patients died 2–13 hr after injection of ^{99m}Tc-hexamibi. Legislative autopsy was allowed for only four patients, which was performed within 18 hr postmortem. The clinical course for the four male patients, aged 66–87 yr, is shown in Table 1. Three had known prior myocardial infarction and three received streptokinase immediately after radioactive labeling of the myocardium. None of the patients were in clinical shock when ^{99m}Tc-hexamibi was injected.

Scintigraphy was performed on the ward by a Siemens mobile camera using a parallel-hole collimator. The data were stored in a dedicated computer. Three planar views were taken (anterior, 45 and 70-degree left anterior oblique) with the decreased patient in supine position (Fig. 1). After autopsy, 1-cm thick slices from apex to base of the heart were placed on a Siemens (LFOV) gamma camera for imaging. These slices were evaluated visually for myocardial infarction before and after staining with formasan.

The planar views of the heart in vivo are not directly comparable with the sliced sections so two nuclear medicine specialists were asked to place the perfusion reduction in the anteroseptal, lateral, or posteroinferior wall from the planar views of the left ventricle as proposed by Wackers (*3*). Scintigraphic defects in planar views or slices of the heart were defined as areas with less than 70% or 50% of maximum counts on the digitalized images (Fig. 2). The extent of perfusion defects was determined as the percentage pixel area below those thresholds.

The pathoanatomical abnormalities were evaluated by two pathology specialists. The scintigraphic slices drawings of the infarcted area before and after formasan staining were combined and the infarcted area was determined by a stereological method. Light and electron microscopy were not performed because formasan staining is the most sensitive method to delineate myocardial infarction.

RESULTS

Localization of perfusion defects from planar scintigraphy in three views of the heart in situ showed excellent agreement with scintiphotos of the slices placed on the

TABLE 1
Clinical Course in Four Patients Dying of Cardiogenic Shock from Myocardial Infarction

Patient	Sex	Age	Blood pressure at ^{99m}Tc injection	2-hr intervals 0....10....20....30
1	M	69	160/100	S——H*————————D————————P—A
2	M	80	200/110	S————————H————————————D————————P—A
3	M	66	170/110	S————————————H*————————————D————————P—A
4	M	87	90/	S————————H*—D—P————————————————————A

S = symptom start, H = admittance, H* = streptase given at admittance, D = death, P = planar scintigraphy, and A = autopsy with heart slices.

gamma camera (Table 2). An example of the slice scintigrams is given in Figure 1 (Patient 2 has an infero-lateral infarction and a prior inferior infarct).

The areas with infarct at autopsy were less extensive on visual inspection (8%–41%) and after formosan staining (6%–44%) than the areas with perfusion defects found on scintigraphic slices (83%–92%) (Table 3).

DISCUSSION

In the course of a prospective study of myocardial scintigraphy in acute ischemic syndromes, we encountered four patients who died and were autopsied within 31 hr. The size of the ischemic areas, determined by postmortem acute myocardial scintigraphy were compared to the pathological anatomy. From our experience, myocardial scintigraphy with ^{99m}Tc-hexamibi seems to be a reliable tool to localize myocardial ischemia even in the worst cases: relatively low count rates and excellent tissue attenuation at infarction of the posterior wall or of the subendocardial muscle. The technique can be used both acutely, but also

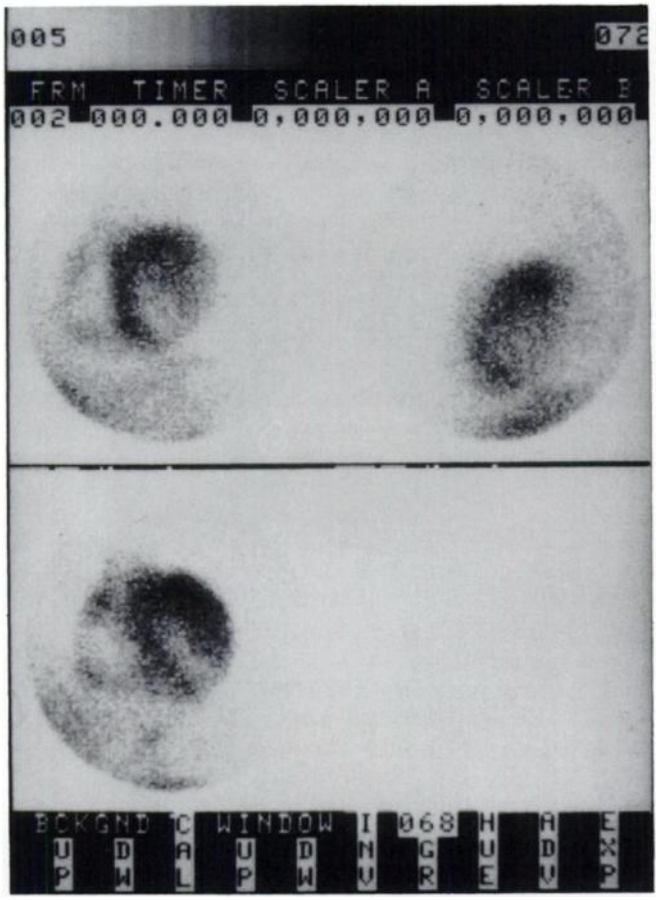

FIGURE 1. Planar scintigraphy in three projections of the heart (Patient 1).

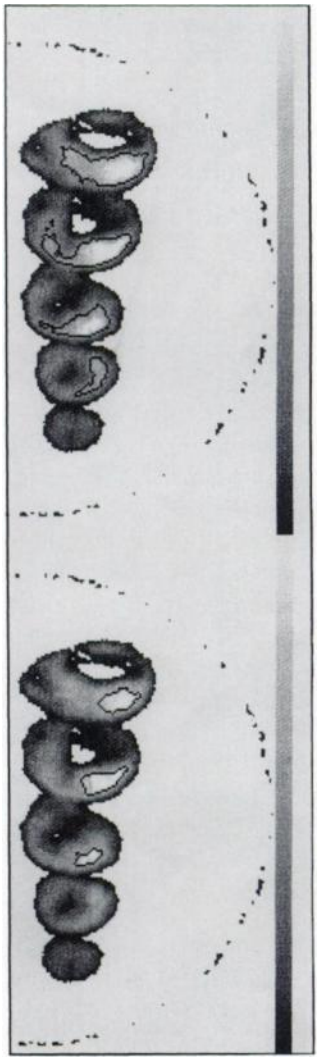

FIGURE 2. Scintigraphy of slices of the heart after autopsy (Patient 1). The encircled area represents more than 50% or 70% of maximum counts in upper and lower panel.

TABLE 2
Location of Perfusion Defects at Scintigraphy

	Planar scintigraphy			Scintigraphy of slices		
Patient	Anterior	Lateral	Inferior	Anterior	Lateral	Inferior
1	1	0	2	1	0	2
2	1	0	2	1	0	2
3	0	1	2	0	1	1
4	1	1	2	2	0	2

No defect = 0 and defect or large defect = 1 or 2.

TABLE 3
Size of Ischemia (<50% Uptake) and Infarction in Percent of Total Area from Scintigraphy of Sliced Sections of the Heart and Pathoanatomical Findings Before and After Formasan Staining

Patient no.	Ischemic area	Necrotic area	Formosan-stained area
1	84	37	9
2	92	8	6
3	83	22	12
4	86	41	44

in blinded studies to make a "frozen" picture of perfusion distribution before intervention.

In acute animal experiments good correlation between "areas at risk" and the distribution area of ligated vessels has been found (*6,7*), although infarct size might be reduced with time by release of the occlusion, which might be equivalent to the clinical situation after trombolysis (*7*). The same good agreement was found by Wackers et al. (*3*) using thallium scintigraphy on 23 patients with fatal transmural infarction. In these patients, a surprisingly high number died of ventricular rupture, but five patients were identified who died within 12 hr of cardiogenic shock. In four of these patients, there was disagreement between scintigraphy and the extent of myocardial infarction.

This study supports the findings in our small group of four patients where the pathoanatomical changes were more limited than ischemia predicted from scintigraphy of slices of the heart. Additional factors may be responsible for this disagreement:

1. It takes some time for pathoanatomical changes to develop, however, formasan staining identifies areas of infarction.
2. In the clinical course of irreversible cardiogenic shock, a gradual worsening of myocardial perfusion at low blood pressure might lead to more extended ischemia, but our patients were not in shock at the time of injection.
3. Post-stenotic ischemia in parts other than the infarcted area might arise from increasing demands on residual myocardial tissue.
4. Myocardial hypoperfusion might lead to a metabolic shift from fatty acid to glucose metabolism in larger areas of the myocardium, which might be associated with a change in radiotracer uptake.

Our findings create serious doubt as to the possibility of delineating myocardial infarction by scintigraphy in fatal cases of cardiogenic shock as proposed by Wackers et al. (*3*), since the zone of necrosis was smaller than the area of hypoperfusion.

Improvements in the scintigraphic technique to delineate perfusion defects could be obtained by higher contrast of gated planar or tomographic investigations of the heart (*2,8*). Such investigations are difficult in severely ill patients and impossible to carry out in most cardiac wards. Further study is necessary to determine whether acute myocardial scintigraphy can be of value in the handling of patients admitted on clinical suspicion of ischemia.

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