Editorial Comment

Technetium-99m Pyrophosphate And Indium-111 Antimyosin Antibody Scintigraphy Appear to Be Comparable Methods for Infarct Detection*

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The present study. In this issue of the Journal, Takeda and his colleagues (1) describe their studies comparing the accuracy and sensitivity in myocardial infarct detection of technetium (Tc)-99m pyrophosphate and indium-111 antimyosin imaging in dogs with temporary and permanent coronary artery occlusions. The myocardial tissue localization of Tc-99m pyrophosphate and antimyosin antibody was compared in 11 dogs 24 to 68 h after left anterior descending coronary artery occlusion. In four dogs the coronary occlusion was permanent, and in seven it was temporary and followed by reperfusion. Tc-99m pyrophosphate and antimyosin antibody concentrations were determined in serial 2 to 3 mm wide endocardial and epicardial samples and multiple short-axis left ventricular slices within the infarct zones. The number of samples with increased antimyosin antibody was not significantly different from that with increased Tc-99m pyrophosphate uptake both in dogs with permanent and in dogs with temporary coronary artery occlusion. The intensity of uptake of Tc-99m pyrophosphate exceeded that of the antimyosin antibody, especially in the border zones of reperfused infarcts. Moreover, the area of myocardium with moderate to marked increases in tracer uptake (=2 times normal) was larger with Tc-99m pyrophosphate than with antimyosin antibody (p < 0.001). A specific zone of abnormal Tc-99m pyrophosphate, but without increased antimyosin antibody uptake, was not identified in these studies. Histologic evidence of myocardial necrosis was found in virtually every myocardial sample with increased antimyosin antibody, Tc-99m pyrophosphate, or both.

The investigators (1) conclude that Tc-99m pyrophosphate and antimyosin antibody should represent equally good markers for the detection of irreversible myocardial injury with and without myocardial reperfusion after myocardial infarction. They further suggest that the larger scintigraphic estimates of infarct size with Tc-99m pyrophosphate found in some previous studies were most likely due to greater uptake of Tc-99m pyrophosphate in necrotic myocytes, especially those located at the infarct boundaries rather than increased uptake of pyrophosphate in injured viable myocardial tissue.

Previous studies. This study should help to end any continuing controversy concerning whether Tc-99m pyrophosphate or antimyosin antibody is a markedly superior method for detecting or sizing myocardial infarcts when the proper methods and instrumentation are used. Previous studies (2-18) have demonstrated that both imaging techniques have good sensitivity in detecting myocardial necrosis in experimental animal models and in patients. However, because Tc-99m pyrophosphate uptake has been found in clinical settings where myocardial necrosis was not always evident, including in some patients with unstable angina, cardiomyopathy and heart failure and in some patients with persistently abnormal uptake after myocardial infarction (7,8,18,19), it has been suggested that pyrophosphate uptake may occur in reversibly injured myocardial tissue. Detailed research studies conducted by several groups (13,15-17) have demonstrated that antimyosin antibody uptake occurs in irreversibly injured myocardial tissue and that extensive disruption of membrane integrity is required for it to concentrate in increased amounts. A few comparative studies (14) in which the extent of Tc-99m pyrophosphate and antimyosin antibody uptake were measured in animal models and patients with infarction have suggested that the area of pyrophosphate uptake is greater than for the antimyosin antibody, thus also suggesting that pyrophosphate uptake may occur in reversibly injured cells in some circumstances. On the other hand, extensive animal and clinical studies (3-12) have shown that Tc-99m pyrophosphate uptake occurs predominantly in irreversibly injured tissue. These previous studies have included detailed morphologic comparisons at light and electron microscopic levels (5-8), comparisons of Tc-99m pyrophosphate uptake with abnormalities in myocardial mitochondrial function after experimental coronary artery occlusion (10), comparisons of pyrophosphate uptake with triphenyltetrazolium chloride staining of experimental infarcts (20) and detailed morphologic measurements of infarct size versus the extent of pyrophosphate uptake in experimental animal models with myocardial infarction (11,12).

Estimation of infarct size after reperfusion. Theoretically, experimental or clinical circumstances where transient but marked increases in cytosolic calcium concentration occur in reversibly injured myocytes might lead to increased pyrophosphate uptake (5,6). This may occur if Tc-99m pyrophosphate myocardial imaging is performed immediately after reperfusion. Indeed, differences in infarct size

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with pyrophosphate imaging and single photon emission computed tomography (SPECT) have been shown (12) in canine models with temporary coronary artery occlusion followed by reperfusion depending on whether the pyrophosphate is injected 10 to 15 or 90 min after reperfusion. If the pyrophosphate is injected immediately with reperfusion, there is an overestimation of infarct size in experimental animal studies. However, if the injection is made after 90 min of reperfusion, there is an accurate estimate of infarct size by pyrophosphate and SPECT imaging (12). These studies have led to the recommendation that Tc-99m pyrophosphate be injected no sooner than 90 min after reperfusion. The overestimation of infarct size associated with immediate injection of pyrophosphate after reperfusion is most likely caused by transient uptake of pyrophosphate in selected cells populations with cytosolic calcium overload and reversible myocardial injury (12). In the study of Takeda et al. (1), the injection of Tc-99m pyrophosphate was accomplished 2 h after the start of reperfusion (1); thus, one might anticipate similar findings to those reported in the earlier studies mentioned previously (12).

Indium-111 antimyosin antibody versus Tc-99m pyrophosphate imaging. As more experience has been gained with indium-111 antimyosin antibody imaging, it has become clear that there are several similarities with Tc-99m pyrophosphate imaging (21). First, a certain percentage of patients with dilated cardiomyopathy have abnormal indium-111 antimyosin antibody images in the absence of clinically evident myocardial infarction (21). Second, persistent abnormalities in indium-111 antimyosin antibody scintigrams occur in some patients after myocardial infarction (Haber E and Berger H, personal communication, 1990), just as had been described previously for Tc-99m pyrophosphate (7). The persistently abnormal Tc-99m pyrophosphate scintigrams in patients after infarction have often served as markers of a complicated future course. Deterioration in ventricular function occurs in many of these patients, suggesting that persistently abnormal Tc-99m pyrophosphate scintigrams may be the result of ongoing chronic cellular injury and necrosis (7,22,23).

From the data available, we agree with Takeda et al. (1) that both Tc-99m pyrophosphate and In-111 antimyosin antibody imaging should provide equally acceptable, but alternative, means to detect irreversible myocardial injury and myocardial infarction when the issue is in doubt clinically. Similarly, both techniques should provide a good means to estimate the extent of myocardial infarction when proper imaging instrumentation allowing three-dimensional reconstruction of the infarct is used, attention to the ideal imaging characteristics for both tracers is observed and the images are interpreted by observers experienced in the use of the respective tracers.

For those who have worked to develop these imaging methods and have been frustrated previously by conflicting results and controversy, it is rewarding to have further confirmation that both of these imaging techniques are valuable in the noninvasive detection of acute myocardial necrosis.

References
