CASE REPORT

Demonstration of a Right Ventricular Infarction with Tomographic Thallium Myocardial Imaging

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We present a tomographic Tl-201 myocardial study from a patient who had a right-ventricular infarction. A right-ventricular defect was noted at the apex as well as an inferior left-ventricular defect. Since right-ventricular infarction commonly occurs in association with an inferior left-ventricular infarction, we believe that this new application of computerized gamma-camera technology will lead to increased appreciation of this association.

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Tomographic thallium myocardial imaging reconstructed from multiple separate projections over a wide angle is a new diagnostic modality. Application of this technique demonstrated a right-ventricular (RV) infarction in a 60-year-old white male who presented on Oct/18/81 with prolonged chest pain. He subsequently evolved an inferior myocardial infarction, attested by new 40-msec EKG Q waves in leads II, III, and aVF, associated with a CPK elevation from a baseline of 54 to 1650 and an initial CPK-MB fraction of 16.4%. His early hospital course was complicated by ventricular arrhythmias, ultimately controlled with procainamide, and a RV infarction syndrome requiring the use of Swan Ganz catheterization for adequate management. Later his course was complicated by Dressler's syndrome.

Further evaluation was prompted in Nov/81 by recurrent anginal symptoms and a positive exercise treadmill stress test. Catheterization performed preoperatively revealed a dominant right coronary artery with a proximal 100% occlusion, which visualized by retrograde filling, a left circumflex with a 60–70% proximal stenosis, and left anterior descending coronary with a 70–80% stenosis beyond the takeoff of the diagonal branch. The left main coronary artery was normal. Wall-motion analysis revealed an akinetic posteroinferior wall with a left-ventricular (LV) ejection fraction of 55%. The patient underwent a four-vessel bypass on Nov/25/81.

The patient's subsequent course was complicated by intractable arrhythmias, including nonsustained ventricular tachycardia. Before initiating treatment with amiodarone, a radionuclide ventriculogram was performed (Fig. 1). It revealed an enlarged, diffusely hypokinetic RV with an akinetic apical segment (another manifestation of the RV infarct) and a normal-sized LV chamber with a markedly hypokinetic inferior wall. The LV ejection fraction was 69%. An exercise thallium treadmill stress test was performed; it revealed a focal defect involving the posteroinferior surface on the LV as well as a defect in the apical-inferior wall of the RV. Rest images performed four days later were unchanged from the exercise study.

The myocardial images were acquired by a tomographic gamma camera equipped with a general-purpose parallel-hole collimator and interfaced to a computer system. Sixty-four images were acquired over 180° in a 128 × 128 matrix. Following tomographic reconstruction, the myocardial images were reoriented along the major cardiac axes (Fig. 2) and displayed in a 64 × 64 format (*4*). Figure 3 (left) demonstrates an inferior LV defect and a RV defect in the transaxial short-axis view. The corresponding (normal) anatomic location is presented in Fig. 3 (right). Figure 4 left shows

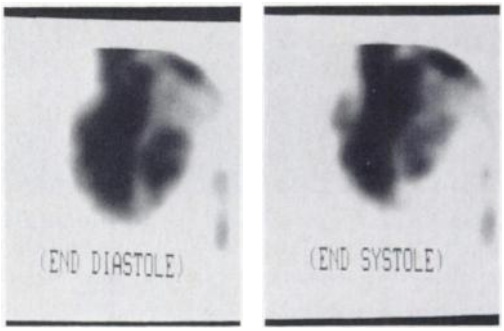

FIG. 1. Radionuclide ventriculogram (35° LAO) using Tc-99m-labeled RBCs: Regional wall-motion analysis revealed a hypokinetic left-ventricular inferior wall and an akinetic right-ventricular apex.

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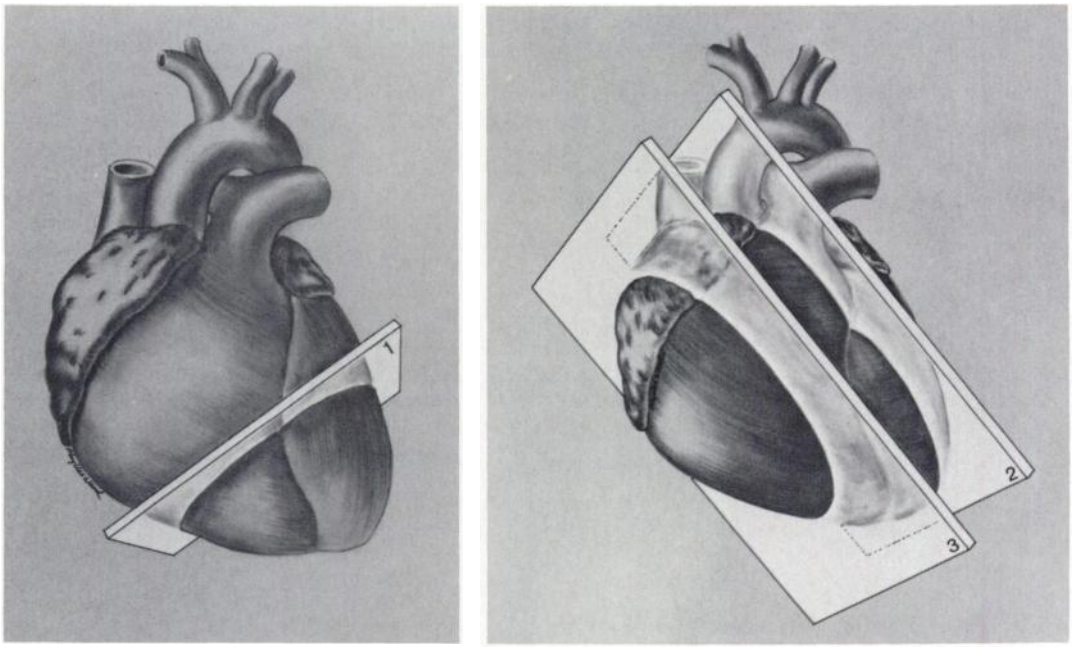

FIG. 2. (left) Normal cardiac anatomy, indicating the orientation of three tomographic sections: transaxial short-axis (slice #1), (right) long-axis through the left ventricle (slice #2) and long-axis through the right ventricle (slice #3).

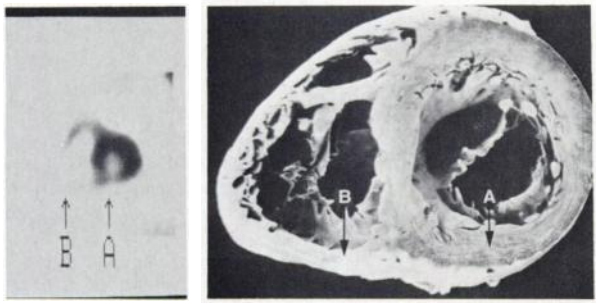

FIG. 3. (left) Transaxial short-axis Tl-201 tomographic section which shows focal defects in left-ventricular inferior wall (A) and in the right-ventricular inferior wall (B). (right) Normal myocardium viewed in cross section (short axis). Arrows (A and B) identify areas corresponding to image defects.

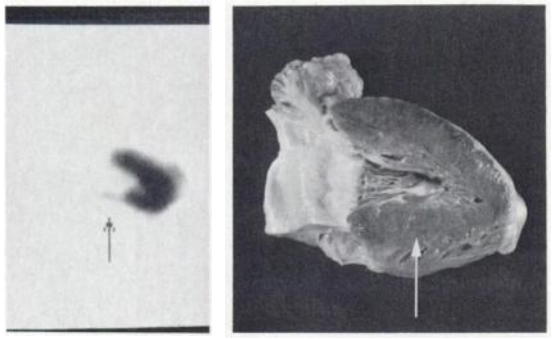

FIG. 4. (left) Long-axis tomographic section through left ventricle (plane #1, Fig. 2), showing focal defect in inferior wall (arrow). (right) Normal myocardium viewed in a longitudinal section. Arrow corresponds to location of image defect observed in Fig. 4, left.

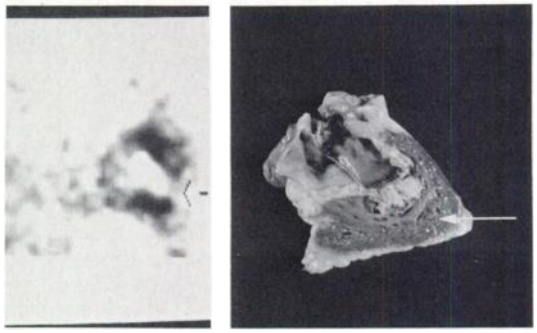

FIG. 5. (left) Long-axis tomographic section through right-ventricle (slice #3, Fig. 2), showing perfusion defect involving infero-apical wall (arrow). (right) Normal right-ventricular myocardium viewed from same perspective as in Fig. 5, left. Arrow indicates location of perfusion defect.

the inferior LV defect in the long-axis view, and Fig. 5 (left) shows the apical RV defect in the long-axis view. Examples of the corresponding normal anatomy are presented respectively in Figs. 4 (right) and 5 (right). We believe that orientation of the images along the major cardiac axes facilitates appreciation of the image defects.

DISCUSSION

It is generally recognized that RV infarction occurs primarily in association with LV inferior-wall infarction. This observation is supported by the knowledge that both the inferior LV wall and the RV are commonly supplied by the right coronary artery. Isner and Roberts (*1*) report in their necropsy series that RV infarction occurred in 24% of 139 patients with an inferior (posterior) infarction of the LV, but did not occur in any of the 97 patients with an anterior-wall infarction. Right-ventricular infarction occurred most commonly when a posterior infarction and a transmural septal infarction occurred together (33 of 65, 50%). Wackers et al. (*2*) imaged a series of 78 patients with acute inferior transmural LV infarctions (new ECG Q waves) using both Tl-201 and Tc-99m

pyrophosphate modalities. Pyrophosphate imaging identified RV infarction in only 31% of their study group.

In the Wackers study, none of the patients imaged developed clinical evidence for RV infarction. Lorell et al. (*3*) applied very stringent clinical criteria for the diagnosis of RV infarction and found evidence for it in 12 of 306 patients (4%) who presented with electrocardiographic evidence for acute inferior or inferoposterior infarction of the left ventricle. Lorell further reported that the clinical presentation of RV infarction is frequently confused with the diagnosis of pericardial effusion or constrictive pericarditis. They point out that the incidence of RV infarction may actually be higher than reported because it is frequently not suspected.

There are currently several reports documenting the increased sensitivity of tomography for the detection of remote LV infarction (*5,6,8*). In our study, not only is the inferior LV defect clearly portrayed, but the associated RV infarct is also identified.

ACKNOWLEDGMENT

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