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Sustained Right Ventricular Dyskinesis Complicated by Right Ventricular Infarction

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We encountered a 66-yr-old man with acute left inferior and right ventricular infarction. Tomographic radionuclide ventriculography and Fourier analysis clearly demonstrated reduced wall motion in the inferior walls of both ventricles and markedly delayed phase angles in the inferior right ventricular segment, indicating dyskinesis, which was confirmed by two-dimensional echocardiography and contrast right ventriculography. Four years later, right ventricular dyskinesis was still present and corresponded to a right ventricular perfusion defect on ^{99m}Tc-labeled tetrofosmin tomogram. Right ventricular imaging with tomographic radionuclide ventriculography with Fourier analysis and ^{99m}Tc-labeled myocardial tomography demonstrates that, even after improved global function and hemodynamics, right ventricular dyskinesis related to right ventricular perfusion defect can be sustained for several years. Thus, these imaging techniques may contribute to diagnosing right ventricular infarction and investigating the pathophysiology.

Key Words: right ventricular infarction; radionuclide ventriculography; tetrofosmin scintigraphy; dyskinesis

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Right ventricular (RV) infarction is an important complication of acute left ventricular inferior infarction, sometimes leading to hemodynamic deterioration and poor patient prognosis (*1,2*). Impairment of RV performance and hemodynamics due to RV infarction can improve spontaneously over time, typically within several days to a few weeks; sustained RV failure or wall motion abnormality is quite rare later (*3*). Poor clinical outcomes in RV infarct patients are due to generally hemodynamic deterioration, RV failure and arrhythmias at an acute phase, probably related to RV infarct size. Therefore, it is very important clinically to evaluate the presence and extent of RV infarction. However, unless hemodynamic or electrocardiographic alterations are manifested, RV infarction is often not diagnosed, probably because of difficulties in identifying regionally impaired RV perfusion and wall motion, which can be prolonged even after the recovery of global RV function (*4,5*). Technetium-99m-pyrophosphate scintigraphy is useful for delineating infarcted myocardium per se, but the availability is limited to several days following infarction. Two-dimensional echocardiography, which has proved to be of value for bedside monitoring of regional wall motion and predicting an increased RV pressure due to pump failure has technical limitations in some cases, and other conventional imaging modalities seem less useful. Recent advances in scintigraphic tomography may help to detect RV infarction-related dysfunction and perfusion abnormalities more precisely (*6–8*); that is, improvement of spatial and temporal resolutions for cardiac imaging can be achieved by ^{99m}Tc-labeled perfusion agents with an ideal

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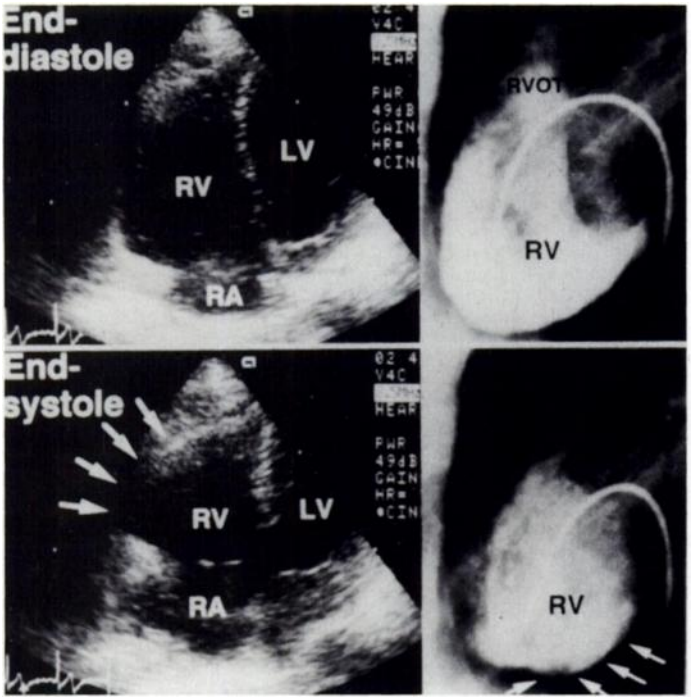

FIGURE 1. Two-dimensional echocardiograms from the apical four-chamber view (left panels) and contrast right ventriculograms from the left lateral view (right panels) at end-diastole and end-systole demonstrate right ventricular dyskinesis (arrows). LV = left ventricle; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract.

dosimetry, computer-assisted analysis of cardiac performance and rapid data processing, and a two- or three-head gamma camera. We observed a patient with sustained RV dyskinesis and a perfusion defect complicated by acute inferior infarction for 4 yr using tomographic radionuclide ventriculography (*6*) and myocardial perfusion tomography with a ^{99m}Tc tracer (*7*).

CASE REPORT

A 66-yr-old man was admitted with acute inferior infarction and complicated RV infarction. Coronary angiography revealed a complete occlusion of the right coronary artery at the origin with relatively rich collaterals. At an acute stage, he was stable with a heart rate of 79/min and blood pressure 112/74 mmHg, had no symptoms or signs suggestive of right or left heart failure, hypotension, or cardiogenic shock and any heart block was not detected. There were no significant hemodynamic abnormalities despite an increased pulmonary capillary wedge pressure of 19 mmHg; cardiac output 4.5 l/min, cardiac index 2.96 l/min/mm2, right atrial pressure 5/1 mmHg and right ventricular pressure 22/3 mmHg. Planar and tomographic ^{99m}Tc-pyrophosphate scintigraphies performed 4 days after the onset clearly demonstrated intense accumulation at the inferior regions of both ventricles. Radionuclide ventriculography from the 45° left anterior oblique view using 740 MBq of ^{99m}Tc-labeled human serum albumin revealed RV asynergy, and RV ejection fraction was 33%. Subsequently, gated blood-pool tomography was performed using a large-field-of-view rotating gamma camera with a high-resolution, parallel-hole collimator and a dedicated minicomputer system to produce short-axis tomograms (*6*). Briefly, gated tomographic data were obtained at 10° increments for 60 sec per increment during a 180° rotation from the 45° left anterior oblique to the 45° right anterior oblique view using a multiple-gated mode with a framing rate of 10 frames per cardiac cycle and stored in a 64 × 64 word matrix nuclear medicine computer system. After transaxial reconstruction using a filtered backprojection algorithm, short-axis tomograms were created. The functional short-axis tomograms of amplitude and phase angle derived from Fourier analysis with first-order harmonics (*6,8,9*) showed regional abnormalities in both ventricles, that is, definitely reduce amplitude (asynergy) in the inferior walls of both ventricles and markedly delayed phase angles (dyskinesis) in the RV inferior and posterolateral walls. Two-dimensional echocardiography and contrast right ventriculography

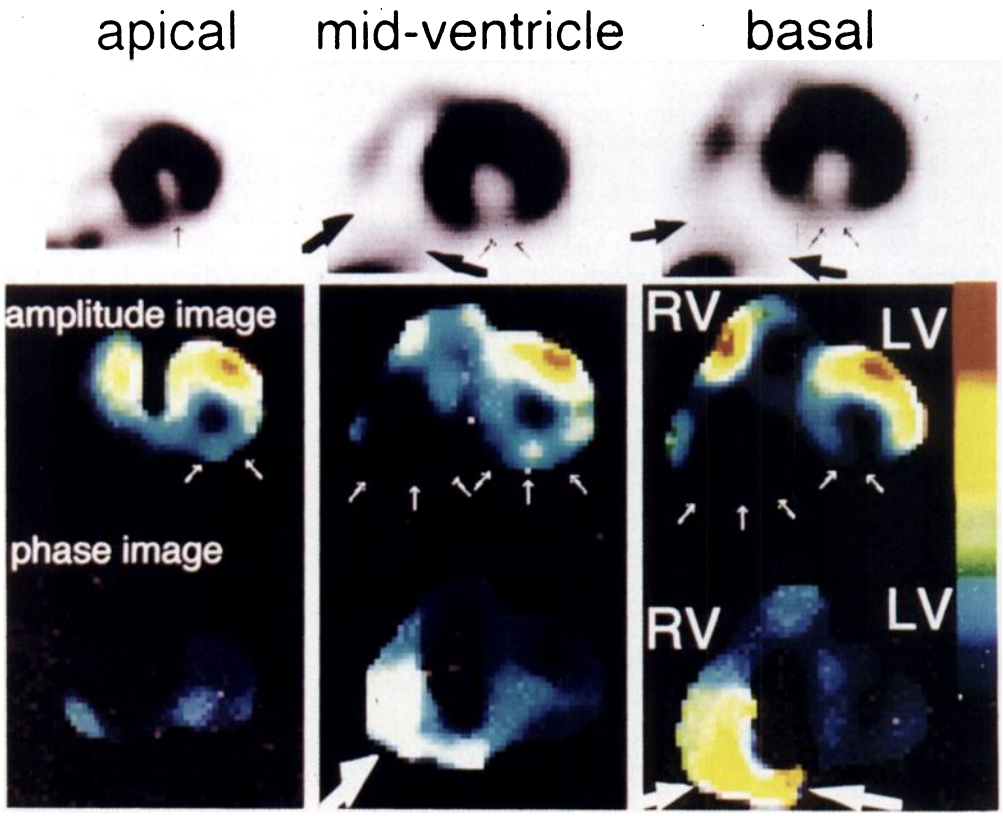

FIGURE 2. Apical, mid-ventricular and basal short-axis tomograms of ^{99m}Tc-tetrofosmin scintigraphy (upper panels) and radionuclide ventriculography (middle and lower panels) 4 yr after myocardial infarction. Amplitude images (middle panels) clearly demonstrate reduced wall motion in the inferior segments of both ventricles (small white arrows), which well correspond to perfusion defects of left (small black arrows), and right ventricles (large black arrows). Note that markedly delayed phase angles are observed in the RV inferior segments (white large arrows in lower panels) showing perfusion defects. Abbreviations are the same as in Figure 1.

confirmed these observations retrospectively (Fig. 1). Four years later, RV dyskinesis detected by a markedly delayed phase angle on the functional short-axis tomograms was still present. Furthermore, myocardial SPECT with 740 MBq ^{99m}Tc-labeled tetrofosmin was performed at rest. Data were obtained at 5° increments for 30 sec per increment during a 180° rotation using the before mentioned rotating gamma camera and collimator and short-axis tomograms were reconstructed by a filtered backprojection algorithm. For delineating RV myocardial perfusion, 50% of the maximal activities was cut off. The location of RV dyskinetic wall motion corresponded to an RV perfusion defect on the tetrofosmin short-axis tomograms (Fig. 2). The patient had initially suffered from non-sustained ventricular tachycardia, but there was no evidence of heart failure or pulmonary or systemic embolization during follow-up.

DISCUSSION

Even after improved global RV function and hemodynamics, RV dyskinesis related to the regional RV perfusion defect was sustained for 4 yr. Similar experimental observations (*4*) have been reported, but, in these cases, regional RV dyskinesis disappeared over a period of several weeks, probably due to coronary reperfusion and small RV infarct size, in contrast to the present case. In a majority of cases, the RV is resistant to ischemia and infarction because it requires less oxygen, and its collateral circulation is more extensive (*4,5*). In the present patient, collaterals were unlikely to limit infarct-size or to improve the RV wall motion abnormality (*4*) because, as a result of delayed admission, coronary reperfusion was not achieved, and the right coronary artery was chronically occluded during the 4-yr follow-up. The present findings in RV infarction suggest that regionally impaired RV contractile function may recover slowly and, in some cases, may be sustained. RV infarction is routinely recognized by hemodynamic alterations, electrocardiography, echocardiography and pyrophosphate scintigraphy; however, the diagnosis is made very infrequently when RV failure or low cardiac output is not clinically manifest. Despite the ability for assessing regional wall motion abnormality of both ventricles and for precisely measuring a ventricular volume, gated blood-pool tomography is not routinely utilized probably because of the time-consuming characteristics and economical problems. However, a three-head gamma camera and more powerful computer system currently available might overcome these limitations. Technetium-99m-labeled perfusion tracers, such as sestamibi and tetrofosmin both of which are used for a routine clinical practice, are more useful for delineating RV myocardial perfusion compared to thallium because the shorter half-life allows us to use a higher dose of the tracer, and the greater photopeak is more suitable for a conventional gamma camera. Although RV perfusion imaging using ^{99m}Tc-labeled sestamibi has been demonstrated (*7,10*), there is no available literature focused on the detection of RV infarction by tetrofosmin scintigraphy. It seems unlikely that there is any clinical difference in an image quality or clinical utility between the two perfusion tracers because of their similar dosimetry and physical characteristics.

CONCLUSION

Recent advances in SPECT, a powerful computer system and ^{99m}Tc-labeled tracers might contribute to regional assessment of RV performance and perfusion (*6,7*) and to making these tomographic techniques more widely available clinical tools. Further investigation is, however, necessary to establish the diagnostic values of tomographic gated blood-pool and tetrofosmin scintigraphies for detecting regional abnormalities of RV function and perfusion. Although prolonged RV dyskinesis complicated by left inferior and RV infarction appears to be quite rare, regional RV dysfunction may be detected more frequently by using these techniques. Despite largely reversible global RV dysfunction, RV involvement has been related to increased morbidity in the acute and chronic stages (*2*), and the precise identification of an RV perfusion abnormality and asynergic wall motion could affect the long-term therapeutic strategy in RV infarct patients. The natural history of sustained RV dyskinesis or wall motion abnormality, however, remains to be established, and the clinical techniques presented here may contribute to the evaluation of regional RV performance and prognosis.

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