Diagnosis and prognosis of right ventricular infarction

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SUMMARY The values of several non-invasive methods for the diagnosis of right ventricular necrosis in inferior myocardial infarction were compared in 51 consecutive patients who underwent serial radionuclide ventriculography, pyrophosphate scintigraphy, and cross sectional echocardiography. In addition a unipolar electrocardiographic lead V4R was recorded on admission, daily, and during episodes of further pain. Profound right ventricular dysfunction was evident in 50% of patients studied by radionuclide methods after inferior myocardial infarction but recognition on clinical groups alone was poor. Functionally important right ventricular infarction was best detected and followed serially by radionuclide ventriculography. Echocardiographic methods for evaluating right ventricular ejection fraction correlated poorly with radionuclide methods. Increased uptake of radioactivity by the right ventricle on pyrophosphate scintigraphy usually indicated poor right ventricular function, but a scan that was negative in the right ventricular territory did not exclude dysfunction. ST segment elevation in V4R was not specific for right ventricular infarction and its routine use may lead to overdiagnosis of this condition. Serial measurements suggest that profound right ventricular dysfunction persists after acute inferior infarction and is associated with considerable morbidity and mortality. Of 25 patients with severe right ventricular dysfunction, six died in the late hospital period. In the remaining 19 patients mean right ventricular ejection fraction over a two month period did not improve; six patients had persistent right ventricular dyskinesia and features of chronic right ventricular failure developed in three survivors.

Right ventricular necrosis has been recognised at necropsy for many years,^{1,2} but it was not until 1974 that Cohn *et al* first described its potentially serious haemodynamic consequences.³ Since then awareness of right ventricular infarction has increased and the reported incidence in patients with acute inferior myocardial infarction is 25-53%.⁴⁻⁶ Early recognition of right ventricular dysfunction is important because the time of onset of its often profound haemodynamic sequelae is unpredictable and these may be prevented by the administration of an appropriate intravenous volume load. If right ventricular

infarction is neglected or treated as left heart failure with diuretics cardiogenic shock may supervene.

Clinical recognition of right ventricular infarction now rests largely on a typical, though not uniformly present, clinical picture backed by evidence of right ventricular dilatation and dysfunction. Because routine right heart catheterisation in every patient presenting with acute inferior infarction is impracticable, non-invasive diagnosis by electrocardiographic or radioisotopic methods to detect or to raise the suspicion of important right ventricular necrosis at an early stage would be helpful. Such an electrocardiographic method was suggested by Erhardt in 1974 who proposed that right ventricular infarction could be diagnosed by elevation of the ST segment in the right precordial bipolar chest lead CR4R,⁷ and more recently there have been favourable preliminary reports of the use of the more con-

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ventional unipolar chest lead V4R.⁸ In addition cross sectional echocardiography may be used to visualise the right ventricle and assess its function.^{9 10}

In search of a reliable non-invasive diagnostic method we prospectively evaluated radioisotope ventriculography, pyrophosphate scintigraphy, echocardiography, and the electrocardiographic lead V4R to determine the prevalence, clinical implications, and prognosis of right ventricular involvement in acute infarction of the inferior myocardial wall.

Patients and methods

PATIENT POPULATION

We studied 51 consecutive patients (13 women and 38 men, mean age 59 (range 36–73)) admitted to a coronary care unit with acute infarction of the inferior myocardial wall. None had evidence of previous myocardial infarction. In all patients ischaemic chest

pain lasted longer than 30 min and was followed by electrocardiographic abnormality in leads II, III, and aVF with evolutionary ST segment/T wave changes or the appearance of Q waves or both. Myocardial infarction was confirmed in all cases by a significant rise in serum activity of at least two enzymes (creatinine kinase, aspartate aminotransferase, or lactate dehydrogenase). Clinical right ventricular infarction was diagnosed if three or more of the following were present simultaneously within 48 hours of admission—persisting hypotension (systolic blood pressure <100 mm Hg), raised jugular venous pressure, clear lung fields (clinically and radiographically), and oliguria (<300 ml urine per 24 hours or <10 ml urine per hour for two consecutive hours).

ELECTROCARDIOGRAPHY

A unipolar right chest lead V4R was recorded with a conventional 12 lead electrocardiogram on admission, daily for four days, and during any further epi-

Fig. 1 Pyrophosphate scintigrams showing positive and negative pyrophosphate uptake. RV, right ventricle; LV, left ventricle.

Fig. 2 Distribution of right ventricular ejection fraction determined by first pass radionuclide angiography 2-4 days after inferior infarction. RVN, radionuclide ventriculography.

sodes of chest pain. ST segment elevation was regarded as important if it was >0.5 mm above the isoelectric line.

NUCLEAR VENTRICULOGRAPHY

Right ventricular function was initially estimated at day 2-4 from a gated bolus of 800 MBq of technetium-99m pyrophosphate imaged in the 30° right anterior oblique projection over 7-15 cardiac cycles by a Siemens high resolution LEM gamma camera that was appropriately positioned after a transmission scintigram had been obtained from a posteriorly positioned flood source. Right ventricular ejection fraction was derived from the first pass data by the techniques previously described by this laboratory.¹¹ Thereafter right and left ventricular ejection fractions were calculated from a gated blood pool equilibrium study with phase analysis of images generated over 500 cardiac cycles and acquired in the 30° left anterior oblique projection by means of

Fig. 3 Comparison between radioisotopically determined right ventricular ejection fraction and ST elevation in leads V4R and V3R. RVN, radionuclide ventriculography.

Fig. 4 Comparison between radioisotopically determined right ventricular ejection fraction and results of pyrophosphate scintigraphy. RNV, radionuclide ventriculography.

single region of interest methods. Estimation of right and left ventricular ejection fractions were repeated at day seven and at week eight by gated blood pool ventriculography after the injection of 800 MBq of ^{99m}Tc labelled human serum albumin.

PYROPHOSPHATE SCINTIGRAPHY

Myocardial infarct scintigraphy was performed at day 2–4, two hours after the injection of 800 MBq of ^{99m}Tc pyrophosphate. Images were recorded in the anterior and 45° left anterior oblique projections. Scintigrams were graded on a scale from 0 to 4 on the basis of the level of radioactivity in the cardiac region: 0, no activity; 1, slight indefinite activity; 2, definite activity but less intense than bone; 3, activity equal to bone; 4, activity greater than bone. Scintigrams graded ≥ 3 were considered to be abnormal. In addition, the area of increased left ventricular uptake was described as inferior or inferolateral with either anterior or posterior extension. Right ventricular infarction was deemed to be present if, in the

45° left anterior oblique projection, radioactivity was present anterior to the interventricular septum and posterior and directly adjacent to the bony chest wall (Fig. 1).

ECHOCARDIOGRAPHY

Cross sectional echocardiography was attempted in 38 patients during their hospital stay (days 3–5) and repeated eight weeks later using an Irex 3 real time phased array imaging system and a 2.5 MHz transducer. Cross sectional echocardiograms of the right ventricle were obtained in the parasternal long axis view, parasternal short axis views at the level of the aortic and mitral valves, and in the apical four chamber view. Echocardiograms were recorded on videotape for subsequent play back. End systolic and end diastolic dimensions of the right ventricle were measured in each view by means of stop frame analysis, and we used average chamber dimensions to calculate right ventricular ejection fraction by the formula shown⁹: $\text{RVEF} = (\text{Ded}^2 - \text{Des}^2) \div \text{Ded}^2$, where Ded is mean end diastolic dimension and Des is mean end systolic dimension.

Electrocardiography, scintigraphy, blood pool ventriculography, and echocardiography were performed and interpreted by independent observers. Statistical analysis was performed by the paired Student's *t* test and conventional linear regression formulas.

Fig. 5 Comparison between right ventricular ejection fraction (RVEF) determined by radionuclide ventriculography (RNV) and echocardiography (echo).

Fig. 6 Right (RVEF) and left ventricular ejection fraction (LVEF) one week and eight weeks after acute inferior myocardial infarction.

Results

Figure 2 shows the range of right ventricular ejection fractions determined by radionuclide methods in the acute stage. Severe right ventricular dysfunction was noted in 25 patients in whom the mean right ventricular ejection fraction was <0.25 (normal range $0.53-0.71$). The condition was diagnosed clinically in only three of these patients (mean right ventricular ejection fraction 0.16). All three were treated by volume loading, which greatly improved their clinical state. In retrospect eight other patients with mean right ventricular ejection fraction of 0.16 had some of the clinical features of right ventricular dysfunction which were not recognised clinically at presentation, and without exception they had a complicated acute phase with persisting hypotension and oliguria. A further 14 patients with a mean right ventricular ejection fraction of 0.21 had no clinical features of right ventricular dysfunction.

ELECTROCARDIOGRAPHY

Forty eight patients developed new abnormal Q waves in the inferior leads. Acute ST/T changes in the absence of Q waves developed in two patients and one patient had left bundle branch block. Figure 3 shows the results of radioisotopically determined right ventricular ejection fraction and the presence or absence of ST segment changes in the right chest lead. When the ST segment elevation in lead V4R was >0.5 mm it had a sensitivity of 92% for diagnosis of severe right ventricular dysfunction (right

ventricular ejection fraction <0.25) but a specificity of only 40%. Compared with pyrophosphate scintigraphy ST elevation in V4R had a similar sensitivity (88%) but specificity was 42%.

PYROPHOSPHATE SCINTIGRAPHY

Pyrophosphate scintigraphy confirmed inferior infarction in all 51 patients and increased right ventricular uptake was noted in 25. Uptake was increased in the following regions of the left ventricle: inferior (39), inferolateral (5), posterior extension (3), anterior extension (2), diffuse uptake (2). A scan positive in the right ventricular territory usually indicated poor right ventricular function but three patients with right ventricular dysfunction had negative scans (Fig. 4).

ECHOCARDIOGRAPHY

Echocardiography was attempted in 38 patients, and in two patients echocardiograms were of inadequate quality for examination. The right ventricular ejection fraction that was determined echocardiographically correlated poorly with that measured by the radioisotope technique ($r = 0.43$) (Fig. 5).

FOLLOW UP

Serial measurements in this group showed that there was a significant improvement in both right and left ventricular ejection fraction in the two months after infarction that was detected by first pass and equilibrium blood pool ventriculography and by echocardiography (Fig. 6). There was a significant correlation between first pass and equilibrium radionuclide methods for measuring right ventricular ejection fraction ($r = 0.93$). Improvement in right ventricular function, however, was limited to those patients who had moderate to good right

Fig. 7 Right ventricular ejection fraction (RVEF) one week and eight weeks after acute inferior infarction. The response of those with initially poor function is compared with that of those with initially good function.

ventricular ejection fraction soon after infarction (Fig. 7). In those with early severe right ventricular dysfunction no improvement was detected. In this group there were six late hospital deaths, and three of the survivors developed features of predominant chronic right heart failure.

Discussion

Necropsy studies have shown that involvement of the right ventricle in acute inferior myocardial infarction is more common^{2 12 13} than its recognition and treatment in routine clinical practice would suggest. In the clinical setting there is no substitute for right heart catheterisation to identify the deranged haemodynamic function produced by right ventricular infarction and to monitor treatment, but use of this technique in all patients presenting with acute inferior myocardial infarction is impracticable and unnecessary. In addition, without fluoroscopy interpretation of the pressure trace may be misleading³ and ascertaining the precise level of right ventricular end diastolic pressure may be difficult,¹⁴ but this is critical if this variable is to be used as the only haemodynamic index of right ventricular infarction. Moreover, in the series of Candell-Riera *et al* eight of 18 patients with inferior infarction and scintigraphic evidence of right ventricular involvement had no definite haemodynamic evidence of right ventricular dysfunction.¹⁵ Thus it may be that pressure monitoring will detect only gross dysfunction of the right ventricle, whereas the ejection fraction may be abnormal long before there is any increase in pressure. Using haemodynamic criteria alone, Candell-Riera *et al* found that 36% of patients had right ventricular infarction,¹⁵ whereas Erhardt and Isner and Roberts in their necropsy series reported rates of 45% and 50% respectively.^{7 16} Reliance on invasive haemodynamic measurements alone may therefore lead to underdiagnosis.

We found that the right ventricle was affected in 50% of patients with acute inferior wall infarction as detected by radionuclide ventriculography or pyrophosphate scintigraphy and this accords with the studies of Sharpe *et al*, Garty *et al*, and Croft *et al* who have reported frequencies of 40%, 52.7%, and 47.6% respectively.^{5 17 18}

CLINICAL FEATURES

The clinical signs of right ventricular decompensation have already been mentioned. Clinical recognition of right ventricular infarction is poor even when the whole syndrome is present. In our study only 12% of cases of severe right ventricular dysfunction were detected clinically and all of these

patients responded well to an appropriate intravenous volume load. A further eight patients had a complicated acute phase with some of the clinical features of right ventricular involvement; they did not receive volume loading and they were subsequently shown to have poor right ventricular function. In cases in which the clinical index of suspicion is high further evidence from invasive or non-invasive investigation may be necessary to overcome reluctance to administer an appropriate volume load.

ELECTROCARDIOGRAPHY

ST elevation in the right precordial lead V4R has been reported to be a sensitive and specific index of right ventricular infarction.^{7 8} Other workers have found the test to be less specific, however, with 10% of patients with acute anterior infarction showing a similar elevation.¹⁷ Similarly, false positives may occur in patients with pericardial diseases and left bundle branch block. Our study suggests that ST elevation in V4R is a more sensitive indicator of right ventricular dysfunction than radioisotope measurements but that its lack of specificity may lead to overdiagnosis.

PYROPHOSPHATE SCINTIGRAPHY

Various workers have pointed out the special role of pyrophosphate scintigraphy in the diagnosis of right ventricular infarction.^{5 19 20} As an isolated procedure, however, it has limitations for detecting all patients with right ventricular necrosis, mainly because it depends on the delineation of a small amount of radioisotope in the right ventricular free wall that is partly concealed by the bony skeleton. In addition, maximum pyrophosphate accumulation occurs in myocardial regions where blood flow is reduced to 30–40% of normal.²¹ With lower flow rates pyrophosphate uptake falls despite the increasing degree of necrosis. The chance of detecting necrosis is highest between 36–72 hours after the onset of chest pain.²² Any delay in scanning for whatever reason will lead to false negative scans. Furthermore, other conditions (for example unstable angina and defibrillation) have been shown to give false positive pyrophosphate scans. Given these limitations, we have shown that right ventricular positive pyrophosphate scans are usually indicative of poor right ventricular function but a scan with no apparent uptake in the right ventricular territory does not exclude the diagnosis.

ECHOCARDIOGRAPHY

A few studies have assessed the value of echocardiography in the detection of right ventricular infarction.^{10 23 24} The limitations of M mode echo-

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cardiography in evaluating patients with coronary artery disease and segmental wall motion abnormalities are well recognised. Various abnormalities have been demonstrated by cross sectional echocardiography in patients with right ventricular infarction; these include segmental dyskinesia, paradoxical septal motion, dilatation of the right ventricle, tricuspid incompetence, and localised pericardial effusions. Using cross sectional echocardiography, we obtained images of the right ventricle in several views. Patient build, relative inaccessibility of the adult right ventricle, its complex geometry, and difficulty in delineating endocardial surfaces accurately may have accounted for the poor correlation with first pass and equilibrium radionuclide methods that are unaffected by such variables.

RADIONUCLIDE VENTRICULOGRAPHY

Radionuclide ventriculography has been used extensively to study both left and right ventricular function and count based methods have been used to overcome some of the problems of complex geometry of the right ventricle.²⁵ Because of the theoretical difficulties with gated blood pool studies for analysis of right ventricular function, we performed both first pass and gated studies in our patients. We successfully excluded right atrial counts by phase analysis, and this gave a good correlation ($r=0.9$) between the two radionuclide methods for determining right ventricular ejection fraction.

Right ventricular ejection fraction varies widely in normal subjects^{25,26}; in our laboratory the normal range is 0.53–0.71. Garty *et al* regarded a right ventricular ejection fraction of <0.45 as evidence of right ventricular infarction; thus they included patients with very mild right ventricular dysfunction.¹⁷ When they used this criterion they found that low right ventricular ejection fraction has a low specificity for detection of right ventricular infarction. For the purposes of our study, severe right ventricular dysfunction was taken to be a right ventricular ejection fraction of <0.25 , and 50% of our patients fell into this category. Our data show that use of stricter criteria for right ventricular dysfunction enhances the specificity of the test.

On the basis of radioisotopically determined right ventricular function, it was possible to categorise patients into low and high risk groups and thus obtain some indication of prognosis. Previous studies have shown that patients with right ventricular infarction have a higher complication rate and poorer prognosis than those in whom the right ventricle is not affected.^{8,16} Our data suggest that patients with right ventricular infarction fall into two groups: those with slight impairment of func-

tion whose clinical condition improves as the right ventricular ejection fraction increases and those with early severe right ventricular dysfunction who have a higher late hospital mortality (24%) and in whom low right ventricular ejection fraction persists with associated complications after discharge from hospital.

We found radionuclide angiography to be a useful non-invasive investigation for the detection of right ventricular infarction in patients with inferior infarction that was better than electrocardiographic or echocardiographic methods. The technique is useful for indicating which patients may benefit from invasive haemodynamic monitoring and it also indicates short term prognosis after inferior infarction.

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