Diagnosis and prognosis of right ventricular infarction

Sir,

The important paper by Dr Rodrigues and colleagues (1986;56:19-26) showed that in a series of 51 patients with acute infarction of the inferior myocardial wall, right ventricular ejection fraction was depressed in all cases, while pyrophosphate scintigraphy was positive in 25 (≈50%) of the 51 patients. Moreover, severe right ventricular dysfunction was taken to be a right ventricular ejection fraction of <0.25 and 50% of their patients fell into this category. They also argued that this stricter criterion (right ventricular ejection fraction <0.25) enhances the specificity of radionuclide ventriculography in the diagnosis of right ventricular involvement.

We should like to make the following comments:

(a) It is surprising that they found a clear cut depression of right ventricular ejection fraction in all patients; this finding does not accord with the pathological and radionuclide reports and it suggests incorrect selection of the patient population. Did they exclude patients with chronic pulmonary obstructive disease, valvar heart disease, or right bundle branch block?

(b) Rodrigues et al obtained negative pyrophosphate scans in about half of their patients. If this finding is compared with that in (a), it follows that half of their patients showed right ventricular dysfunction in the absence of scintigraphic, and perhaps also clinical and electrocardiographic, findings consistent with right ventricular necrosis.

(c) Using a right ventricular ejection fraction <0.25 as the criterion for right ventricular dysfunction Rodrigues et al reported involvement of the right ventricle in 50% of patients. What then, was the meaning of the lower limit (0.53) of normal range for right ventricular ejection fraction?

(d) Garty et al emphasised that regional wall motion abnormalities are the most constant finding with the greatest sensitivity and specificity in the diagnosis of right ventricular infarction. We and our coworkers demonstrated a statistically significant correlation between depressed right ventricular ejection fraction and the presence of regional wall motion abnormalities of the right ventricle in a series of patients with acute infarction of the inferior myocardial wall. Analysis of right ventricular regional wall motion was not reported by Rodrigues et al.

(e) Rodrigues et al did not report left ventricular ejection fractions in the acute phase of infarction (days 2 to 4) nor give details of treatment.

(f) Could Dr Rodrigues and colleagues explain why mean right ventricular ejection fraction was 0.0-21 in the 14 patients without clinical features of right ventricular dysfunction?

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References


This letter was shown to the authors who reply as follows:

Sir,

We thank Dr Palagi and colleagues for their interest in our paper and for their comments. They pose a number of questions which we will try to answer succinctly.

Patient selection is important; our patients had been admitted to our coronary care unit and over 90% had sustained transmural myocardial infarction. We excluded patients with chronic obstructive pulmonary disease or valvar heart disease. We have previously reported that after anterior

[Note: The rest of the text is not visible in the provided excerpt.]
transmural anterior myocardial infarction left ventricular ejection fraction was below the lower limit of normal in virtually all patients and was <0.25 in 40%. In that study gross right ventricular dysfunction as detected empirically by right ventricular enlargement and abnormal wall movement was seen in 43% of all patients surviving inferior infarction.

Dr Palagi and colleagues may have misunderstood the point regarding pyrophosphate scintigraphy. All patients had a positive uptake in the myocardium, but only in 25 was the uptake detectable in the right ventricle. We discussed the possible reasons for negative pyrophosphate scintigrams in the text.

The aim of our study was to compare the reliability of the various non-invasive diagnostic methods of determining the frequency of right ventricular involvement in acute inferior wall myocardial infarction and, more importantly, to examine the clinical implications and prognosis of right ventricular involvement. By using the arbitrary figure of 0.25 for right ventricular ejection fraction we were able to identify those patients with more severe right ventricular dysfunction whose clinical progress was likely to be complicated both in the short and the long term. Those with less pronounced right ventricular dysfunction (ejection fraction 0.25 to 0.53) showed subsequent improvement in right ventricular function and in the main their progress has been satisfactory. The results would not have been substantially different if the equally arbitrary value of 0.3 for right ventricular ejection fraction had been taken.

Wall motion analysis was performed with phase analysis and, as stated in the summary, persisting right ventricular dyskinesia was evident in evidence patients with poor residual ventricular function, but when a segmental wall motion score was used as an isolated variable it was not of further discriminant value. The complex anatomy of the right ventricle, however, makes the accurate study of abnormal wall motion difficult from any assessment in a single plane.

Our report showed left ventricular ejection frac-

tion at days 2 to 4 which is at a comparable time to our previous study and after the spontaneous acute changes in left ventricular ejection fraction in the first 24 hours have subsided. Treatment was standardised according to our own therapeutic schedule and there were no deviations from this.

Finally, we suggest that right ventricular infarction only becomes clinically apparent when gross right ventricular damage is sustained. Should accurate non-invasive methods of diagnosis be unavailable we believe that the index of clinical suspicion for right ventricular dysfunction should be high in all cases of transmural inferior myocardial infarction. We have reread the paper by Dr Palagi and colleagues and we suggest that our paper supports their final conclusion “that the characteristic feature of inferior MI is a decreased RVEF ...”. Different patient selection is the likeliest reason for the variation in the reported frequency of poor right ventricular function.

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References