RIGHT VENTRICULAR INFARCTION — CLINICAL, HAEMODYNAMIC, ECHOCARDIOGRAPHIC AND THERAPEUTIC CONSIDERATIONS

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SYNOPSIS

Right ventricular infarction complicating infarction of the inferior wall of the left ventricle occurs in a significant number of patients. The syndrome that is produced is characterised by an elevated jugular venous pulse, clear lung fields and in some instances hypotension. Diagnosis can be confirmed either by haemodynamic pressure measurement of the right side of the heart, by technetium pyrophosphate scintigraphic scan and lastly by M mode or 2 dimensional echocardiography. The diagnosis of right ventricular infarction has therapeutic importance because diuretics are contraindicated in this condition and fluid loading instead should be instituted.

A patient with right ventricular infarction is described.

INTRODUCTION

Myocardial infarction has traditionally been regarded as involving predominantly the left ventricle of the heart in the great majority of instances. Hypotension or cardiogenic shock complicates myocardial infarction in about 15% of cases and have until recently been believed to be always due to severe left ventricular dysfunction (1). In recent years, a unique syndrome where right sided heart failure accompanied sometimes by hypotension but without left heart failure occurring as a result of right ventricular infarction is being increasingly recognised. Since no case of right ventricular infarction has been reported from Singapore to date, we would like to describe such a patient with a plea for a wider recognition of this syndrome.

CASE REPORT

A 48-year-old Chinese male was admitted to the Coronary Care Unit of the University Department of Medicine at Singapore General Hospital with an acute transmural inferior myocardial infarct. The patient suffered an anteroseptal myocardial infarct four years ago but had been symptom free since. His risk factors for ischaemic heart disease included hypertension and being a heavy cigarette smoker.

On admission, he was generally well with a blood pressure of 120/80 mm Hg. Clinical examination revealed that the jugular venous pressure was elevated to 10 cm above the sternal angle. The apex beat was not displaced and the heart sounds were dual and regular without any cardiac murmurs. The lungs were clear. The electrocardiogram showed acute transmural inferior infarct with Q waves and T wave inversion in Leads II, III and AVF (Figure 1). The peak serum creatinine kinase was markedly elevated at

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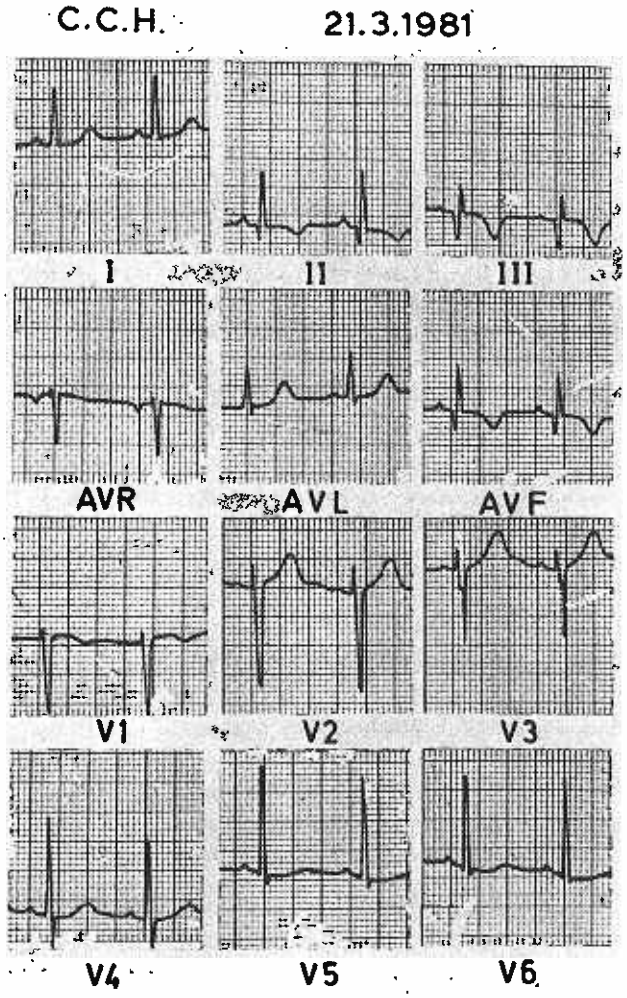

Fig. 1 Twelve lead ECG showing transmural inferior myocardial infarction.

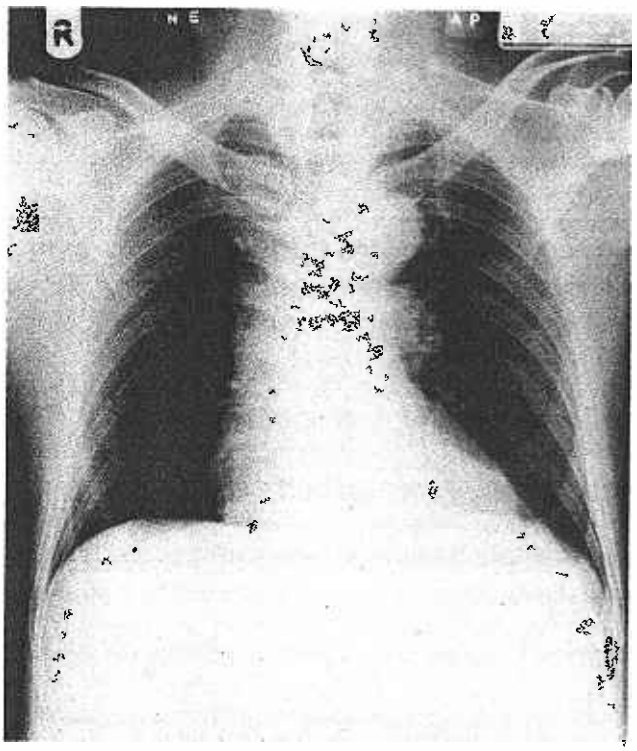

Fig. 2 Chest Xray showing minimal cardiomegaly and clear lung fields.

968 IU/litre. The chest x-ray showed mild cardiomegaly with clear lung fields (Fig 2) and the routine blood biochemistry was normal. In view of the raised jugular venous pressure and clear lung fields, he was diagnosed as having right ventricular infarction in addition to inferior myocardial infarction of the left ventricle. A Swan-Ganz catheter was therefore inserted and the pressures in the right heart was measured. The mean right atrial pressure was elevated to 13 mmHg and a prominent "V" wave and "y" descent were noted. The capillary wedge pressure was 14 mm Hg (Fig 3). The ratio of the mean right atrial pressure to the mean pulmonary capillary wedge pressure was 0.92. M mode and cross sectional echocardiography were then performed. In the M mode study the end diastolic right ventricular dimension was 2.2 mm and that of the left ventricle was 4.8 mm giving an end diastolic right ventricular to end diastolic left ventricular dimension ratio of 52% (Fig 4). The cross sectional echocardiographic study using the subxiphoid approach revealed a dilated right ventricle with akinesia of its free wall. (Fig 5).

The patient made an uneventful recovery from the myocardial infarction and was subsequently discharged from hospital. When reviewed in the outpatient's clinic 6 months later he was found to be well.

DISCUSSION

In the majority of instances myocardial infarction involves predominantly the left ventricle of the heart. In the cases which are complicated by pump failure, haemodynamic monitoring has been shown to be invaluable for optimal management (2, 3). In recent years, this procedure has led to the detection of the syndrome of predominant right ventricular failure resulting from right ventricular infarction occuring in some patients with transmural inferior myocardial infarction of the left ventricle (4). Clinically, these patients have signs of right ventricular failure with little or no evidence of left ventricular failure in marked contrast to the usual clinical situation where right ventricle failure is secondary to left ventricular failure. In these patients, the right atrial pressure is elevated with either normal or slightly elevated pulmonary capillary wedge pressure (4). A mean right atrial pressure to a mean pulmonary capillary wedge pressure ratio of 0.65 or more as was seen in our patient has been used as a diagnostic criteria for right ventricular infarction (5). In patients with extensive right ventricular infarction, the right atrial and right ventricular pressure wave forms may be identical suggesting absence of any effective right ventricular contraction (6). This haemodynamic profile resembles that of constrictive pericarditis. However, in the presence of hypovolemia the haemodynamic features of right ventricular infarction may be masked (5, 6).

Right ventricular infarction can also be diagnosed employing non invasive investigative techniques such as scintigraphy and echocardiography. Sharpe and co-workers have found that technetium pyrophosphate scintigraphy and gated radioangiography with 99m technetium albumin can both detect the occurrence of right ventricular infarction accurately (5). Using M mode echocardiography, these same

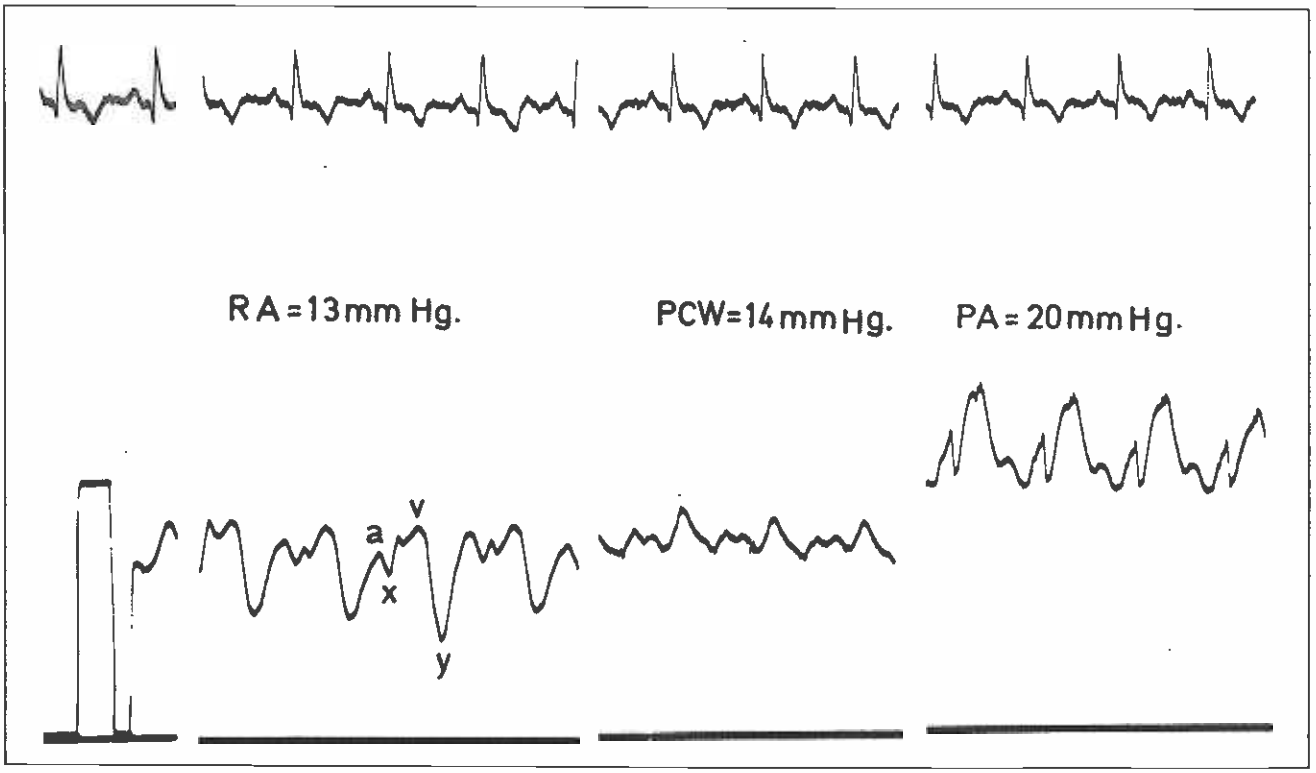

Fig. 3 Pressure tracings of right atrium, pulmonary artery and pulmonary capillary wedge. RA = right atrium PCW = pulmonary capillary wedge PA = pulmonary artery.

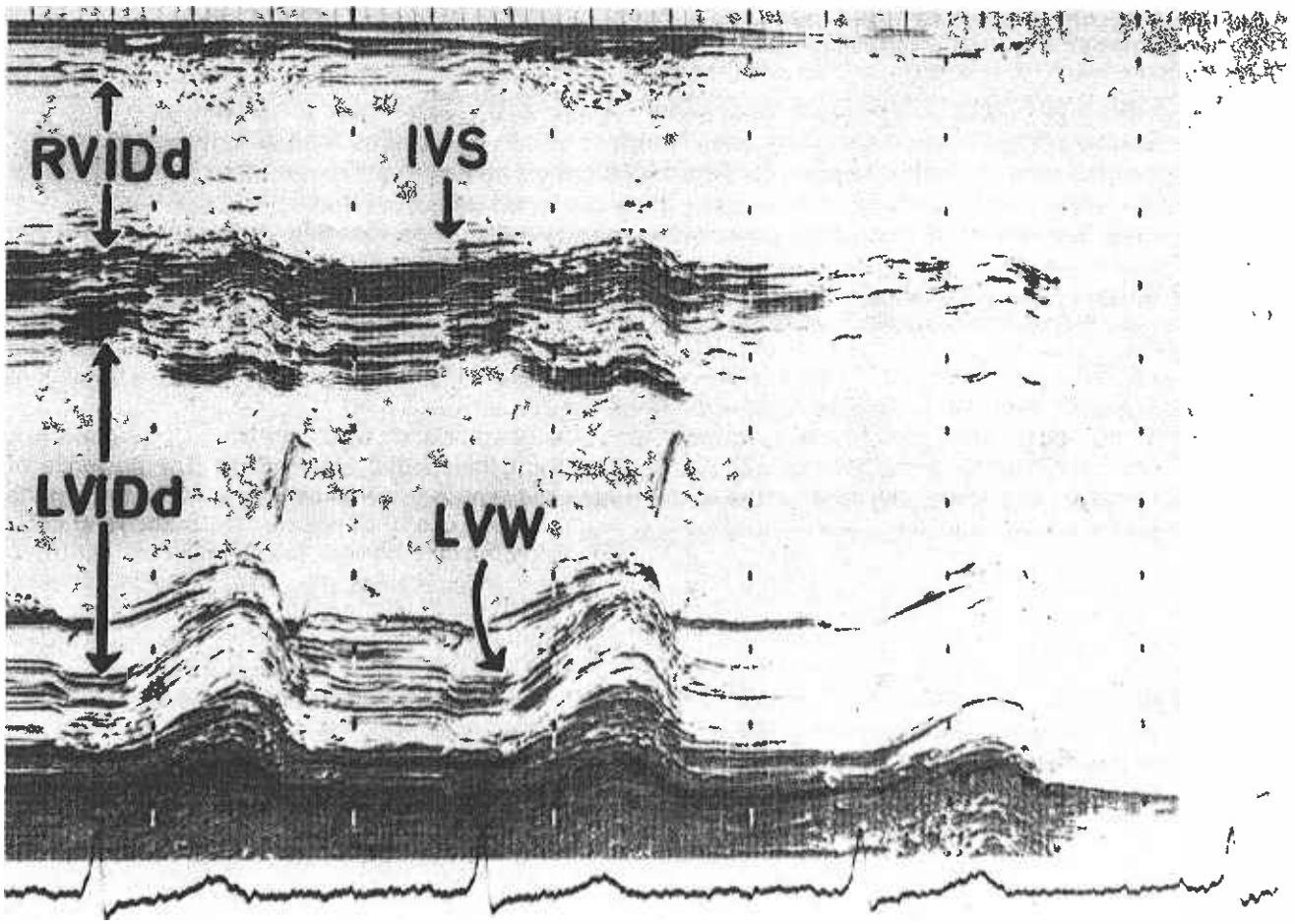

Fig. 4 M mode echocardiogram showing right and left ventricular dimensions. LVIDd = left ventricular internal dimension in end diastole. RVIDd = right ventricular internal dimension in end diastole. IVS = interventricular septum. LVW = posterior wall of left ventricle.

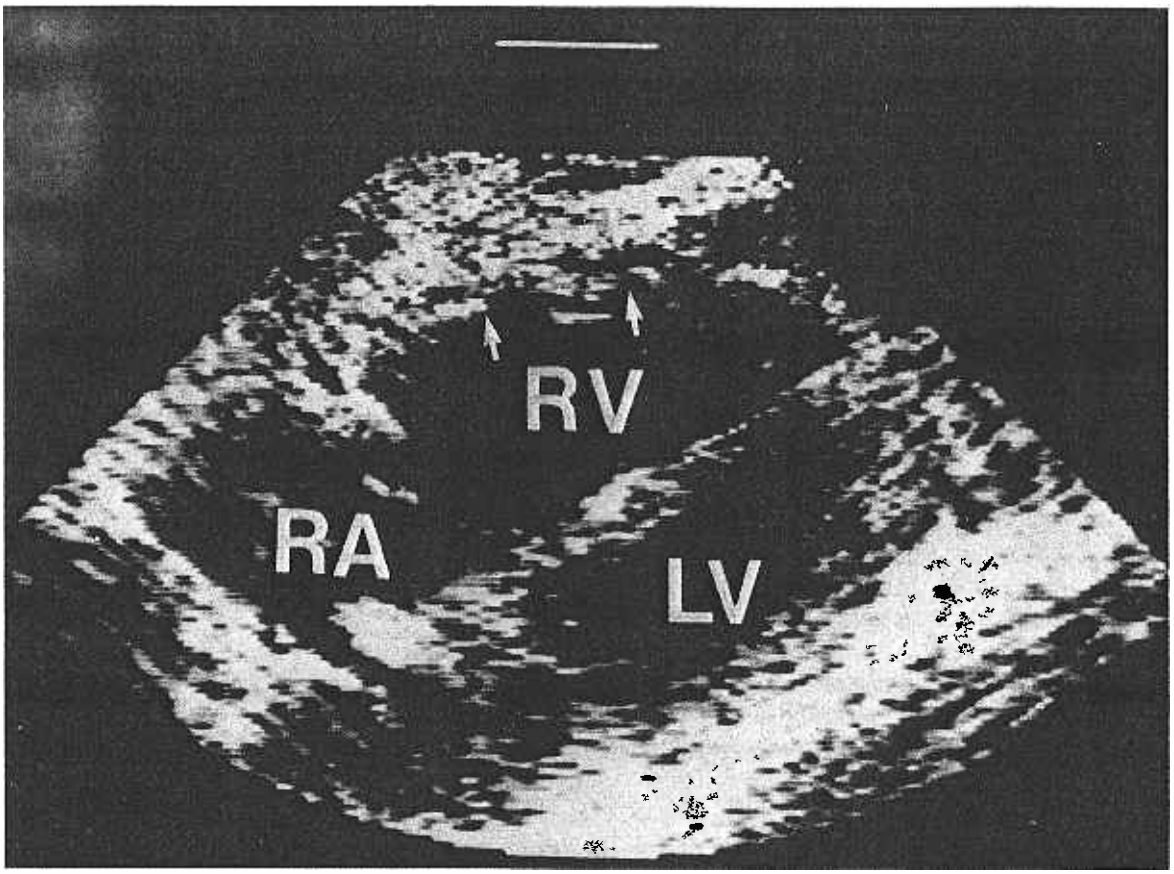

Fig. 5 Two dimensional echocardiogram from the subxiphoid view showing dilated right ventricle. In real time, the whole of the free wall of the right ventricle (indicated by the 2 white arrows) was noticed to be akinetic except for the apex. RV = right ventricle. LV = left ventricle. RA = right atrium.

group of workers have found a significantly greater right ventricular end diastolic dimension in patients with inferior myocardial infarction associated with right ventricular infarction. They also noted that in this group of patients with inferior myocardial infarction, 4 out of 5 cases associated with right ventricular infarction had a right ventricular end diastolic diameter to left ventricular end diastolic diameter ratio greater than 0.51 as was seen in our patient. In contrast, only 1 out of 9 patients without right ventricular infarction had a ratio greater than this. We also confirmed dilatation of the right ventricle employing cross sectional echocardiography using the subxiphoid approach. In addition akinesia of the right ventricular free wall presumably corresponding to the infarcted area was clearly seen from the real time cross sectional study. As far as we are aware, cross sectional echocardiography has not been widely employed for the diagnosis of right ventricular infarction, but this study clearly indicates that this technique deserves further evaluation.

In addition to the inferior surface of the left ventricle as well as the posterobasal interventricular septum, the right coronary artery also supplies the right ventricle. It is therefore not surprising that autopsy studies (8) and technetium pyrophosphate scintigraphic studies (5, 9) have shown that right ventricular infarction occurs commonly in association with infarction of the inferior and septal walls of the left ventricle. The incidence of right ventricular infarction in association with inferior or posterior myocardial infarction is 24% in autopsy studies (8) and 37.5% in technetium pyrophosphate studies (9). However, clinical evidence of this condition is much lower (11), suggesting that clinical manifestations of right ventricular dysfunction after right ventricular infarction is largely dependent on the impedance to right ventricular emptying viz pulmonary vascular resistance and left ventricular compliance. This is consistent with data showing near normal systemic venous pressures despite experimental destruction of the right ventricle (12-14).

The diagnosis of right ventricular infarction has important therapeutic implications. The presence of systemic venous engorgement frequently leads to the inappropriate use of diuretics. After right ventricular infarction, many patients, despite elevated right ventricular filling pressures, may have low left ventricular filling pressures resulting in a low cardiac output and hypotension. Injudicious use of diuretics will aggravate the situation further by lowering the left ventricular filling pressure. Awareness of the haemodynamic consequences of right ventricular infarction enables optimal management of this condition. In extensive right ventricular infarction, there may be no effective right ventricular contraction. Filling of the left ventricle will then depend on right atrial to left atrial pressure gradient (4). Therapy should be directed towards maintaining optimal left ventricular filling pressures. Despite elevated systemic venous pressures, fluids should be given to maintain left ventricular filling pressures and this has corrected

hypotension in several cases (11). It has been postulated that vasodilators may also improve the clinical situation by reducing pulmonary and systemic vascular resistances (11, 15) but the actual efficacy of this form of treatment must await future studies. If the correct therapy is given, patients with hypotension associated with right ventricular infarction generally have a much better prognosis compared to patients with cardiogenic shock due to severe left ventricular dysfunction (4). Because of this, the recognition of right ventricular infarction is of crucial importance in order to enable correct therapeutic decisions to be made.

Addendum

Since the publication of this paper, a study regarding the value of using lead V4R in addition to the standard 12 lead ECG has been described as being of great value in the diagnosis of right ventricular infarction (16). In this study, the ST segment in V4R was found to be elevated in patients with this condition. If this finding is subsequently confirmed, this would be a considerable aid to the diagnosis of right ventricular infarction.

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