Right ventricular infarction: a haemodynamic diagnosis¹

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Right ventricular infarction is a rarely recognized clinical entity. This report describes a patient who perished with a massive right ventricular infarction. The pathogenesis of right ventricular infarction is discussed. Haemodynamic studies in this patient suggest that an increase of right atrial mean pressure relative to any change in pulmonary artery diastolic pressure may be a strong haemodynamic indicator of right ventricular infarction.

The incidence of right ventricular infarction, based on necropsy studies, has been reported to be between 2 and 13 per cent of patients dying from coronary heart disease (Bean, 1938; Wartman and Hellerstein, 1948). In a recent pathological study from our institution (Ratliff and Hackel, 1974) the frequency of right ventricular infarction in association with left ventricular infarction was 34 per cent (35 of 102). Right ventricular infarction is only rarely recognized clinically and there are no agreed electrocardiographic criteria of this entity. The purpose of this report is to describe a patient who expired with a massive right ventricular infarction. Haemodynamic studies in this patient before his death revealed findings thought to be compatible with acute right ventricular failure.

Case report

A 61-year-old man presented to the emergency room on 27 September 1971 at 7.00 am with a 9-hour history of dizziness, shortness of breath, and severe substernal chest pain. While in the emergency room he was noted to have a blood pressure of 50/0 mmHg. An electrocardiogram taken at that time revealed ST segment elevation in leads II, III, and aVF, and first-degree heart block. No previous history of chest pain, myocardial infarction, hypertension, diabetes mellitus, or pulmonary disease was obtained.

The patient was transferred to the coronary care unit. Physical examination revealed a blood pressure of 88/60 mmHg, a pulse of 88 beats/min, and a respiratory rate of 40 per minute. The skin was cool, but was not diaphoretic. There was pronounced distension of the jugular veins with tall 'A' waves. The lungs were clear. Examination of the heart showed an apex impulse in the fifth

intercostal space, 5 cm to the left of the midsternal line. No abnormal praecordial activity was noted. The heart sounds were distant and an S4 gallop was heard at the apex. Examination of the abdomen was normal except for right upper quadrant tenderness. Peripheral oedema was absent.

On admission the patient's haemoglobin was 9.4 g/100 ml and the white blood cell count was 22,900/mm³ with a shift to the left. The serum levels were: sugar 180 mg/100 ml, BUN 26 mg/100 ml, Na 127 mEq/l., K 6.7 mEq/l., Cl 89 mEq/l., and CO₂ of 10 mm/l. Blood gases on nasal oxygen revealed a pH of 7.36, Po₂ of 159 mmHg, a PCO₂ of 11 mmHg, and bicarbonate of 6 mEq/l. Serial enzymes and electrocardiograms were compatible with an evolving diaphragmatic myocardial infarction. Radiographic evaluation revealed mild left ventricular enlargement and absent pulmonary congestion.

A Swan-Ganz flow-directed catheter (Ganz *et al.*, 1970) was used to obtain right-sided cardiac pressures. The pressures were measured with a Hewlett-Packard transducer (No. 1280C) and recorded on a Hewlett-Packard multichannel oscillograph. A point 10 cm above the bed or catheterization table was used as zero reference. Cardiac output was measured by the dye-dilution technique using indocyanine green. Dye curves were recorded on a Waters densitometer (No. XP-302) and were calibrated using known quantities of dye in the patient's blood. The cardiac outputs were calculated by the method of Thompson *et al.* (1964). Arterial and venous oxygen samples were measured on an Instrumentation Laboratories CO-Oximeter (No. 182). The haemodynamic data are presented in the Table. The initial study on 27 September 1971 at 1.00 pm revealed a right atrial mean pressure of 18 mmHg and a pulmonary artery diastolic pressure of 14 mmHg. The cardiac index was 3.1 l./min per m² and the AV O₂ difference was 6.9 vol per cent. Subsequent data continued to show a pronounced discrepancy between the right atrial mean pressure and the pulmonary artery diastolic pressure. There was progressive haemodynamic deterioration with

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TABLE *Haemodynamic data: a rise in right atrial pressure relative to pulmonary artery diastolic pressure is noted on all three observations*

Date	27.9.71	28.9.71	
Time	1.00 pm	9.30 am*	5.30 pm
Pressures (mmHg)			
Right atrium	a = 21 (18) v = 17	(16)	(22)
Right ventricle	25/5-14		
Pulmonary artery	25/14 (16)	18/8 (13)	25/9 (17)
Pulmonary artery wedge		(12)	(9)
Radial artery	92/64 (76)	84/60	84/36 (54)
Cardiac index (l./min per m ²)	3.1	2.7	
Stroke index (ml/beat per m ²)	30	27	
AV O ₂ difference (vol %)	6.9		7.8
Heart rate (beats/min)	100	95	105

* The patient was treated with an isoprenaline drip on 27 and 28 September. Isoprenaline was discontinued at the time of the observations indicated at 9.30 am on 28 September.

a fall in cardiac and stroke index as well as blood pressure. During the first 24 hours in hospital the patient responded to therapy which included nasal oxygen, an isoprenaline drip, and intermittent doses of intravenous frusemide, bicarbonate, and digitalis. Over the final 12 hours gradual deterioration occurred with a decrease in urinary output and an altered sensorium in spite of therapy with intravenous glucagon and noradrenaline. At 6.00 pm on 28 September 1971 the patient suffered a respiratory and cardiac arrest with ventricular fibrillation and could not be resuscitated.

At necropsy the heart was enlarged, weighing 475 g. The right atrial appendage contained a well-fixed mural thrombus. The right ventricle was moderately dilated and the trabeculae carneae flattened. The left atrial and left ventricular cavities were not remarkable. There was a recent transmural infarct at the base of the heart involving the posterior left ventricular wall and the posterior third of the interventricular septum. The infarct extended transmurally from endocardium to epicardium in the left ventricle and completely across the septum. The infarct also extended posteriorly into the thinned right ventricular wall for a distance of 5.5 cm, and well around to the lateral margin of the right ventricle (Fig.). The right coronary artery was dominant and its orifice was narrowed by approximately 75 per cent. The sinoatrial nodal artery originated about 1 cm distal to the orifice and was patent. There were multiple narrowed atheromatous lesions along the course of the right coronary artery with a point of 85 per cent narrowing 3 cm from the coronary orifice and a second narrowed

point of about 85 per cent narrowing in the posterior part of the right coronary artery 8 cm from the coronary orifice. In addition to the old atherosclerotic narrowing, the right coronary artery was completely occluded by a recent thrombus that filled the lumen from the coronary orifice to just 1 to 2 cm proximal to its posterior descending branch. The main left coronary artery was moderately involved by old atherosclerosis that narrowed the lumen by about 50 per cent. The left anterior descending and left circumflex coronaries were severely stenosed by old atheromatous plaques.

Discussion

To understand the pathogenesis of right ventricular infarction the normal vascular supply of the right ventricle and the distribution of the right coronary artery must be considered. Farrer-Brown (1968) studied the vascular patterns of the myocardium of the right ventricle in 50 human hearts. He found that the right coronary artery supplied all the right ventricular free wall except the anterior margin which was supplied by branches of the left anterior descending. The posterior descending branch of the right coronary artery supplied the posterior wall of the right ventricle as well as in the majority of hearts the diaphragmatic and posterior surface of the left ventricle. The right marginal artery of the right coronary supplied the lateral wall of the right ventricle. A pattern of perfusion with both branching and straight type arteries was found in the distribution of the right coronary artery, similar to the pattern of the left coronary artery. The left coronary artery was an important source of blood supply to the right side via a number of branches, the most important of which was the left anterior descending branch.

The most complete clinical and pathological correlative study of right ventricular infarction was described by Wade (1959). He reviewed the pathological studies of 11 cases and added 10 additional cases previously reported. The clinical description in 7 of his cases revealed the presence of right-sided congestive failure in 5. The electrocardiogram showed a diaphragmatic or posterior infarction in all cases. In contrasting the group of patients with right ventricular infarction with those with left ventricular infarction, some interesting differences were noted. Those patients with right ventricular infarction had a higher incidence of multiple infarctions, right-sided mural thrombi, pulmonary embolism, pericarditis, right ventricular hypertrophy, and chronic pulmonary disease. Examination of the coronary arteries revealed a 'severe' right coronary occlusion in all cases and 'moderate to severe' involvement in at least one or both remaining main coronary branches in most of the cases. The site of

FIG. A complete cross-section of the heart near the base of the heart. The acute myocardial infarction involved the posterior third of the interventricular septum and the posterior and posterolateral walls of the right ventricle. The arrows delineate the extent of the infarction. The infarcted myocardium is pale and contains extensive areas of haemorrhage. Haemorrhage is particularly evident in the left side of the posterior interventricular septum, in the subendocardial myocardium of the posterior wall of the left ventricle, and through the full thickness of the posterior wall of the right ventricle. The infarction is transmural in both left and right ventricle. Note the dilatation of the right ventricle and the thinning of the infarcted right ventricle, interventricular septum, and left ventricle. (A=anterior, P=posterior, R=right, L=left.)

infarction in the right ventricle was posterior in 18 cases and anterior in 3. The great proportion of the infarcts were transmural. The pathological data obtained in our patient conform to the findings noted by Wade.

The lower incidence of right ventricular infarction when compared to involvement of the left ventricle is probably due to a number of factors. These include: the lower metabolic requirements of the right ventricle since it is a low pressure chamber, the pattern of coronary flow in the right coronary artery being both diastolic as well as systolic, the rate and extent of collateral vessel development noted in experimental studies of right coronary artery occlusions when compared to occlusion of the left anterior descending, and the available collateral

vessels from the left coronary system (Gregg, 1950; Wiggers, 1954; Farrer-Brown, 1968; Ramo *et al.*, 1970). The pathogenesis of right ventricular infarction from pathological data in man (Wade, 1959) and from experimental data in the pig (Peter *et al.*, 1972) thus includes a severe degree of coronary atherosclerosis and the development of right ventricular hypertrophy.

Acute myocardial infarction predominantly involves the left ventricle. Since right ventricular function does not parallel changes in left ventricular function in the setting of acute infarction, the use of mean right atrial pressure or central venous pressure is not a reliable guide in determining the filling pressure of the left ventricle (Rapaport and Scheinman, 1969; Hamosh and Cohn, 1971;

Forrester *et al.*, 1971). More recent studies have shown that the level of pulmonary artery diastolic pressure is highly correlated with the filling pressure of the left ventricle (Kaltman *et al.*, 1966; Sapru, Taylor, and Donald, 1968; Falicov and Resnekov, 1970; Hamosh and Cohn, 1971).

A number of haemodynamic studies revealed a group of patients where the level of right atrial mean pressure was raised relative to any change in pulmonary artery diastolic pressure or left ventricular end-diastolic pressure (Fluck *et al.*, 1967; Lassers *et al.*, 1970; Hamosh and Cohn, 1971). Acute diaphragmatic or posterior myocardial infarctions were present in all these patients. In 5 of these patients studied pathologically, definite infarction of the right ventricle was seen in 4 and obstructive lung disease in 1. Thus the pronounced increase in right atrial mean pressure relative to any change in pulmonary artery diastolic pressure noted in our patient and other patients with diaphragmatic or posterior myocardial infarctions may be a manifestation of right ventricular dysfunction in the setting of right ventricular infarction.

There are certain clinical clues to the diagnosis of right ventricular infarction. These include a patient with a diaphragmatic or posterior infarction, associated with conditions that may have led to impaired right ventricular function, and the finding of right ventricular failure out of proportion to left ventricular failure. Though these clues may lead to a suspicion of right ventricular infarction the haemodynamic alterations described should increase the objectivity and accuracy in the diagnosis of acute right ventricular infarction.

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