

# Rupture of the Right Ventricle in Acute Myocarditis

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Rupture of the heart occurs almost always as a result of myocardial infarction and then usually in the left ventricle. Less frequently, ruptures are caused by bacterial infection, formation of abscesses, and aneurysms (Hudson, 1965). The diagnosis of rupture of the heart in this case was made before necropsy, thereby warning the dissector to avoid injury which has been suggested to be the cause of previously reported cases of rupture of the right ventricle (Bowen, 1962). The present case report is the first, as far as we know, of a ruptured right ventricle due to diffuse myocarditis of suspected viral aetiology.

## *Case Report*

The patient (F.G.), aged 38, was admitted to the Casualty Department of the Royal Melbourne Hospital on October 1, 1965. He had been well and was pronounced fit after routine medical examination in December 1958. His blood pressure then was 150/80 mm. Hg. In February and July 1959 he suffered recurrent sore throat and colds. Routine examinations in November 1962 and August 1964 revealed no abnormalities. There was no family history of cardiac disorder.

He had been well until four days before admission when he felt pains in the chest which disappeared and recurred two days later, with accompanying general vague aches and pains in the neck and shoulders. He consulted another doctor who recorded a pulse rate of 88, a blood pressure of 120/80 mm. Hg, no abnormalities, and diagnosed influenza. Later that day his chest felt tight and uncomfortable, aggravated by lying down and relieved by sitting up. He improved the following day. Four hours before admission he had epigastric pains associated with nausea aggravated by the slightest movement. He felt faint and short of breath. His doctor found him cyanosed with a temperature of 36.4°C., a pulse rate of 145 a minute and blood pressure of 90/60 mm. Hg, and arranged admission to hospital.

He was immediately transferred to the Coronary Care Unit with the provisional diagnosis of myocardial infarction. On arrival he was deeply cyanosed, took an occasional gasp of air, but peripheral pulses and heart sounds were absent. Both external jugular veins were

distended to the jaw and pupils were fully dilated. External cardiac massage was started immediately and artificial ventilation was given.

The electrocardiogram monitored by needle electrodes showed idioventricular rhythm. Sodium bicarbonate (200 mEq) and atropine 2.4 mg. were infused into the femoral vein after blood samples had been obtained. Rhythm remained idioventricular and isoprenaline hydrochloride 0.2 mg. was given slowly intravenously with a further 100 mEq of sodium bicarbonate, as the pH was reported to be 7.25. The rhythm changed to ventricular tachycardia. He remained cyanosed, pulseless, and without heart sounds, with continued distension of both external jugular veins, despite a good electrical rhythm. He was intubated, his condition remaining unchanged. After propranolol (5.0 mg. intravenously) the rhythm changed to sinus tachycardia, but soon reverted to ventricular tachycardia. D.C. countershock of 200 watt sec. resulted in slow idioventricular rhythm. The diagnosis of ruptured ventricle following myocardial infarction was made on the grounds of a good electric rhythm in the absence of peripheral pulses and heart sounds, and because of persistent venous distension and cyanosis, despite technically adequate massage and ventilation. Resuscitation was abandoned after 45 minutes.

At necropsy the positive findings were confined to the cardiovascular system.

The heart weighed 525 g. There were about 50 ml. of partly clotted, partly fluid blood in the pericardial sac. There was a rupture anteriorly at the beginning of the pulmonary artery pathway and close to the septum. The rupture showed frayed rims and haemorrhage into the adjacent epicardial fat (see Fig. 1, the white marker points to the rupture). The coronary arteries showed only minimal bright yellow atheromatous plaques without any occlusion. The muscle wall of the left ventricle was thickened, and showed a whitish ill-defined nodular swelling in the endocardial half of the muscle. The outer portion showed a reddish colour. This nodularity had to a great extent disappeared in the fixed specimen. In the anterior portion of the left ventricular muscle, small ill-defined areas of about 2 sq.cm. or so, partly fibrous, partly translucent, were present.

There were no congenital or acquired abnormalities of the valves of the heart and changes were confined to the heart. No other organ showed abnormality.

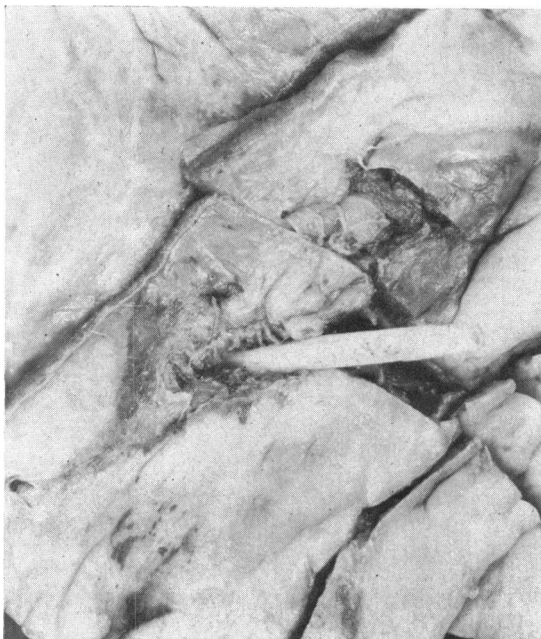


FIG. 1.—Glass tube pointing to rupture of the right ventricle.

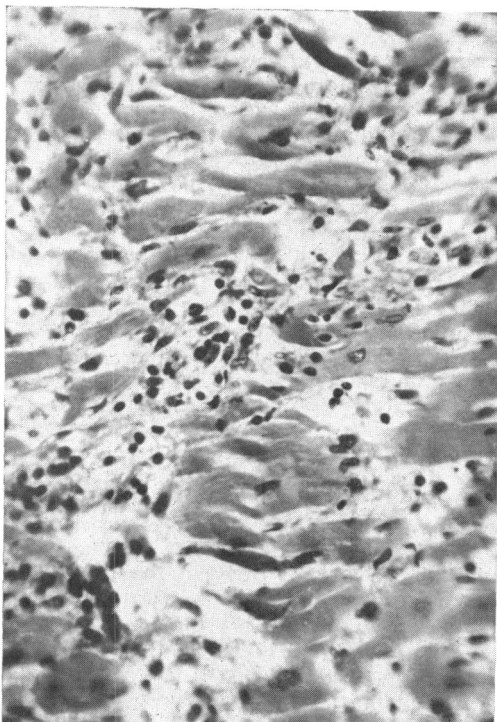


FIG. 2.—Microscopical section of left ventricular muscle showing inflammatory exudate. ( $\times 270$ .)

There was extensive cellular infiltration equally affecting the right and left heart. Microscopically the lesion was more diffuse than was expected from the apparently contrasting naked eye zones (Fig. 2). Essentially the appearances were rather uniform. They consisted of an interstitial exudate of small round cells, macrophages, and very rarely polymorphs. These cells were sometimes very numerous, sometimes scanty, and they did not disturb the pattern of the surrounding cardiac muscle. The exudate extended also into the epicardium, where the cells were either diffusely arranged or in small rounded foci. The muscle fibres showed normal cross striation and nuclei, but in areas where the inflammation was more severe many fibres had lost their nuclei and appeared more homogeneous, the cross striation no longer visible with crossed Nichol prisms. Other muscle fibres showed degenerative features of their nuclei. The damage to muscle fibres was generally more severe in the right ventricle. No bacteria were seen.

The pathological diagnosis was considered to be myocarditis with rupture of the right ventricle.

#### Discussion

Rupture of the ventricle was correctly diagnosed before necropsy on the evidence of persistent cyanosis, jugular venous distension, good electrical rhythm, and the absence of adequate peripheral pulsation, despite efficient external cardiac massage and artificial ventilation with oxygen.

The differential diagnosis in such cases of sudden unexpected death (excluding severe cardiopulmonary disease and cerebral haemorrhage) is massive pulmonary embolism. This was considered unlikely in the absence of predisposing conditions and because of the absence of detectable peripheral venous thrombosis.

Rupture of the heart in the absence of trauma in a man aged 38, with a short history of chest pain, was considered to be due to myocardial infarction. The only atypical clinical feature was the deep plum-coloured cyanosis. In our experience, myocardial infarction with ventricular rupture more commonly presents with pallor and cyanosis.

Retrospectively, the vague history of a "flu-like" illness four days before death suggested a virus infection, and the praecordial discomfort two days before death, relieved by upright posture and aggravated when supine, was compatible with pericarditis.

A viral aetiology though not proven by culture was supported by the microscopical findings of widespread patches of small cell infiltration and muscle cell degeneration and the absence of bacteria.

The episode of severe pain in the chest and epigastrium associated with nausea and aggravated by

any movement, with resulting faintness and shortness of breath, was consistent with rupture and minor leakage of blood into the pericardial cavity.

The necropsy findings of rupture of the right ventricle in an area of diffuse cellular infiltration and muscle degeneration, with partly clotted, partly liquid blood in the pericardium, were consistent with a diagnosis of ante-mortem rupture of the myocardium. The possibility of post-mortem traumatic injury by the dissector as suggested by Bowen (1962) was dismissed in this case as the pathologist was warned of the suspected diagnosis before the necropsy, and great care was taken in opening the chest. Rupture due to external cardiac massage was also considered unlikely because rupture had been suspected on clinical grounds and because this complication was not observed in necropsies following external cardiac massage on patients with extensive infarcts (Stock, 1966). Rupture due to myocardial infarction was excluded because of the presence of diffuse inflammatory infiltration affecting the entire heart.

This appears to be the first case report of ruptured right ventricle due to myocarditis of suspected viral origin. Spontaneous rupture of the right ventricle has been reported by Pulvertaft (1932) in a girl aged 19 with coal-gas poisoning. Howell and Piggot (1950) reported three elderly patients with spontaneous rupture of the right ventricle, and Knight (1962) found a further two elderly patients without evidence of myocardial pathology. None of these ruptures had been diagnosed before necropsy. Bowen (1962) suggested that they might have been caused by post-mortem trauma. Ruptured ventricle due to pyogenic abscesses has been described by Krumbhaar and Crowell (1925), who found three cases among 611 reported ruptures, and Davenport (1928) found a further two patients with similar pathology in a review of a further 92 cases. Weiss and Wilkins (1937) described two cases of ruptured ventricle associated with *Staph. aureus* in one and pneumococcus in the other. They found only 7 reported cases of rupture of the heart due to abscesses. Sossai (1946) described a further case of ruptured heart due to an abscess.

Davenport (1928) referred to the clinical silence of myocardial abscess until perforation had occurred,

which he stated was accompanied by low thoracic pain and collapse. The clinical progress of this case also showed remarkable absence of symptoms and signs referable to the heart until two days before death when symptoms of pericarditis appeared. The pain associated with the rupture was epigastric with associated nausea and deterioration on movement. The appearance of this clinical picture should direct close attention to clinical and electrocardiographic abnormalities of the heart.

#### Summary

The clinical and necropsy findings of a case of rupture of the right ventricle following acute myocarditis are presented.

The significance of the diagnosis before necropsy is discussed in relation to the differential diagnosis of the cause of ventricular rupture.

Review of the published reports appears to indicate that this is the first case of right ventricular rupture due to myocarditis of suspected viral aetiology.

Attention is directed to the need for the early recognition of the clinical features of the condition.

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