ELECTROCARDIOGRAPHIC STUDIES VI

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Case 6. Constrictive Pericarditis with Pericardiectomy Followed by Intramyocardial Haemorrhage

This patient was a Coloured male aged 56. He first consulted a doctor in June 1959 when he complained of swelling of the lower extremities as well as swelling of his face and of his abdomen. These symptoms had been present for approximately 3 months. He also admitted to having had some precordial pain not related to effort. Physical examination at that time revealed the presence of distended jugular veins without pulsations, a pulsus paradoxus, generalized anasarca with ascites, and a liver which was markedly enlarged. The heart was increased in size with soft heart sounds; no murmurs and no third heart sound were audible.

The patient had a raised temperature.

Blood examinations. Haemoglobin 11·5 g.%, white cell count 6,900 per c.mm., and ESR 103 mm./first hour (Westergren). Normal differential count.

Urine. No abnormalities were present on routine examination.

X-ray of the chest. The heart is markedly enlarged and flask shaped. The lungs showed bilateral apical infiltrations suggestive of an old tuberculous lesion. No congestive changes were observed in the lung fields.

The diagnosis of active tuberculous pericarditis with tamponade and commencing constriction, was made.

A pericardial aspiration was carried out—1,000 ml. of haemorrhagic fluid was removed and air was instilled into the pericardial sack. Subsequent X-rays of the chest demonstrated a thickened pericardium.

The patient was treated by keeping him in bed and prescribing an anti-tuberculous regimen. During the ensuing days and weeks the clinical evidence of constriction increased with a further rise in jugular venous pressure and a drop in the systemic blood pressure from levels of 120/80 to 95/85 mm. Hg. The anasarca with ascites and enlargement of the liver persisted.

Cardiac catheterization was carried out on 7 August 1959 and the catheter findings confirmed the presence of the constrictive phase of pericarditis.

Surgery was advised and carried out on 19 August 1959. Much thickened parietal and some visceral pericardium was freed from the heart. Bleeding during the procedure from the operative site was quite troublesome.

The operation was completed but the post-operative condition of the patient deteriorated. He remained in a hypotensive state and died on 20 August 1959.

The pre- and post-operative electrocardiographic findings are of particular interest in relation to the autopsy findings.

ELECTROCARDIOGRAM

Fig. 1: Pre-operative. Sinus rhythm 100 per minute. Mild left axis deviation. Semi-horizontal heart position with an early transition between leads V_1 and V_2. P wave in standard lead 2 was 3 mm. in amplitude and 0·1 sec. in duration, and suggested right auricular hypertrophy. PR not prolonged. QRS normal in duration, amplitude and pattern. No deviation of ST segment. Symmetrical T wave inversion in all leads facing the epicardial surface of the heart, namely in standard leads 1, 2, 3 and in aVL, aVF and leads V_2 to V_6.

Conclusion. These electrocardiographic findings are typical of the constrictive phase of tuberculous pericarditis.

Fig. 2: Post-operative. Sinus rhythm 110 per minute. T wave still that of right auricular hypertrophy. Right axis deviation. Vertical heart position. Transition between V_3 and V_4. QRS pattern is now markedly changed with great reduction in amplitude and a QS pattern in leads facing the anterolateral aspect of the heart, namely in standard leads 1, aVL and V_4 to V_6. ST segments displaced in an upward direction of the leads facing the anterolateral aspect with the T wave taken up in the ST segments.

Conclusions. The electrocardiogram was now indicative of a recent through-and-through anterolateral myocardial infarction.

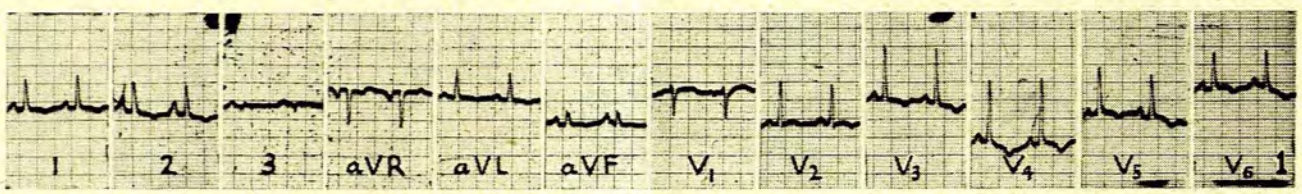

Fig. 1

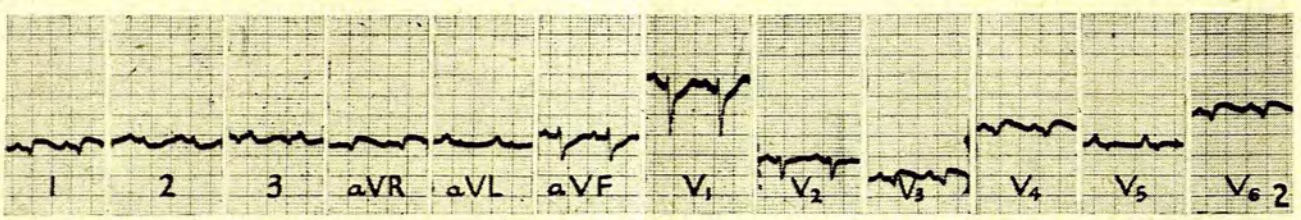

Fig. 2

DISCUSSION

The pre-operative electrocardiogram was typical of the constrictive phase of tuberculous pericarditis. The patient's post-operative electrocardiogram suggested the presence of a recent anterolateral myocardial infarction. This was thought sufficient to explain the post-operative hypotensive state which resulted in the patient's death.

The autopsy confirmed the tuberculous pericarditis and revealed extensive massive haemorrhage into the substance of the anterolateral wall of the left ventricle. The haemorrhage replaced practically the whole of the thickness of the left ventricular muscle. This finding explained the electrocardiographic appearance of myocardial infarction. The mechanism of the haemorrhage could not be explained satisfactorily.