Electrocardiographic features suggestive of a left ventricular aneurysm following a high-velocity missile injury

A case report

T. H. DIAMOND, R. SMITH, D. P. MYBURGH, L. STEINGO

Summary

Electrocardiographic features suggestive of a transmural anterior myocardial infarction with resultant left ventricular aneurysm formation were found in a 22-year-old man who had sustained a ballistic missile injury to his chest.

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High-velocity missile injuries to the heart may either be caused directly by the missile or by shrapnel (haemopericardium, tamponade or exsanguination from myocardial lacerations), or indirectly by the transmission of the high energy of the bullet to the nearby myocardium.[1-4] This 'shock wave' may extend up to a distance of 20 times the diameter of the missile.[5] Any injury of this type occurring within the left hemithorax may therefore result in massive myocardial necrosis.[1,6,7] The latter would present early as cardiogenic shock or rhythm disturbances, or months later as myocardial necrosis mimicking an extensive myocardial infarction.[1,6,8]

The electrocardiographic and echocardiographic findings in a case of the late complications of high-velocity missile injury to the heart, viz. 'shock wave' myocardial necrosis with secondary aneurysm formation, are presented.

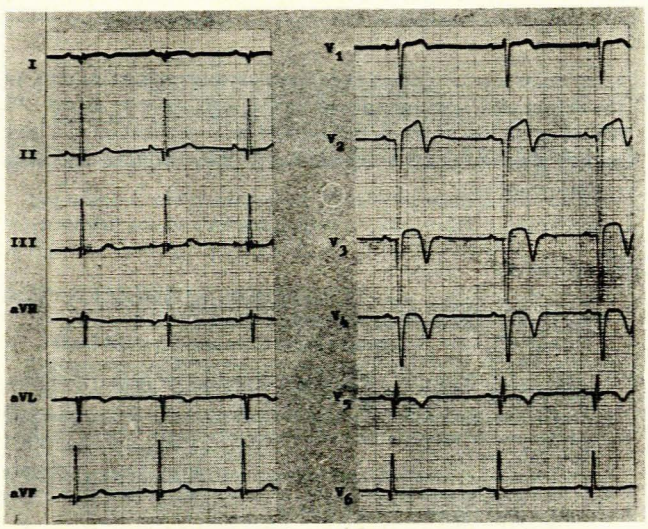

Fig. 1. Resting ECG illustrating persistent ST-segment elevation.

Institute for Aviation Medicine, Verwoerdburg, Tvl
T. H. DIAMOND, M.B. B.CH.
R. SMITH, M.B. B.CH.
D. P. MYBURGH, *SM.*, M.B. CH.B., F.A.C.C.
L. STEINGO, M.B. B.CH., F.C.P. (S.A.) (Present address: Cardiology Dept, University of Witwatersrand, Johannesburg)

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Case report

A 22-year-old White man applying for a private pilot's licence was investigated after having been diagnosed on electrocardiographic criteria as having had an extensive anterior myocardial infarction. He had been healthy until 48 months previously, at which time he had been admitted to hospital as a victim of a sniper with an Armourlite assault rifle in Ireland. He had received a single bullet injury to the left hemithorax. Further details were vague, but he underwent an emergency left explorative thoracotomy. Large vessels were sutured and shrapnel fragments were removed, but no cardiac surgery was performed. The only immediate postoperative complication was respiratory failure. The patient was artificially ventilated for 2 weeks, and thereafter made a slow recovery with no further complications. No evidence of cardiac damage was revealed and the patient was discharged. Electrocardiographic findings were not available to us.

His history negated any cardiovascular impairment, angina or limitation in effort tolerance. He plays regular contact sport such as soccer and rugby. Risk factors for atherosclerosis included his smoking (10 cigarettes per day for the previous 5 years).

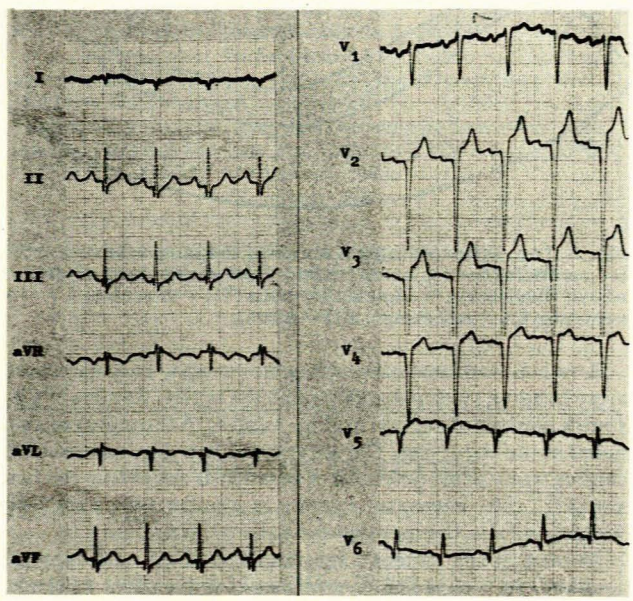
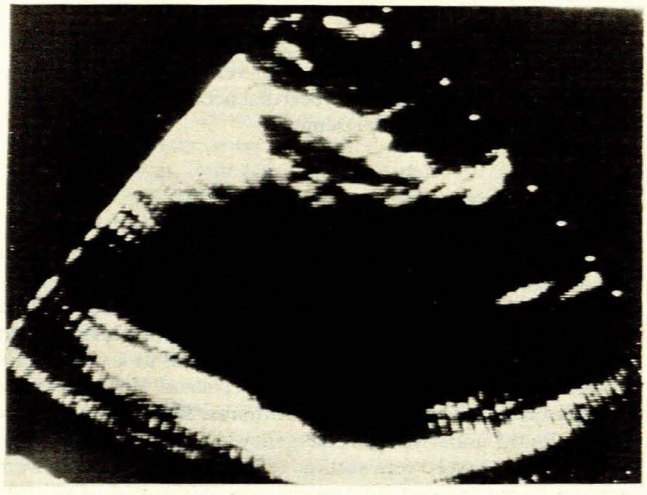


Fig. 2. Stress ECG illustrating exercise-induced ST-segment elevation.

The patient looked clinically healthy and was well built. The heart rate was 75/min and the blood pressure was 108/70 mmHg. There were no signs of cardiac failure. A left parasternal cardiac impulse was palpable. On auscultation, in addition to the normal heart sounds, a fourth heart sound was audible. The rest of the clinical examination was negative.

The ECG (Fig. 1) recorded 48 months after injury showed deep Q waves in leads I, aVL and V2-V6, with ST-segment elevation in leads V2-V4, compatible with an extensive anterior myocardial infarction or necrosis. A stress ECG (Fig. 2) at the time showed a further increase in ST-segment elevation in leads V2-V5, thereby increasing the likelihood of his having an associated left ventricular aneurysm.[9] A 24-hour Holter ECG was non-contributory and a chest radiograph was within normal limits.



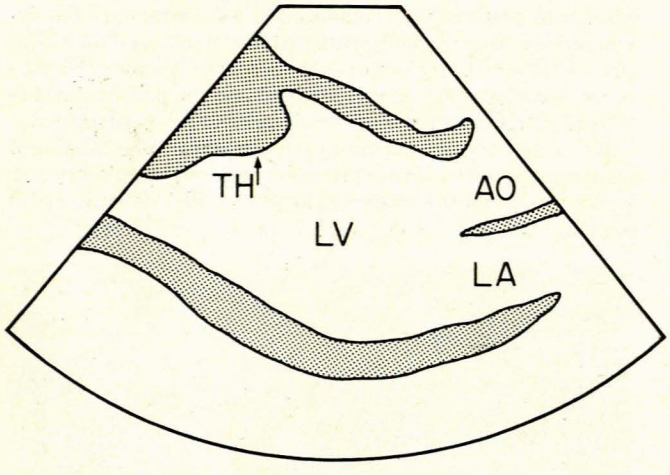
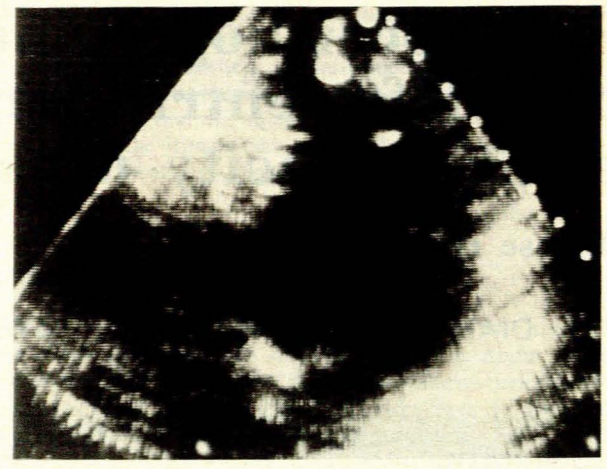


Fig. 3. Top: **echocardiogram showing the long-axis view of the left ventricle (apex towards the left).** Bottom: **idealized diagram of above, illustrating the apical thrombus (TH = thrombus; LV = left ventricle; LA = left atrium; AO = aorta).**

A two-dimensional echocardiogram using a Toshiba phased-array imaging system demonstrated the typical features of a left ventricular apical aneurysm (Figs 3 and 4). A thrombus was also noted in the aneurysm. The accuracy of two-dimensional echocardiography in the detection of ventricular aneurysms has been noted.[10,11] The aneurysm appears as a bulge with a shelf-like separation from the normal ventricular myocardium.

In view of the patient's age, history and the cardiovascular findings, we would like to postulate that the cause of the left



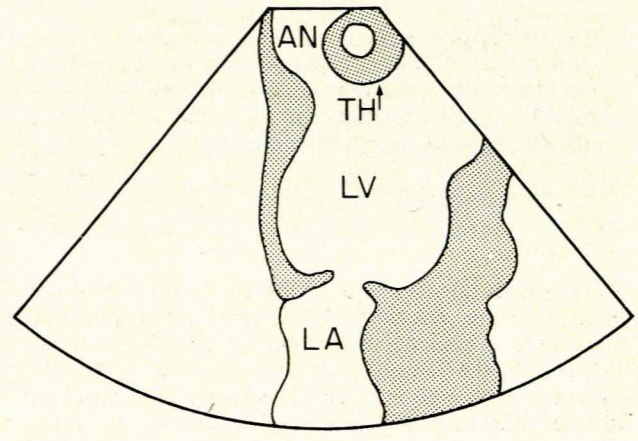


Fig. 4. Top: **echocardiogram showing an apical four-chamber view.** Bottom: **idealized diagram of above, illustrating the presence of a calcified thrombus in the apical aneurysm of the left ventricle (AN = aneurysm).**

ventricular aneurysm and the electrocardiographic findings was the myocardial necrosis caused by the high-velocity missile. We would again like to stress this important complication of a high-energy missile injury — 'shock wave' tissue necrosis and aneurysm formation.

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