SPONTANEOUS HAEMOPERICARDIUM

H. GRUSIN, M.B., B.CH. (RAND), M.R.C.P. (LOND.)

Lately Physician, Baragwanath Hospital, Johannesburg

Haemopericardium as opposed to haemorrhagic pericarditis may be due to trauma or disease causing rupture of a vessel or heart chamber into the pericardium, to haemorrhagic states such as leukaemia, or to neoplasms of the heart or pericardium.[1] In this paper 2 cases of haemopericardium are reported for which no obvious cause could be found.

CASE REPORTS

Case 1

J.N., a male African aged 43, was admitted to Baragwanath Hospital complaining of backache and haematuria for one week. He was dyspnoeic and had the signs of congestive cardiac failure, namely raised jugular venous pressure, moderately enlarged tender liver, and sacral and pedal oedema. The heart was enlarged to percussion and the sounds muffled. He had cold hands and a paradoxical pulse; blood pressure 120/100 mm. Hg. The electrocardiogram showed QRS complexes of normal voltage and non-specific T-wave inversions.[2]

The following abnormal signs were also found: temperature 99·6°F; spongy, bleeding gums; haematuria but no casts (blood-urea normal); tenderness over the mid-dorsal spine (X-ray showed osteoporotic collapse of several vertebrae).

A bedside X-ray showed globular enlargement of the heart shadow (Fig. 1). The pericardial sac was tapped and 120 ml. of blood-stained fluid withdrawn. Repeat X-ray after pericardial aspiration showed a thin parietal pericardium (Fig. 2). The aspirated fluid contained 6·6 g.% of haemoglobin and numerous red blood-cells. There were no organisms or inflammatory cells; the result of guinea-pig inoculation was negative. The triad of spongy gums, haematuria, and haemopericardium, suggested that

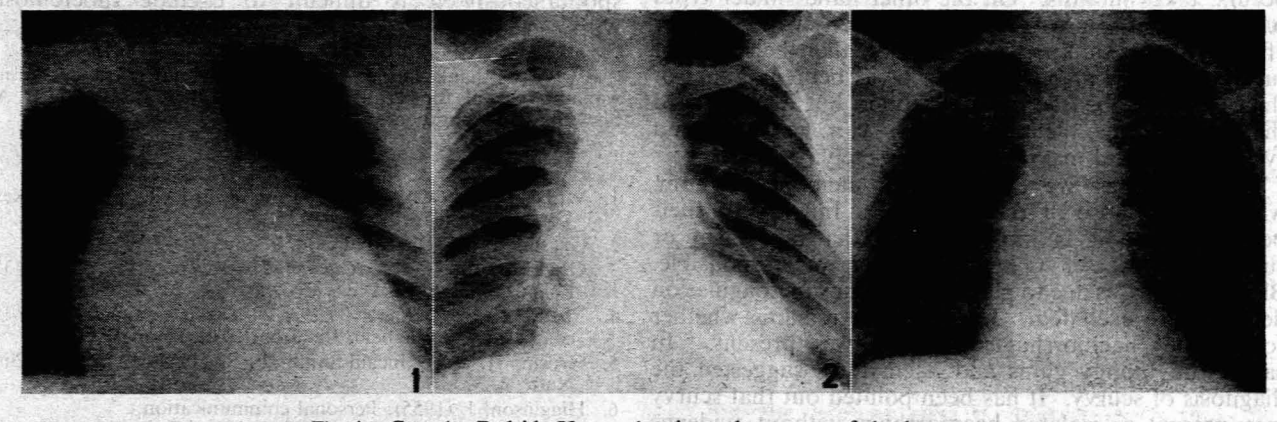

Fig. 1. Case 1. Bedside X-ray, showing enlargement of the heart.

Fig. 2. Case 1. X-ray after aspiration, showing thin parietal pericardium.

Fig. 3. Case 1. X-ray 2 weeks after aspiration. Two years later the appearances were unchanged.

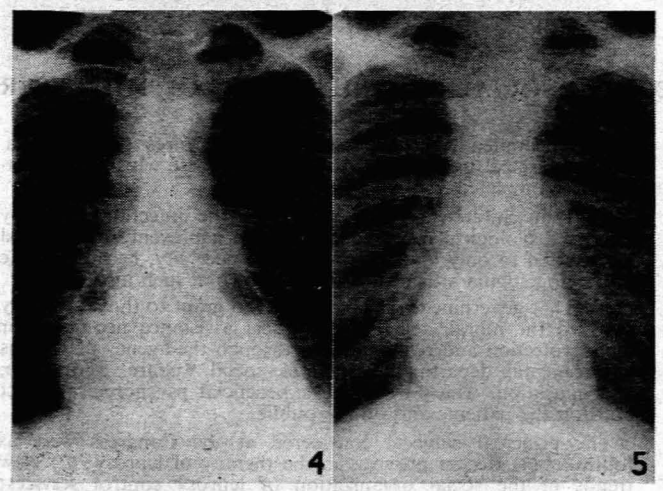

Fig. 4. Case 2. X-ray after aspiration.

Fig. 5. Case 2. X-ray 2 weeks later. Subcutaneous emphysema appeared after pericardial aspiration.

the patient might be suffering from a bleeding disease. However, there was no purpura and the blood count, platelet count, bleeding time and coagulation time lay within normal limits. The patient denied taking any drugs before admission. There was no history of trauma. The Mantoux test 1/1,000 was positive.

On the assumption that he might be suffering from scurvy he was treated with ascorbic acid, 500 mg. *per diem* intramuscularly. Within 10 days he had lost all his abnormal signs and his heart had returned to normal size (Fig. 3). His gums improved, his haematuria cleared rapidly, and renal function tests and pyelography revealed no abnormality. Two years later he had no clinical or radiological evidence of cardiac or renal disease. His spine was still osteoporotic.

Case 2

J.T., a male African aged 32, was admitted to Baragwanath Hospital complaining of chest pain and haemoptysis for one week. He was febrile (100°F), dyspnoeic, and in a state of mild congestive cardiac failure. The heart was not obviously enlarged but the sounds were muffled. He showed striking pulsus paradoxus. The electrocardiogram showed changes consistent with the presence of pericardial fluid, i.e. low voltage QRS complexes, with flattening or mild inversion of the T waves in all leads. Radioscopy revealed a big motionless heart shadow. One hundred ml. of blood-stained fluid were removed by pericardial tap; the parietal pericardium was thin (Fig. 4). As in the previous case there were

no clinical or laboratory signs of a bleeding disease and no evidence of infection in the pericardial fluid. There was no history of trauma or previous medication. The Mantoux 1/1,000 was positive.

The patient was maintained on a vitamin-C-free diet and given no other treatment. Within a fortnight he had recovered completely by clinical and radiological standards (Fig. 5) and has not relapsed after 2 years.

DISCUSSION

The first question to decide was whether these 2 patients were suffering from haemopericardium or haemorrhagic pericarditis. The clinical features and laboratory findings were against rheumatic or uraemic pericarditis. The problem was whether low-grade tuberculous pericarditis could be excluded; in Africans the commonest causes of haemopericardium are trauma and tuberculous pericarditis. The clinical course of tuberculous effusion is quite different from that described in these 2 patients. The onset is usually insidious, there is constitutional disturbance, the effusion is massive, the parietal pericardium is thick and resolution, when it occurs, takes months. On the other hand milder types of tuberculosis are occasionally seen with smaller effusions and a thin pericardium. Whether the course of tuberculous pericarditis can ever be as benign as that of the cases described here is not certain but it is believed that tuberculous pleural effusions can be small and symptomless and may absorb completely.[3,4]

If these cases are examples of haemopericardium, what is the cause of the bleeding? In view of their benign course it is most unlikely that neoplasm or rupture of a vessel or heart chamber was responsible. Bleeding diseases due to platelet deficiency or coagulation defects were excluded. The question remains whether some other haemorrhagic diathesis was present. In case 1 the spongy gums and haematuria suggested the diagnosis of scurvy. It has been pointed out that scurvy may present as isolated haemorrhage without evidence of bleeding elsehwere.[5] Such haemorrhage is usually seen in the gums or the muscles of one leg, and is unusual in serous cavities apart from joints. In a series of 40 scorbutics in this hospital haemorrhagic effusions into serous cavities were not encountered and, conversely, in patients with haemorrhagic pleural or pericardial effusions other evidence of overt scurvy was rarely found. In case 2 the diagnosis of scurvy is untenable unless the patient had access to vitamin-C-containing foods unbeknown to us.

In Africans an occasional case of haemorrhagic pleural or pericardial effusion with thin parietal pericardium is encountered, for which no cause can be found at post-mortem.[6] Full blood-studies have not been done in all these cases; moreover, it is known that even in tuberculous effusions evidence of infection may not be found microscopically unless serial sections are painstakingly examined.[7]

This paper is intended to draw attention to the fact that haemorrhagic pericardial effusions in Africans occur for no obvious cause. The occasional example may be due to scurvy but in other cases which apparently occur spontaneously it is difficult to exclude tuberculosis with certainty.

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