Pericarditis in diabetic ketoacidosis

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A 25-year-old insulin-dependent diabetic man who was admitted to hospital with severe diabetic ketoacidosis and dehydration showed sequential electrocardiographic abnormalities of acute pericarditis. Though the patient had retrosternal chest pain, no pericardial friction rub was heard. None of the usual causes of pericarditis was found and the electrocardiographic abnormality may have been attributable to subepicardial injury caused by dehydration associated with the ketoacidosis. The abnormalities on the electrocardiogram were transient, returning to normal after 5 days. Whatever the exact underlying nature of the pericarditis, it is important to recognise that such transient changes may occur as, in the absence of other obvious causes of pericarditis, the condition is benign.

We report a 25-year-old diabetic man, admitted to hospital with ketoacidosis, whose electrocardiogram showed sequential changes of acute pericarditis. The exact aetiology of the pericarditis was not identified and these electrocardiographic abnormalities may be attributable to the abnormal metabolic state, similar to the so-called 'pseudo-pericarditis', as described in the only previous report by Bennett and Blake (1971).

Case report

A 25-year-old man with insulin-dependent diabetes of 16 years' duration, who had had several previous episodes of ketoacidosis, was admitted to hospital with a 6-day history of increasing thirst and polyuria. There was no history of infection or other illness. His usual insulin regimen was 20 units soluble insulin and 40 units protamine zinc insulin. On examination he was drowsy and disoriented, very dehydrated, and had Kussmaul's respirations with a strong odour of ketones on the breath. The rectal temperature was 34.6°C, pulse 120/minute, and blood pressure 110/70 mmHg (14.6/9.3 kPa). Auscultation of the heart and lung fields was normal. The abdomen was soft and bowel sounds were present.

Initial investigations showed a blood glucose of 41.3 mmol/l (750 mg/100 ml), plasma bicarbonate 5 mmol/l, blood urea 18.6 mmol/l (112 mg/100 ml), plasma sodium 131 mmol/l, potassium 5.1 mmol/l, with a calculated serum osmolality of 335 mosmol/kg. The haemoglobin was 13.8 g/dl, erythrocyte sedimentation rate (Westergren) 21 mm in the first hour, platelet count $150 \times 10^9/l$, and there was a moderate leucocytosis of $15.2 \times 10^9/l$ (neutrophils 77%, lymphocytes 22%, and monocytes 1%) which returned to $5.8 \times 10^9/l$ after 48

hours with treatment of the ketoacidosis. There was no evidence of any precipitating infection clinically, and blood cultures, throat swab, and midstream urine specimens were all sterile. During the first 24 hours he received 8.5 l intravenous fluids (1.0 litre 0.45% saline, 2.0 litre 0.9% saline, 1.5 litre 5% dextrose-saline, and 4.0 litre 5% dextrose), with 1000 units soluble insulin and 195 mmol potassium added to the intravenous infusion. No bicarbonate or antibiotic therapy was administered. After the initial 24-hour period, the patient became fully conscious and oriented and the biochemical findings were blood glucose 4.7 mmol/l (85 mg/100 ml), plasma bicarbonate 21 mmol/l, blood urea 4.6 mmol/l (28 mg/100 ml), plasma sodium 135 mmol/l, potassium 5.3 mmol/l, with a calculated serum osmolality of 290 mosmol/kg.

After recovery the patient complained of central chest pain which had started 24 hours before admission. This pain was very sharp in character, worse on deep inspiration, and after the initial 24-hour period of intravenous fluid therapy rapidly improved. At no time was a pericardial rub audible. Chest x-ray examination was normal, with no evidence of pneumomediastinum. On admission the electrocardiogram had shown definite ST elevation in leads I, V2-V6; after 48 hours the ST elevation had largely resolved, and after 96 hours the electrocardiogram showed T wave inversion in leads III, aVF, and V4-6 and after a further 24 hours the electrocardiogram had returned to normal (see Fig.). The patient had had a normal electrocardiogram during a previous admission six months earlier for less severe diabetic ketoacidosis. Cardiac enzymes (AST, ALT, and urea-stable LDH) repeated on 3 occasions were normal, bilirubin 13.7 μ mol/l (0.8 mg/100 ml), alkaline phosphatase 71 units/l, uric acid 0.3 mmol/l (4.9 mg/100 ml), total serum protein 57 g/l, albumin 32 g/l, with normal electrophoretic pattern, antinuclear factor negative on

FIG. Serial electrocardiograms of patient with ketoacidosis showing characteristic ST elevation of pericarditis in leads I, V2-V6 in admission tracing (top), with less apparent changes after 48 hours (middle), and at 96 hours (bottom) only T wave inversion in III, aVF, and V4-6.

two occasions, ASO titre less than 200 Todd units per ml, with negative rheumatoid arthritis latex agglutination and Rose Waaler tests. Viral antibody studies on paired sera for the Coxsackie group, influenza A and B virus, para-influenza, adenovirus, mumps, mycoplasma, and Q fever were negative. The patient had received BCG vaccination some 11 years earlier.

One week after admission the patient was discharged on 20 units soluble and 40 units protamine zinc insulins, and when seen three months later remained in good health.

Discussion

The transient electrocardiographic abnormalities observed in diabetic ketoacidosis include ST depression, prolongation of the QT interval, abnormalities of the T wave, and prominent U waves. The changes observed are not fully understood but are believed to be related to the stage of the metabolic disturbance and especially the plasma potassium concentration (Friedberg, 1966). The ST segment elevation with retention of natural concavity, characteristic of pericarditis, is not a recognised electrocardiographic abnormality of ketoacidosis. Bennett and Blake (1971) have, however, described electrocardiographic abnormalities suggestive of pericarditis in 7 cases of diabetic ketoacidosis. No patient had chest pain, only one had a transient

pericardial rub, all had a normal chest x-ray, and the abnormalities on the electrocardiogram lasted only 36 to 48 hours, and because of the benign, non-infective nature of the apparent pericarditis they coined the term 'pseudopericarditis'. To our knowledge no other similar reports have been published. However, as the electrocardiographic abnormalities are those of pericarditis, we would prefer the latter term. Bennett and Blake (1971) considered the electrocardiographic abnormalities were possibly the result of subepicardial injury caused by dehydration with loss of pericardial lubricant. Though the abnormalities alternatively could be a result of acidosis giving rise to subepicardial myocardial metabolic changes, severe metabolic acidosis itself has not been shown to produce ST segment shifts (Stewart *et al.*, 1965). Armanino and Ory (1946) reported 5 cases of diabetic ketoacidosis with acute dry pleurisy, associated with pleural pain and friction rub, which completely resolved with rehydration. Pneumomediastinum may also give rise to chest pain in diabetic ketoacidosis (McNicholl *et al.*, 1968) but chest x-ray examination was normal in our patient.

No cause for a possible acute pericarditis was found in our case and though the diagnosis may well have been a non-specific (idiopathic) pericarditis with failure to isolate a causative virus, the transient,

self-limiting sequence of events is against this (Wood, 1968). The serial cardiac enzymes, which may be non-specifically raised in some instances of diabetic ketoacidosis (Knight *et al.*, 1974), were normal.

Although this is the first such case of pericarditis in our own experience of diabetic ketoacidosis, we feel it is important, whatever the exact underlying aetiology of the electrocardiographic abnormalities, to recognise that such transient changes may occur. The condition is possibly the result of dehydration injury and, in the absence of other obvious causes of pericarditis, is benign.

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