Recurrent alcoholic ketoacidosis with hyperglycaemia in a non-diabetic patient

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INTRODUCTION

Alcoholic ketoacidosis is a well-known complication of excessive alcohol consumption (Dillon et al, 1940). It is generally regarded as rare, but may be more common than is often appreciated (Thompson et al, 1986). The condition is distinct from diabetic ketoacidosis, although occasionally plasma glucose levels may be slightly or moderately elevated, causing diagnostic difficulties (Hart and Frier, 1996). This article presents an unusual case of an alcoholic man with recurrent attacks of alcoholic ketoacidosis with hyperglycaemia, without demonstrable diabetes.

DISCUSSION

The patient detailed here had a strong history of excess alcohol consumption, and suffered two episodes of severe illness marked by metabolic acidosis and heavy ketonuria. Blood for serum ketone estimation was not taken early in either admission, but on the second occasion a serum lactate was measured and was only moderately elevated at 3.9 mmol/litre — not sufficient to explain the degree of acidosis. The clinical and investigative findings are consistent with the diagnosis of alcoholic ketoacidosis.

The patient was unusual in two respects — first that he experienced two similar events in a period of 4 months and second that both episodes were associated with mild transient hyperglycaemia, despite the patient being demonstrated not to have diabetes by current World Health Organization criteria (Expert Committee, 1985).

Alcoholic ketoacidosis may be recurrent in the same patient (Dillon et al, 1940; Cooperman et al, 1974), but the association with hyperglycaemia is unusual (Hart and Frier, 1996; Cooperman et al, 1974), plasma glucose levels usually being normal or low.

Hyperglycaemia with ketoacidosis obviously rises a diagnostic problem, as the patient may have diabetic ketoacidosis (Simpson and Cundy, 1992), and as the hyperglycaemia is generally mild (<15 mmol/litre), the 'euglycaemic' variety of diabetic ketoacidosis is a particular possibility (Munro et al, 1973). However, a history of previous alcohol abuse, no history of preceding diabetes, and consideration of the possibility of alcoholic ketoacidosis, should help resolve the dilemma. Nevertheless, diagnostic difficulties do occur, and patients with alcoholic ketoacidosis

CASE REPORT

A 39-year-old man was admitted urgently with abdominal pain and vomiting to a surgical ward. He had a history of excessive alcohol intake and previous attacks of acute alcohol-related pancreatitis. Examination was unremarkable apart from a tachycardia of 120/minute in sinus rhythm and generalized abdominal tenderness. Investigations revealed normal haemoglobin, urea and electrolyte levels. White cell count (WCC) was 11.5/mm^3 and plasma glucose 13.1 mmol/litre (there was no past or family history of diabetes). Urine was heavily positive for ketones. Serum amylase was normal. Arterial blood gases showed a metabolic acidosis (pH 7.18, pCO_2 2.1 kPa, pO_2 18.7 kPa, base deficit 19 mmol/litre and standard bicarbonates 10 mmol/litre). No definite diagnosis was made, but the patient was treated with intravenous 0.9% sodium chloride (NaCl) infusion, and his symptoms settled and the blood gases normalized spontaneously over the next 1–2 days. Apart from the initial raised plasma glucose, all subsequent levels were normal.

Three months later the patient was again admitted urgently with abdominal pain and vomiting, this time to a medical ward. In the period since his last admission he had continued to drink excessive amounts of alcohol, and had had a particularly heavy 'binge' over the preceding 2–3 days. On examination he was moderately dehydrated and tachycardic, with hyperventilation and generalized abdominal tenderness. Serum amylase and electrolytes were normal, but urea and creatinine were slightly raised at 8.1 mmol/litre and 142 μmol/litre respectively. WCC was 22.1/mm^3 and urine was heavily positive for ketones. Plasma glucose was 13.9 mmol/litre. Arterial blood gases again showed a metabolic acidosis (pH 7.22, pCO_2 1.5 kPa, pO_2 18.5 kPa, base deficit 20 mmol/litre and standard bicarbonate 8 mmol/litre). A serum lactate level was taken at the time and later returned at 3.9 mmol/litre. A diagnosis of either euglycaemic diabetic ketoacidosis or alcoholic ketoacidosis was made. He was treated with 0.9% NaCl intravenous rehydration, as well as a glucose–potassium–insulin infusion. He improved clinically and biochemically over the next 2 days, and again, apart from the initial raised plasma glucose level, all subsequent levels were normal.

Six weeks after the second admission an outpatient glucose tolerance test was performed, with the patient on a free diet and no medication. The fasting plasma glucose level was 7.3 mmol/litre, and the plasma glucose level 2 hours after a loading dose of 75 g of glucose was 6.4 mmol/litre. Over the subsequent 2 years the patient had 3 further hospitalizations (one with a haematemesis as a result of a Mallory–Weiss oesophageal tear, and two with non-specific abdominal pain). No further episodes of alcoholic ketoacidosis occurred, nor did the patient develop diabetes.

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have been mistakenly treated for diabetes with insulin for some time after the acidotic event (Hart and Frier, 1996).

The reason for hyperglycaemia in alcoholic ketoacidosis is uncertain. This patient had suffered previous attacks of alcoholic pancreatitis, and although his glucose tolerance test was not diagnostic of diabetes, the fasting plasma glucose was elevated, and the patient presumably did therefore have some degree of beta cell damage. Possibly this, combined with high secretion of counter-regulatory hormones during the episode of alcoholic ketoacidosis, led to the observed transient hyperglycaemia, as is similarly observed with the 'stress hyperglycaemia' associated with myocardial infarction (Husband et al, 1983).

Interestingly, of two other reported cases of alcoholic ketoacidosis with hyperglycaemia, one subsequently had a normal glucose tolerance test (Hart and Frier, 1996), but in the other, impaired glucose tolerance was demonstrated (Simpson and Cundy, 1992).

Alcoholic ketoacidosis with hyperglycaemia is rare, and recurrent attacks such as in our patient have not been previously reported. Fortunately, an appreciation of the possibility prevented improper treatment with insulin, which could of course be disastrous in an alcoholic subject without diabetes. HM

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