Unusual presentation of more common disease/injury

The perils of high carbohydrate drinks in the undiagnosed diabetic patient

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Diabetes mellitus is increasing in its incidence and prevalence. Reduction in refined carbohydrate (sugar) intake is an important part of nutritional advice to patients with known diabetes. Sugar is available in a variety of confectionary products. It is also available in especially packaged 'convenience foods' as high energy drinks. Among people without diabetes, such food can have its own health risks.

A significant group of patients with diabetes remain undiagnosed. This group are at especially high risk from all the negative metabolic effects of high sugar intake available as high calorie drinks.

The authors report two patients without previously known diabetes who presented similarly with marked hyperglycaemic states, leading to severe metabolic disturbances. Both were obese, had common precipitating factors and consumed large quantities of sugary soft drinks ('lucozade'). Both patients recovered well. They were followed up for over 7 years. One is not diabetic and the second is diabetic requiring a small dose of metformin.

BACKGROUND

Affluent societies are currently consuming large quantities of refined sugar including sugar laden soft drinks. This causes long-term health problems, however it can also cause avoidable acute complications that can be life threatening. Our patients survived despite severe hyperketotic state.

CASE PRESENTATION**Case 1**

A 26-year-old caucasian man was found collapsed at home by his father in a deleterious and confused state. He had been unwell the previous fortnight with 'flu-like' illness. Three days prior to admission he had extreme thirst and polyuria. He had been vomiting and getting increasingly confused. Medical history was unremarkable apart from delayed puberty.

On admission he was markedly obese (weight 120 kg, body mass index (BMI) 43), confused and severely dehydrated. He had a pulse of 120/min. Blood pressure was 95/50 mm Hg. Laboratory investigations: arterial pH of 6.87 and bicarbonate of 2.8 mmol/l, base excess -29.0, serum sodium of 108 mmol/l, potassium 1.8 mmol/l, urea 10 mmol/l, creatinine 180 µmol/l. Plasma glucose 89.8 mmol/l, phosphate 0.0 mmol/l. Urine test showed 4+ ketonuria. He was treated for diabetic ketoacidosis with large quantities of intravenous fluids and intravenous insulin infusion. In spite of intravenous infusion of potassium chloride of 340 mmols daily, his potassium dropped to 1.4 mmol/l. Within 24 h his respiration deteriorated and he became more acidotic with pH of 6.74 and started to retain CO₂. In spite of adequate hydration his renal function also deteriorated. He required immediate ventilation. Haemodialysis was commenced and continued for the next 3 weeks. After extubation he required further period of ventilation.

A CT scan of the brain showed multiple bilateral small cerebral infarcts. On the sixth week he was amnesic to recent events but was able to mobilise. By this time, his renal function had normalised as had his electrolytes. Soon after extubation he was noted that he was markedly insulin resistant requiring around 200 units of insulin daily. However insulin requirements reduced substantially prior to discharge.

He stayed in hospital for 7 weeks and by the time of discharge, he had lost 38 kg in weight. He was off insulin and achieved good glycaemic control on diet alone. On further questioning he admitted that 3 days prior to admission he was drinking an average of 9 litres of sweet fluids a day mainly 'lucozade' and 'coca-cola'. Two months following discharge he had impaired fasting glycaemia and a normal HbA_{1c}. His glucose tolerance test has been normal for on annual checking 6 years following discharge. However, he has developed behavioural problems, probably a consequence of cerebral infarction during his severe illness.

Case 2

A 31-year-old male caucasian computer engineer was found in a confused state by his mother. He had a recent viral illness with general tiredness and malaise. For 2 weeks he was surviving on large quantities of sugary drinks in the form of 'lucozade', fruit juices, milk shake and coffee. His main complaints were severe thirst and passing too much urine.

On admission he was noted to be markedly overweight (weight 115 kg, BMI 43), confused and severely dehydrated. Clinical examination was otherwise unremarkable. Laboratory investigations confirmed hyperosmolar non-ketotic diabetic state with measured plasma osmolality of 403 mosm/l with mild renal impairment (urea 17.5 mmol/l,

creatinine 181 mmol/l) and neutrophil leukocytosis. Plasma glucose 84.3 mmol/l. There was no ketonuria. He was treated with half normal saline, small doses of insulin and broad-spectrum antibiotics. Despite adequate hydration and good metabolic control, his condition deteriorated and he became aggressive. His serum sodium crept up to 170 mmol/l and he developed oliguria. He was ventilated and transferred to a dialysis unit. After 3 weeks of dialysis his renal function recovered. He was slightly confused but CT brain scan was unremarkable. Although at this stage he needed small doses of insulin, by the time of discharge he was on diet alone. He needed a few months of rehabilitation.

Three months following discharge his glucose tolerance test was only slightly impaired with normal HbA1c. One year later he went for repeat oral glucose tolerance test fasting, initial reading: 7.7 mmol/l; reading at 60 min: 15.8 mmol/l; reading at 120 min: 16.8 mmol/l. He is currently doing well on metformin alone and his HbA1c is 7.3%.

INVESTIGATIONS

Investigations in both patients confirmed severe hyperglycaemia with dehydration and renal failure. Severe ketosis was present in one of the patients (case 1)

DIFFERENTIAL DIAGNOSIS

- Diabetic ketoacidosis
- Non-osmolar diabetic state

TREATMENT

The two cases presented differently. The first patient's renal function normalised with fluids management. The second patient required dialysis before full recovery of renal function.

OUTCOME AND FOLLOW-UP

Both patients recovered well. They were followed up for over 7 years. One is not diabetic and the second is diabetic requiring a small dose of metformin.

DISCUSSION

Thirst is a common symptom of hyperglycaemia. Diabetes mellitus is the commonest cause of hyperglycaemia and is increasing in its incidence and prevalence globally.¹⁻⁴ The diabetic diet by reducing the amount of refined carbohydrates, reduces the likelihood of hyperglycaemia in the patient with known diabetes.

However, prior to the diagnosis and recognition of diabetes in the individual patient, the often raging thirst will lead the patient to drink large quantities of fluid. High energy/high carbohydrate drinks may be desirable for some people. In the patient with known diabetes these are hopefully avoided. However, in the patient with unrecognised diabetes mellitus, high carbohydrate drinks can have devastating consequences.

Unrecognised diabetes mellitus and prediabetes are both relatively common conditions with a prevalence of over

5% of men and 4% of women in England have diagnosed diabetes and 3.1% of men and 1.5% of women aged 35 and over have undiagnosed diabetes.⁵ The circumstances leading to the critical metabolic disturbances presented here can potentially affect the group of patients with unrecognised diabetes. Similar effects can be expected in patients with diabetes who choose to ignore sound dietetic advice and consume large amounts of high carbohydrate beverages.

McDonnell *et al*⁶ have reported hyperosmolality and hyponatraemia in five newly

diagnosed paediatric patients. As in the two cases presented here, their patients required intensive therapy. Prior to hospital admission, their patient's fluid intake had consisted of several litres of glucose high carbohydrate and 'isotonic' drinks. Their patients also required prolonged period of fluid replacement. Three of the five cases required haemofiltration.

As we have indicated, fluid replacement was a major challenge in both cases we have presented. As well as hyponatraemia and hyperosmolality, hypokalaemia were the major problems in our first case and required large amounts of intravenous potassium replacement.

We speculate that this potassium deficit might have been related to the type of fluids consumed prior to admission. In addition, profound metabolic acidosis would certainly have been a major contributor to this hypokalaemia. There are important differences between the cases described here and those previously described by McDonnell *et al*.⁷ Although one of our patients had significant ketonuria, they did not continue to be insulin dependent over the long term. Indeed our first case has had normal glucose tolerance for 6 years following the initial hospital admission. Diabetes has disappeared at least in one of our cases reported after losing significant amount of weight.

Presentation with ketoacidosis in a patient who did not have type 1 diabetes is atypical.

Ketosis can occur in patients with type 2 diabetes, although it is rare. Ketosis-prone type 2 diabetes mellitus has been reported. Typically, the patients are obese, middle-aged men with a strong family history of type 2 diabetes mellitus. The pathophysiologic mechanisms are unknown, but preliminary evidence suggests that these patients have a unique propensity to glucose desensitisation. Determination of autoimmune markers (islet-cell and glutamic acid decarboxylase antibodies) is useful in excluding patients with slow-onset type 1 diabetes or latent autoimmune diabetes. The presence of positive auto antibodies and measurement of basal or glucagon-stimulated C-peptide levels may be useful in predicting near-normoglycemic remission and long-term insulin dependence in obese patients with a history of diabetic ketoacidosis. The remission phase is usually less than 2 years when patients are treated with diet alone; however, low-dose sulfonylurea and metformin therapy may delay the recurrence of hyperglycaemia/clinical diabetes.

Learning points

- ▶ Ketosis-prone type 2 diabetes^{7 8} has been reported and can typically occur in the obese, middle-aged men with a strong family history of type 2 diabetes.
- ▶ Severe impairment of insulin secretion and action are found at presentation and aggressive diabetic management results in marked improvement in β -cell function and insulin sensitivity sufficient to allow discontinuation of insulin therapy within a few months.
- ▶ Consumption of high carbohydrate drinks can be dangerous in the patient with polydipsia with unknown or known diabetes mellitus. Avoidance of these should be incorporated into public awareness campaigns.
- ▶ Fluid management in such patients can be a major challenge. A detailed history to ascertain the amount and type of fluids consumed may be useful in deciding the type and amount of fluid replacement.

Competing interests None.

Patient consent Obtained.

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