CASE REPORT

Diabetes unmasked by electric shock

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Following an accidental electric shock, a boy with no previous symptoms developed hyperglycaemia, rapidly evolving into diabetes. He was aglycosuric for 24 hours after the shock. Islet cell antibodies were shown shortly after the accident. Although destined to develop diabetes, it seems likely that the electric shock unmasked impaired glucose tolerance. Glucose homeostasis should be monitored in children following significant electric shocks.

In acute illnesses and injury of the central nervous system, raised blood glucose (BG) concentrations may occur and can be important prognostically.^{1,2} Similarly, electric shocks to the brain alter the neuroendocrine axis, resulting in hyperglycaemia.³ We have found no previous reports of electric shocks precipitating diabetes in children.

CASE REPORT

A 13 year old boy scout who was mowing his neighbour's lawn, severed the lawn mower's electric cable, causing an electric shock to throw him to the ground five feet away. He sustained an electric burn to his hand. He was healthy before the accident with no significant medical or family history. He was slim and in early puberty.

No cardiac arrhythmia was recorded. Urinalysis for myoglobin was negative, as were glucose tests on two occasions the day following admission. Thirty six hours after admission, evening urinalysis showed notable glycosuria. Capillary BG was 20 mmol/l. The following morning glycosuria was absent and fasting laboratory BG was 7.6 mmol/l. The evening BG again rose to 16.4 mmol/l. Glycosylated haemoglobin (HbA_{1c}) was 7.3% (normal range 4–8.5%).

He was discharged home with instructions to monitor urine and capillary BG. Initially he remained asymptomatic but recorded intermittent glycosuria without ketonuria. Capillary BG concentrations ranged between 6 and 11 mmol/l. Over succeeding weeks he often had no glycosuria in the morning, but intermittent glucose excretion during the day with variable BG concentrations. Six weeks after the original insult he developed thirst and polyuria. After four months he lost weight, experienced nocturia, and postprandial BG concentrations were raised. The HbA_{1c} concentration rose to 8.9% and then 9.7%. He was commenced on insulin.

A retrospective analysis of serum taken 48 hours after the electric shock showed pancreatic islet cell antibodies (ICA); when remeasured one and six weeks later, the ICA titre was 80 JDFU. Six weeks after presentation a mid morning serum insulin was 13.3 mU/l accompanied by a C peptide concentration of 1.3 nmol/l (normal range 0.14–1.39). Serum amylase and thyroid function were normal.

Two months after starting insulin (0.3 units/kg/day) he had regained 8 kg weight. Insulin dose was reduced, but subsequently increased to concentrations consistent with total insulin dependency. HbA_{1c} returned to normal.

DISCUSSION

Can type 1 diabetes be precipitated by electric shock? This boy did not have diabetes at the time of the shock and he was aglycosuric in the initial 24 hour monitoring period. There was a rapid progression of glycaemic deterioration in association (in retrospect) with raised islet autoantibody markers, but symptoms consistent with diabetes only developed later.

High titres of complement fixing ICA (markers of β cell destruction) in the general population or in siblings of patients with diabetes have strong predictive value for disease development.^{4,5} An ICA titre of 80 JDFU has a predictive value of 100% within 10 years in first degree relatives of patients with type 1 diabetes.⁶

The presence of high titres of ICA presumably predated the electric shock, suggesting that he was predestined to develop diabetes, although the timing and predictability were uncertain. Almost certainly the electric shock precipitated hyperglycaemia and glycosuria.

Interestingly, Claude Bernard, in 1855, showed that transfixing the brain medulla with a metal probe caused acute hyperglycaemia and glycosuria in rats, so called "picure" diabetes.⁷ Recently it has been postulated that thrombotic strokes in specific areas of the brain have metabolic consequences, leading to diabetes mellitus.⁸

Electric shock can disturb hypothalamic neuroendocrine control of glucose homeostasis, acting via autonomic outflow to the viscera.³ These complex mechanisms are responsible for acute hyperglycaemia in response to certain central stimuli, seen in acutely ill children with central nervous system (CNS) infections, seizures, and head trauma.² The effect of neurogenic influences on the development of diabetes is further illustrated by the occurrence of diabetes in a number of predominantly neurological genetic disorders,⁹ and the association of depression with insulin resistance and impaired glucose homeostasis.¹⁰

Little is known about the effect of accidental electric shock on blood glucose control. There is controversy about the effects of electroconvulsive therapy (ECT) on glucose homeostasis and diabetes.¹¹ When ECT is performed on individuals without diabetes, transient hyperglycaemia may occur¹²; it causes minimal alterations in type 2 diabetes, but in patients with pre-existing type 1 diabetes, it may provoke dangerous hyperglycaemia.¹¹

Hyperglycaemia following ECT has been reported in an adult without known pre-existing diabetes, but with known risk factors, who became insulin dependent.¹³ That case may represent the unmasking of disease as in our case.

In conclusion, it may be more than coincidence that this young boy developed type 1 diabetes after an electric shock. Electric shocks to the CNS are known to impair glucose homeostasis, and in a predisposed person it may be sufficient to precipitate diabetes. We think it prudent, therefore, in all

Abbreviations: BG, blood glucose; CNS, central nervous system; ECT, electroconvulsive therapy; ICA, islet cell antibodies

patients who have had a significant electric shock to monitor urinary glucose at each voiding for 48 hours, and blood glucose before and after morning and evening meals if glycosuria occurs.

Whether the risk of electric shock applies only to those already at risk of diabetes (for example, with circulating antibodies or a positive family history) remains to be seen.

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ECHO

Flying on a wing and a prayer

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Spirometry predicts hypoxia during flights more accurately than hypoxic challenge. So Buchdahl *et al* found when they tested their initial premise—that in children with cystic fibrosis (CF) and chronic lung disease fingertip pulse oximetry beforehand could predict O₂ desaturation in flight—by direct measurement during flights.

In a five year study of children with CF flying long haul on organised holidays, 10 of the 82 children developed desaturation <90% during the outward or return flight, two of whom needed in flight O₂. Pulse oximetry one month before flying correctly predicted only two of the 10, spirometry predicted seven. Sensitivity for pulse oximetry was 20% and specificity 98% compared with 70% and 96% respectively for spirometry. Spirometry was the better predictor in this selected group, but not by much, the authors say.

Reliable measures for assessing likelihood of hypoxia are long overdue. Commenting in an editorial, Webb, from the Adult Cystic Fibrosis Unit in Manchester, UK, points to exclusion of the most severely affected children and probable attention to the children's medical fitness during their holiday as drawbacks to this latest study. He cites evidence, in adult patients, that resting O₂ saturation or FEV₁ did not correctly predict hypoxia during simulated flight conditions. The Manchester unit recommends in flight O₂ for patients whose PaO₂ drops below 6.6kPa during a simulated flight test.

Many other risk factors in CF complicate the picture. Emphasising the need for careful planning and preparation, Webb lists the minimum essentials for such patients planning a holiday abroad.

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