CASE REPORT

DIABETIC KETOACIDOSIS AFTER CHANGING INSULIN PENS

Diabetic ketoacidosis is a medical emergency, characterised by hyperglycaemia, hyperketonaemia and metabolic acidosis. It is commonly precipitated by infection and inadequate doses of insulin caused by underdosing with insulin during intercurrent illness, non-compliance or poor injection technique. A recent paper in the *British Medical Journal* reports on 2 cases where recurrent diabetic ketoacidosis was caused by unrecognised difficulty when switching from one prefilled insulin injection device to another.

In the first case, a 68-year-old man with type 1 diabetes was admitted with critically ischaemic feet. He had an emergency right, below-the-knee amputation, but the left leg was deemed salvageable. Postoperatively he was stable and restarted injecting insulin twice daily with subcutaneous NovoMix 30 insulin using a FlexPen. He subsequently developed fever, vomiting and increased finger-stick glucose readings. No sepsis was found. His venous plasma glucose concentration was 20.9 mg/l and urinary ketones were present. He had a metabolic acidosis, and diabetic ketoacidosis was diagnosed. He was treated with intravenous insulin, fluid and antibiotics and the condition was resolved.

The left foot remained critically ischaemic and he underwent a left, below-the-knee amputation and was metabolically stable postoperatively. Again, when he restarted subcutaneous insulin his blood glucose rose to 24.7 mg/l, he showed urinary ketones and was profoundly acidotic. Once again, he had diabetic ketoacidosis.

Consultation with the diabetes team picked up the problem. He had been changed from a NovoPen 3 to a FlexPen. When his injecting technique was examined, he was rewinding the dial rather than depressing the plunger and so no insulin was delivered. Once he knew how to use the new pen he became stable again.

In the second case, a 17-year-old boy who had type 1 diabetes for 5 years was admitted with ketoacidosis and hyperglycaemia. He had had 3 episodes of diabetic ketoacidosis in the previous month and on each occasion had responded to intravenous insulin and fluids. He was injecting NovoRapid insulin via a FlexPen with meals and Levemir insulin by NovoPen 3 twice daily. When his injection technique was examined during his latest admission, he was seen to be trying to inject insulin by 'reverse dialling' with both pen devices. This delivered insulin through the NovoPen 3, but not through the FlexPen. Once he knew how to use the devices the incidents of ketoacidosis stopped.

These 2 cases highlight simple differences between insulin delivery devices that may lead patients to fail to inject their insulin.

Bhardwaj V, *et al. BMJ* 2006; **332:** 1259-1260.

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SINGLE SUTURE

EATING LESS AND LIVING LONGER

We know that eating very little can extend lifespan. But why? Now Andrzej Bartke and colleagues from Southern Illinois University think that growth hormone could be the key. The team worked with normal mice and mutant mice missing the receptor for growth hormone. Half of each type were allowed to eat as much as they liked and the other half were fed 30% fewer calories than usual. Normal mice on fewer calories lived 20 - 30% longer. However, mice without growth hormone receptor also showed similar increases in longevity on a normal diet. So it seems as though knocking out the growth hormone receptor is similar to restricting calories. But doing both doesn't make the mice live even longer. The mutant mice on the low-calorie diet had similar lifespans to the normal mice on the normal diet. It may be that insulin is the connection – both groups of long-lived mice had greater insulin sensitivity, and caloric restriction in mutant mice did not increase their insulin sensitivity still further.

Bartke A, *et al. Proc Natl Acad Sci* 2006; **103:** 7901.