Hypoglycaemia

Severe relapsing sulphonylurea-induced hypoglycaemia: a diagnostic and therapeutic challenge

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Answers on p 123.

A 62 year old woman was admitted with acute confusion and reduced mobility. She had recently been a surgical inpatient with pseudo-obstruction of the bowel that had been managed conservatively. Since discharge she had experienced several episodes of slurred speech and perioral tingling lasting two to three hours. A presumptive diagnosis of recurrent transient cerebral ischaemic episodes was made. The patient had a five year history of type 2 diabetes mellitus treated with gliclazide 40 mg twice a day. She had impaired renal function (serum creatinine raised at 176 $\mu\text{mol/l}$) and a glycated haemoglobin of 6.5% (non-diabetic range 3.5%–6.5%).

Two hours after admission her level of consciousness decreased and she became

unresponsive to physical stimuli. No focal neurology was evident, but her capillary plasma glucose concentration was 1.8 mmol/l (subsequently confirmed by a simultaneous venous fluoride oxalate plasma sample). The hypoglycaemia was treated with an intravenous bolus of 50 ml of 50% glucose, which promptly restored consciousness. A continuous intravenous infusion of 5% glucose was started and her blood glucose concentration was measured two hourly throughout the night. The gliclazide was discontinued.

The following morning she became confused and lethargic with plasma glucose of 1.8 mmol/l. She was given a further 50 ml bolus of 50% glucose intravenously with prompt clinical effect. Over the next three days she had five further

symptomatic hypoglycaemic episodes of sufficient severity to warrant acute treatment with boluses of 50% glucose (fig 1). Sixty hours after admission she was successfully weaned off the glucose infusion. Her blood glucose was observed for a further 24 hours and once stabilised she was discharged from hospital. Three months after discharge there was no recurrence of symptoms.

Initially the cause of this patient's hypoglycaemia was unclear because she had access to a relative's insulin in addition to her sulphonylurea therapy. A tablet inspection of her medication on discharge from the surgical ward ruled out a dispensing error. A plasma sample taken 12 hours after admission during an episode of hypoglycaemia confirmed a raised serum insulin concentration of 190 pmol/l (insulin concentrations in the presence of hypoglycaemia normally being suppressed to <10 pmol/l). A raised C-peptide concentration (3974 pmol/l) confirmed that this was due to increased pancreatic insulin secretion. Gliclazide concentrations were within the therapeutic range.

QUESTIONS

- (1) Describe the factors that increase the risks of sulphonylurea hypoglycaemia.
- (2) What was the reason for this patient's prolonged hypoglycaemia?
- (3) What pharmacological agents are available to treat hypoglycaemia in this situation?

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Figure 1 The blood glucose profile over the patient's admission; ↓ denotes where boluses of hypertonic intravenous glucose were given.

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