Long-term symptomatic relief of postprandial hypoglycaemia following gastric surgery with a somatostatin analogue

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Summary: Somatostatin has been shown to be effective in the management of the dumping syndrome and there have been reports of the effective use of long acting somatostatin analogue in the management of this condition. However, there have been few reports of the prolonged use of a somatostatin analogue in the late dumping syndrome. We describe a patient in whom this management provided good long term symptomatic relief which was confirmed biochemically.

Introduction

Postprandial reactive hypoglycaemia following gastric surgery is fortunately now rare but can be quite disabling to those affected. Conventional treatment by frequent small meals with increased fibre content and the addition of substances such as guar and pectin is generally unsuccessful. Previous reports of somatostatin infusions and subcutaneous administration of a long acting somatostatin analogue over short periods of time have demonstrated the effectiveness of this hormone in the management of this condition.1–4 Our patient was treated with twice-daily subcutaneous injections of Sandostatin, a long acting somatostatin analogue5,6 for a period of 3 weeks and monitored as an out-patient. He derived good symptomatic and biochemical relief.

Case report

A 50 year old West Indian post-office worker, who had undergone a vagotomy and gastroenterostomy 15 years previously for chronic duodenal ulceration, was referred with a history of intermittent episodes of tremor and faintness occurring between 90 and 120 minutes following meals. These episodes, which were rapidly relieved by glucose, had become so frequent as to prevent him from working. Diarrhoea was not associated with the episodes and the patient had never lost consciousness. His symptoms had failed to respond to treatment with small frequent meals with a high fibre diet, guar gum granules and a reduced carbohydrate intake with each meal.

An extended oral glucose tolerance test showed a lag type storage curve with reactive hypoglycaemia; the lowest blood glucose recorded at 120 minutes was 1.9 mmol/l (Figure 1). A 72 hour fast followed by exercise failed to reproduce hypoglycaemia. He was then reassessed using a mixed test breakfast containing 75 g of carbohydrate after an overnight fast. On the first occasion he was given a subcutaneous injection of normal saline before the breakfast and the blood glucose fell to 2.4 mmol/l at 150 minutes. On a subsequent morning he was given 50 µg Sandostatin subcutaneously at the beginning of the meal. The minimum blood glucose of 4.7 mmol/l occurred at 210 minutes and there was complete abolition of symptoms with resolution of the reactive hypoglycaemia. Fasting plasma insulin levels were comparable on the two test days, (normal saline: 11.7 mU/l, Sandostatin: 12.2 mU/l). The 60 minute value, however, was completely suppressed after Sandostatin (normal saline: > 500 mU/l, Sandostatin: 12.3 mU/l).

He was taught to self monitor blood glucose using BM-glycaemic strips and to administer the subcutaneous injections himself. He was discharged on Sandostatin 50 µg twice daily with his breakfast and evening meal. He was reassessed at weekly intervals and after 2 weeks the Sandostatin was administered 30 minutes after the test meal. This led to a smoother control of postprandial glucose (Figure 1). The patient’s self-monitored glucose were normal whilst taking twice daily Sandostatin, but showed values of 2.0 mmol/l or less on discontinuing the treatment. There was an immediate return of reactive hypoglycaemic symptoms on cessation of Sandostatin.

The 24-hour faecal fat excretion while on the Sandostatin was 45 mmol/day and 2 weeks after discontinuing the injections the 24-hour faecal fat remained elevated at 45 mmol/day (normal < 18 mmol/day).

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Discussion

The main adverse effects of Sandostatin experienced by our patient included local irritation at the injection site, which was relieved by allowing the Sandostatin solution to warm to room temperature prior to injection, and a moderately painful burning sensation on defaecation associated with the passage of bulky pale stools. The latter sensation improved with time. The modestly elevated faecal fat excretion while on the Sandostatin remained unaltered on cessation of the injections. This suggests that there was a degree of malabsorption secondary to the original surgery; this is a common and well recognized phenomenon. Previous long term studies with Sandostatin in other conditions have failed to show serious problems with malabsorption due to steatorrhoea.

There are few previous reports of the prolonged use of a long acting somatostatin analogue in the treatment of postprandial reactive hypoglycaemia following gastric surgery. Long et al. reported the use of an infusion of somatostatin in the treatment of the dumping syndrome and showed a clear decrease in symptoms. Longer term use of this preparation however is impractical.

Reports of the use of somatostatin analogues have been very successful but have been limited to inpatient use. Our patient experienced both symptomatic and biochemical benefit from the long acting analogue whilst being closely monitored over several weeks as an out-patient.

We suggest that the administration of a long acting somatostatin analogue immediately after a meal may offer a practical and effective approach to the treatment of the late dumping syndrome which has failed to respond to other measures. The patients may be managed as out-patients and their progress assessed by self-monitoring with BM glycaemic strips. There is an obvious need for further studies with a larger number of patients but the results from our patient are most encouraging.

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References

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