Use of the alpha-glucosidase inhibitor acarbose in patients with ‘Middleton syndrome’: Normal gastric anatomy but with accelerated gastric emptying causing postprandial reactive hypoglycemia and diarrhea

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Postprandial reactive hypoglycemia, early satiety and diarrhea are well-recognized side effects following full or partial gastrectomy or gastric bypass. It has only recently been realized, however, that patients with normal gastric anatomy may experience similar symptoms and signs due to primary accelerated gastric emptying (Middleton syndrome). In previous case studies, patients responded well to the use of dietary modification (frequent small-volume meals) alone. The authors describe two patients with this syndrome who continued to experience symptoms of reactive postprandial hypoglycemia despite dietary intervention but became asymptomatic following the addition of the alpha-glucosidase inhibitor acarbose.

Key Words: Diarrhea; Dumping syndrome; Gastric emptying; Gastric motility; Hypoglycemia

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biopsies, a short synacthen test and a SeHCAT retention study were normal. Fasting gut hormone levels, glucose hydrogen breath test, video capsule endoscopy, abdominal computed tomography scan and fecal elastase were also normal. A scintigraphic solid-phase gastric emptying (5) study revealed accelerated gastric emptying with a half emptying time of 26 min. An extended glucose tolerance test with a standard 75 g glucose load demonstrated an appropriate rise in serum glucose and return to baseline in 30 min but a subsequent fall to 2.3 mmol/L at 150 min, at which time her hypoglycemic symptoms occurred (Figure 1).

A diagnosis of primary accelerated gastric emptying was made and she was advised to adhere to a ‘grazing diet’. This dietary approach settled her symptoms of diarrhea but did not resolve the hypoglycemic symptoms. She was, therefore, given acarbose 50 mg before her main meals two or three times per day, which resulted in a near complete resolution of symptoms, with only mild fatigue occurring after a large meal.

**DISCUSSION**

Motility disorders of the gastrointestinal tract are not easily identified by their associated symptoms (6), which can consequently lead to delays in diagnosis. A study involving 649 consecutive patients who underwent scintigraphic gastric emptying studies (7) reported that in patients with suspected accelerated gastric emptying on clinical grounds, this was confirmed in 69%, but 7% demonstrated delayed gastric emptying. Of patients suspected to have gastroparesis, only 29% were found to demonstrate delayed gastric emptying on scintigraphic testing and 23% had accelerated gastric emptying. This study implies that the clinical diagnosis of gastric dysmotility is difficult because symptoms do not reliably reflect the underlying nature of the motility disorder, and symptoms believed to indicate gastroparesis may be caused by accelerated gastric emptying.

Early satiety, postprandial diarrhea and subsequent hypoglycemic symptoms, such as nausea, fatigue, sweating and cognitive impairment, should lead the clinician to consider the diagnosis of ‘Middleton syndrome’, in which the primary abnormality is accelerated gastric emptying. Symptoms usually respond well to dietary intervention but, as described in the present report, may occasionally require additional treatment with an alpha-glucosidase inhibitor, such as acarbose, to resolve refractory hypoglycemic symptoms.

**REFERENCES**
