Superior Mesenteric Artery Syndrome in Type 1 Diabetes Masquerading as Gastroparesis

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Objective: We describe and discuss a case of superior mesenteric artery syndrome presenting with gastrointestinal signs and symptoms mistakenly attributed to, and treated as, diabetic gastroparesis.

Research Design & Methods: Case report describing the clinical presentation, including history and physical examination, evaluation, diagnosis and treatment of a patient with type 1 diabetes presenting with gastrointestinal complications.

Results: Clinical suspicion combined with the appropriate radiological evaluation led to a diagnosis of superior mesenteric artery syndrome, with classic findings of reduced aortomesenteric angle and distance. Surgical intervention resulted in resolution of symptoms and recovery of glycemic control.

Conclusions: The possibility of superior mesenteric artery syndrome should be considered in patients with type 1 diabetes presenting with gastrointestinal symptomatology, especially when associated with weight loss.
Patients with type 1 diabetes mellitus experience greater frequency of gastrointestinal complications, such as diabetic enteropathy, manifested by diarrhea, steatorrhea and/or constipation (1), and gastroparesis, which commonly presents as bloating, but may also result in early satiety, epigastric pain or discomfort, nausea, vomiting, postprandial fullness and anorexia. The clinician must be vigilant since these symptoms may be due to less common etiologies such as that reported in this case: superior mesenteric artery syndrome (SMAS). This is only the second reported patient with type 1 diabetes complicated by superior mesenteric artery syndrome (2).

HISTORY & EXAMINATION

An 18-year-old white female with a history of type 1 diabetes since age 6, presented with a 3-year history of poorly controlled diabetes, increasing insulin requirements (up to 3 units/kg/day) and frequent hospitalizations for diabetic ketoacidosis (DKA). The patient’s diabetes had been under excellent control (A1C 6.9%) until she began a low carbohydrate diet. The ensuing 50-pound (22.7 kilogram) weight loss was exacerbated by personal stressors, and the development of gastrointestinal symptoms. She reported nausea, which after dinner progressed to retching and vomiting along with the onset of abdominal pain, tachycardia and the sudden development of severe nighttime hyperglycemia (capillary blood glucose levels often in the 400-600 mg/dl (22.2-33.3 mmol/l) range) with ketonuria, poorly responsive to large doses of insulin (60-80 units of insulin delivered overnight). Associated symptoms included depression, painful sensory peripheral neuropathy, arthralgias, constipation, hair loss, dry skin and amenorrhea.

On exam she had orthostatic changes with a baseline heart rate of 116/min and a blood pressure of 133/92 mmHg. Weight and BMI were at 123 lbs and 17.7 kg/m², respectively. No findings of neuropathy or retinopathy were evident. Glycosylated hemoglobin (HbA1c) was 11.5%. Additional evaluation included a urine free cortisol, DHEA, transglutaminase antibodies, serum IGF-1 and glucagon levels, a complete blood count, and fecal fat, all within normal levels. Urine albumin levels were slightly elevated on repeated evaluations.

A 6-week trial of domperidone and oral dietary supplementation along with an intensive insulin regimen failed to improve the patient’s condition, with only a marginal improvement in A1C to 10.5%.

INVESTIGATION

The patient was further assessed for the possibility of anorexia nervosa and insulin bulimia (avoidance of prescribed insulin administration). She had previously undergone extensive gastrointestinal evaluation (upper GI series & endoscopy), which had only revealed delayed transit time on gastric emptying scintigraphy. Because of the suspicious presentation and negative work-up, a computed tomography angiography (CTA) was ordered which demonstrated dilatation of the first and second portions of the duodenum with normal gastric volume. Compression of the third portion of the duodenum was evident and a reduced aortomesenteric angle of 15° (normal: > 22°) and an aortomesenteric distance of 3.3 mm (normal: > 10 mm) were identified (Figure 1). These findings were consistent with SMAS. Upper gastrointestinal series and upper endoscopy were unremarkable.

An exploratory laparotomy, duodenojejunostomy and division of the ligament of Treitz were subsequently performed to relieve the mechanical
obstruction. The patient’s postoperative course was uneventful: her symptoms resolved and her HbA1c was 8.4 % at the 1-month postoperative visit and 7.0% six months later, concomitant with a 10 pound (4.5 kilogram) weight gain.

CONCLUSIONS
Patients with poorly controlled diabetes may present with symptoms of bloating, early satiety, epigastric pain or discomfort, nausea, vomiting, postprandial fullness and anorexia, often attributed to gastroparesis. We report a case of SMAS in a patient with type 1 diabetes masking as a diagnosis of gastroparesis. This case is interesting in that the most severe manifestations of SMAS occurred after the evening meal. They were exacerbated by the patient lying supine, and were associated with ketogenesis and a significant degree of resistance to insulin therapy.

SMAS is a term used to define the clinical signs and symptoms caused by compression of the third portion of the duodenum between the angle made by the aorta and the superior mesenteric artery (SMA). The compression is caused by a decrease in the aortomesenteric angle and distance, with complete obstruction observed with angles between 6-16° and a distance of 2-8 mm (3-6). SMAS is thought to occur because of duodenal compression following the loss of the mesenteric fat pad, which maintains the angle and distance between aorta and SMA. This can be seen in acute catabolic states, dietary disorders such as anorexia nervosa, chronic wasting diseases, as well as rapid weight loss following bariatric surgery.

The importance of reporting this case lies in heightening the diagnostic index of suspicion for SMAS in patients with type 1 diabetes presenting with suspicious gastrointestinal symptoms and glycemic instability. While often attributed to diabetic gastroparesis, patients manifesting intermittent and recurrent postprandial abdominal pain, nausea, vomiting and anorexia associated with weight loss should be screened for SMAS.

A variety of modalities have been utilized for diagnosis (7, 8); most have been largely replaced by the noninvasive and more accurate helical computed tomography with oral and intravenous contrast and 3-D reconstruction (9).

In conclusion, patients with type 1 diabetes presenting with gastrointestinal symptoms associated with recent weight loss, or low BMI, should be evaluated for the possibility of SMAS. CTA is a quick and reliable diagnostic tool to identify the structural and anatomic abnormalities associated with this syndrome.
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Figure 1: (A) Sagittal CTA demonstrating a narrow (15°) aortomesenteric angle (normal: >22°); (B) Axial CTA demonstrating a reduced aortomesenteric distance of 3.3 mm causing duodenal compression and dilatation of the proximal portion of the duodenum. IVC, inferior vena cava; LRV, left renal vein; MCA, middle colic artery; SMA, superior mesenteric artery.