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Case Report

Intermittent superior mesenteric artery syndrome in a patient with multiple sclerosis

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ABSTRACT

A 42-year-old man with multiple sclerosis presented with recurrent vomiting, in the context of recent weight loss. Computed tomography scan of the abdomen and pelvis revealed duodenal compression by the superior mesenteric artery (SMA), consistent with intermittent SMA syndrome. Subsequent gastroscopy and barium meal follow-through showed resolution of the obstruction. SMA syndrome is rare and has not previously been reported in a patient with multiple sclerosis. We hypothesize that loss of the aortomesenteric fat pad on the background of contorted body habitus from multiple sclerosis placed the patient at risk for intermittent positional compression of his duodenum.

Introduction

Superior mesenteric artery (SMA) syndrome is a rare but serious cause of proximal small bowel obstruction, whereby the third part of the duodenum (D3) is compressed in the acute angle between the SMA and the aorta. It remains challenging to identify on solely clinical grounds, and radiological investigations are the basis of establishing a definitive diagnosis. This patient's presentation was made more atypical by the presence of multiple sclerosis (MS) and lack of risk factors. This unusual case adds to the sparse literature on SMA syndrome and its heterogeneous presentation.

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Case presentation

A 42-year-old male presented with 7 days of vomiting and nil flatus. He had not opened his bowels for 9 days. He denied pain, recent weight loss, urinary symptoms, systemic symptoms, sick contacts nor recent travel. He reported a similar episode 2 months prior with spontaneous resolution.

Past history consisted of non-ST elevation myocardial infarction, depression, and secondary progressive MS, leaving the patient wheelchair bound. One month prior, the patient had suffered a left supracondylar femoral fracture following a fall from sitting height, which had been repaired with open reduction and internal fixation. He had undergone postoperative rehabilitation and regained his premorbid level of function. His regular oral medications were daily amitriptyline 200 mg, nocte mirtazapine 45 mg, as well as twice daily (BD) carbamazepine 400 mg and docusate/senna 50/8 mg. The patient daily consumed 20 cigarettes, 2 standard drinks of alcohol, and 5 grams of marijuana.

On examination, the patient was well, overweight, and displayed chronic abnormal spinal posturing due to MS. Vital signs revealed sinus tachycardia of 105 beats per minute, blood pressure 140/74 mm Hg, temperature 37.8 °C, and oxygen saturation of 99% on room air. Chest auscultation revealed that left basal decreased air entry and crepitations. The abdomen held no scars and was moderately distended with generalized mild tenderness. There were no other findings. The patient had a long-term urethral catheter, draining clear yellow urine. A nasogastric tube (NGT) was inserted, immediately draining 3500 mL of bilious fluid.

Initial serum investigations revealed hemoglobin of 142 g/L, white cell count 20.4 $\times$ 10^9/L, platelets 365 $\times$ 10^9/L, potassium 3.3 mmol/L, estimated glomerular filtration rate >90 mL/min/1.73 m^2, and albumin 31 g/L. C-reactive-protein was 76 mg/L. Other electrolytes, liver function tests, and Troponin T were normal. Urine dipstick was positive for protein, leukocytes, and blood and culture revealed chronic colonization with *Pseudomonas aeruginosa*. Chest x-ray displayed left basal consolidation.

The impression was of proximal small bowel obstruction complicated by aspiration pneumonia. Postulated mechanisms included internal hernia or malignancy. The leading differential diagnosis was gastric outlet obstruction.

A contrast computed tomography (CT) scan of the abdomen and pelvis revealed gastro-duodenal distension with a transition point at D3 due to compression between the SMA and aorta. The aortomesenteric angle and distance were 18° and 8 mm, respectively (Fig. 1). Left lower lobe pneumonia was seen.

The patient was fasted and treated conservatively, with intravenous (IV) resuscitation and NGT on free drainage. He was prescribed IV pantoprazole 40 mg, ceftriaxone 1 g, and azithromycin 500 mg daily and metronidazole 500 mg BD, as well as subcutaneous 40 mg enoxaparin daily. All oral medications were ceased, with IV levetiracetam, 500 mg BD substituted for his usual carbamazepine.

On the third day of admission, following removal of the NGT, the patient underwent diagnostic gastroscopy to D3. Mild distal reflux esophagitis was seen, as well as multiple non-bleeding superficial clean-based gastric ulcers in the gastric fundus, body, and antrum. Biopsies of these revealed only inflammatory change, with no evidence of dysplasia nor *Helicobacter pylori*. In the duodenum, the mucosa was normal and the lumen widely patent, with no evidence of malignancy, ulceration, duodenal web nor extrinsic compression (Fig. 2). Water was flushed into D3 with easy passage downstream. The NGT was resited at the procedure's conclusion.

Barium meal with plain film follow was subsequently performed. While D2 remained dilated and D3 narrow, there was prompt passage of contrast to the small bowel narrow with no evidence of obstruction (Fig. 3).

The clinical impression was of intermittent SMA syndrome. The patient's vomiting resolved on day 3. On day 4, the patient tolerated a clear fluid diet and passed flatus, so the NGT was removed, and the patient placed on normal diet thereafter, without complaint. His bowels eventually reopened on day 7.

His pneumonia improved and antibiotics were converted to oral amoxicillin/clavulanic acid 875/125 mg BD, which were continued for 5 days. IV levetiracetam was ceased and the patient restarted on oral carbamazepine. Pantoprazole was converted to oral formulation and continued post discharge. All other usual oral medications were restarted.

The patient was discharged on day 8. Surveillance gastroscopy at 3 months confirmed resolution of his gastric ulcers. At 4-month follow-up, he had suffered no further episodes.

Discussion

SMA syndrome is a rare disorder in which D3 is compressed between the aorta and SMA. This can result in partial or complete duodenal obstruction that may present acutely, chronically, or intermittently. Variously known as Wilkie's syndrome, aortomesenteric syndrome, mesenteric root syndrome, cast syndrome, or chronic duodenal ileus, it was first described by Rokitansky in 1861 based on autopsy findings [1], with the first case series published by Wilkie in 1927 [2]. It may occur simultaneously with or distinct from Nutcracker syndrome, in which the left renal vein is compressed between the aorta and SMA [3]. Due to the syndrome's rarity, there is a paucity of high-quality data. Since 1950, the largest case series comprises 27 patients [4].

The syndrome's pathophysiology involves a decreased aortomesenteric angle and narrowed aortomesenteric distance. Typical patients have pre-existing low values due to inadequacy of the aortomesenteric fat pad that usually supports the area, often with body mass index less than 18 kg/m^2 or weight percentile for height of <5% [3]. They may also have an abnormally high ligament of Treitz with corresponding upward fixation of the D3 into the aortomesenteric angle, or a low SMA origin [2]. A precipitating event further collapses the space occupied by D3. This may be due to weight loss, rapid height increase, aneurysm, or trauma. Significant weight loss shrinks the supportive fat pad and may be intentional or due to eating disorders, malignancy, malabsorption, or following bariatric surgery [2]. Height increase may occur due