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Successful nutritional therapy for superior mesenteric artery syndrome

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ABSTRACT Superior mesenteric artery (SMA) syndrome is an uncommon cause of duodenal outlet obstruction. Symptoms and signs suggestive of this condition are nonspecific, and a high index of suspicion coupled with appropriate imaging studies are necessary for diagnosis. We present the case of a 70-year-old man who developed SMA syndrome following prolonged hospitalisation for a surgically treated bleeding duodenal ulcer. His SMA syndrome resolved after successful nonoperative management based on accepted guidelines for nutritional therapy, thus avoiding the need for reoperation and its attendant risks in a malnourished patient.

Keywords: duodenal outlet obstruction, duodenal ulcer, nutritional therapy, SMA syndrome, Wilkie's syndrome
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INTRODUCTION

First described by Rokitansky in 1861, superior mesenteric artery (SMA) syndrome, also termed cast syndrome or Wilkie's syndrome,[1] is a rare condition where the third part of the duodenum (D3) is obstructed due to compression by the SMA, leading to duodenal outlet obstruction. It manifests with nonspecific symptoms of nausea, vomiting, anorexia and epigastric pain, and if left untreated, can result in significant electrolyte imbalances and other morbidities associated with malnutrition. The incidence of SMA syndrome has been found to lie between 0.013% and 0.3% of the general population.[2] We describe the treatment and outcome of an elderly man who developed SMA syndrome following operative intervention for a bleeding peptic ulcer.

CASE REPORT

A 70-year-old Chinese man, with a past medical history of well-controlled schizophrenia and mild chronic obstructive pulmonary disease, presented to the emergency department with symptoms of nausea, haematemesis and melaena. He had a significant surgical history – a patch repair of a perforated duodenal ulcer about ten years ago. He also has a history of heavy smoking and social drinking. He was not on any anti-inflammatory, antiplatelet or anticoagulant medication.

Although the patient was not tachycardic, his systolic blood pressure was noted to be 90 mmHg. Following initial resuscitative measures, which included 2 L of crystalloids and two pints of packed cell transfusions, his haemodynamic parameters improved. He then underwent an oesophagogastroduodenoscopy (OGD) under monitored sedation. A large ulcer with a dark necrotic base, consistent with a Forrest IIc ulcer, was noted on the posterior aspect of the first part of the duodenum. No active bleeding was noted at the time and no endoscopic intervention was performed.

The patient was closely monitored and had recurrence of haematemesis 12 hours later. A repeat OGD was abandoned due to poor visualisation, and an emergency laparotomy was performed. Dense adhesions between the inferior surface of the liver, pylorus and duodenum were encountered due to the patient's previous patch repair, and mobilisation of the adhesions resulted in a significant amount of oozing from the liver bed. Oversewing of the bleeding ulcer with pyloroplasty was performed. At this point, the patient was cold and coagulopathic, with evidence of non-ST-segment elevation myocardial infarction. Surgery was converted to a damage control operation. The liver bed was packed with sterile dressings and the abdomen was temporarily closed with a modified vacuum-assisted closure system. He was resuscitated in the intensive care unit, and after normalisation of haemodynamic, cardiac and haematological parameters 48 hours later, the patient underwent truncal vagotomy and definitive primary closure of the abdomen in the operating theatre.

Post surgery, the patient was well initially, although he was still unable to tolerate oral intake by postoperative Day 7 due to high gastric residual volumes and vomiting. The patient underwent a barium study, which ruled out obstruction due to oedema at the pyloroplasty site (Fig. 1) but showed a hold-up of contrast at D3, which was suspicious for SMA syndrome. Computed tomography (CT) confirmed the diagnosis of SMA syndrome (Figs. 2 & 3). Subsequent management of the patient focused on the delivery of adequate nutrition and a weight gain plan. A naso-jejunal feeding tube that bypassed the obstruction was inserted endoscopically (Fig. 4) to enable enteral feeding. OGD also revealed several gastric ulcers.

On arrival at the hospital, the patient weighed 60 kg with a height of 1.63 m. His body mass index (BMI) was calculated to be 22.6 kg/m^2. At the time of the diagnosis of SMA syndrome,

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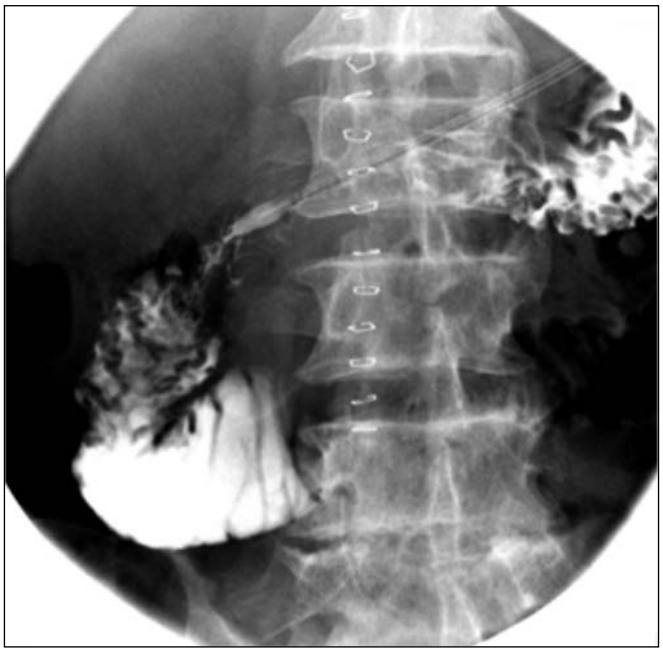

Fig. 1 Barium study radiograph revealed a hold-up of contrast at the D2/3 junction.

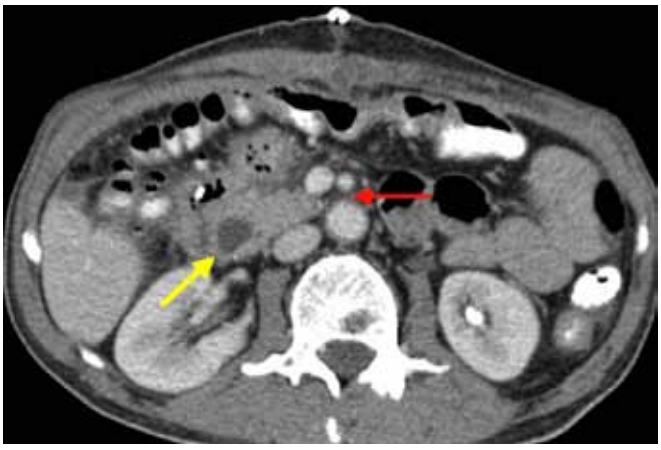

Fig. 2 Axial cut of the CT of the abdomen/pelvis shows a dilated proximal D3 (yellow arrow) and a narrowed aortomesenteric distance (red arrow).

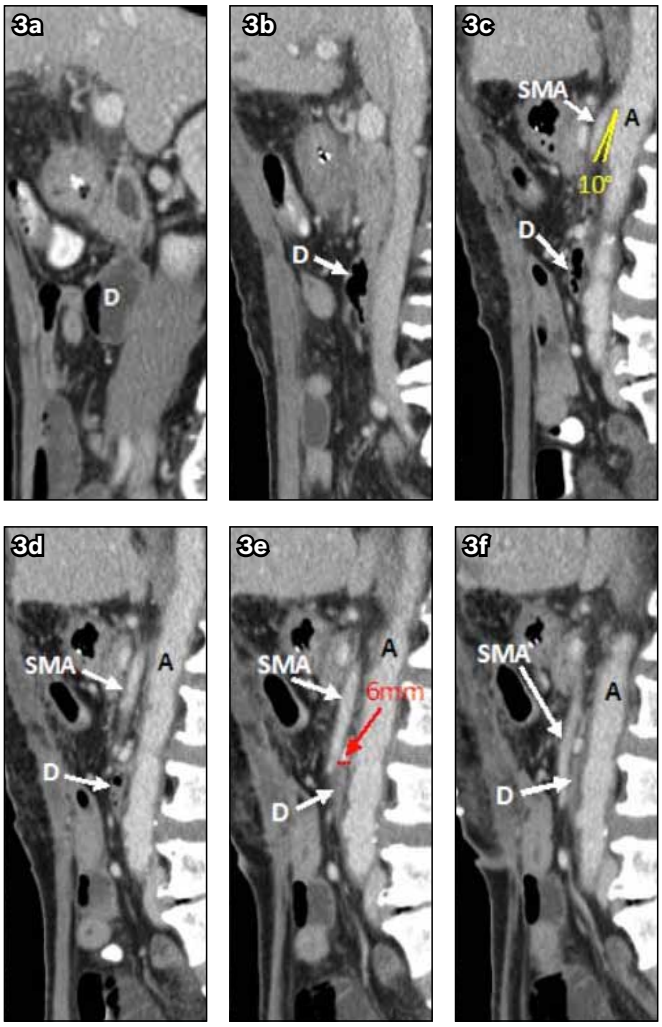

Fig. 3 Sagittal cuts of the CT of the abdomen from right to left show (a) a dilated D2/3 junction to the right of the SMA; (b) the beginning of narrowing in D3; (c) SMA takeoff from the aorta with a reduced aortomesenteric angle (yellow) of 10°; and (d–f) a reduced aortomesenteric distance (red) to 6 mm.
D: duodenum; A: aorta; SMA: superior mesenteric artery

the patient weighed 46.7 kg, with a BMI of 18.3 kg/m^2, after having lost 13.3 kg in a week – a weight loss accounting for approximately 22% of his initial body weight. His albumin was low (24 g/dL), although full blood count, renal function and electrolytes were normal, except for a hypophosphataemia level of 0.45 mmol/L. His phosphate was repleted prior to the commencement of nutritional therapy.

His basal metabolic rate (BMR) was calculated to be 1,050 kcal/day using the Harris-Benedict equation. Based on an activity requirement of light to moderate, his caloric requirement was assessed to be 1,440 to 1,630 kcal/day. His estimated protein requirement was 56 g/day. The patient was started on bolus Ensure (Nestle®) supplements via the naso-jejunal tube at 50 ml per dose, six doses every 24 hours, before a gradual increment to 100 ml, 150 ml, 200 ml and 250 ml per dose. Table I shows the amount of calories the patient received at each dose. As he was at risk of developing refeeding syndrome, his electrolytes, particularly phosphate, potassium and magnesium, were closely monitored as his enteral feeding was increased. The patient was able to tolerate this feeding regimen without nausea or vomiting, and he was discharged home on enteral feeding. Two weeks after the commencement of nutritional therapy, the patient had gained 6 kg in weight. He was well and had tolerated the enteral feeding without problems. A month after nutritional therapy, a repeat barium study and endoscopic examination for follow-up of the ulcers revealed clearance of the previous D3 obstruction and resolution of SMA syndrome (Figs. 5a & b). His albumin level had improved to 38 g/dL and he was restarted on a normal diet as per his pre-morbid state.

DISCUSSION

SMA syndrome arises when there is a loss of aortomesenteric fat around the SMA. The third part of the duodenum, suspended by the ligament of Treitz, sits between the SMA and the aorta, such that any reduction of the aortomesenteric angle leads to mechanical compression of the duodenum.[3] The normal value

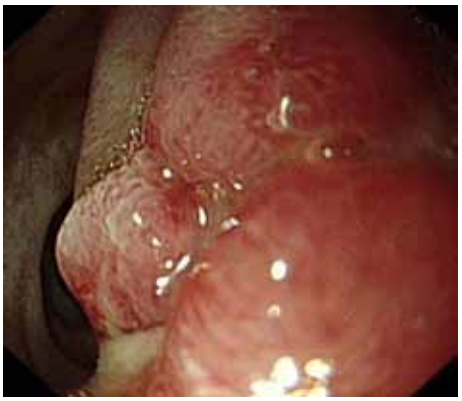

Fig. 4 Endoscopic photograph shows the site of obstruction at D3.

Table I. Calories received by the patient at each dose of supplement.

Volume per dose (ml)	Total volume of supplement (ml)	Caloric equivalent (kcal)
50	300	300
100	600	600
150	900	900
200	1,200	1,200
250	1,500	1,500

of this aortomesenteric angle is 38–65 degrees,[4] but one study with a series of eight patients noted pathological angles of 9–22 degrees.[5] In another study, the normal aortomesenteric distance of 10–28 mm was reduced to just 2–8 mm.[6] Our patient's aortomesenteric angle and distance were 10 degrees and 6 mm, respectively.

Causes of SMA syndrome can be divided into congenital and acquired factors. Congenital factors include a low take-off of the SMA, or an abnormally high origin of the ligament of Treitz,[7] pulling the duodenum toward the root of the mesentery. Acquired factors are numerous, and the syndrome has also been described following cancer, burns, and other surgeries associated with weight loss, such as bariatric surgery.[8] We postulate that in our patient, a thin, elderly Chinese man, it is likely that the stress of recurrent surgeries and a prolonged stay in the surgical high-dependency unit had contributed to the loss of aortomesenteric fat, resulting in the obstruction at D3.

Symptoms of SMA syndrome are nonspecific and include intermittent abdominal pain, nausea, bilious vomiting, early satiety and postprandial bloating.[9] As a result of the nonspecific nature of the symptoms, the diagnosis of SMA syndrome is often delayed and made through a process of exclusion after the consideration of other differential diagnoses, including megaduodenum, pancreatitis and peptic ulcer disease.[5] A barium study is commonly performed as an initial investigation, with the classical finding of SMA syndrome being a hold-up of contrast at D3. The diagnosis can be confirmed with CT of the abdomen. It is important to recognise fluoroscopic features of SMA syndrome and consider this possibility in any patient who might have suffered rapid weight loss and is unable to tolerate oral feeds.

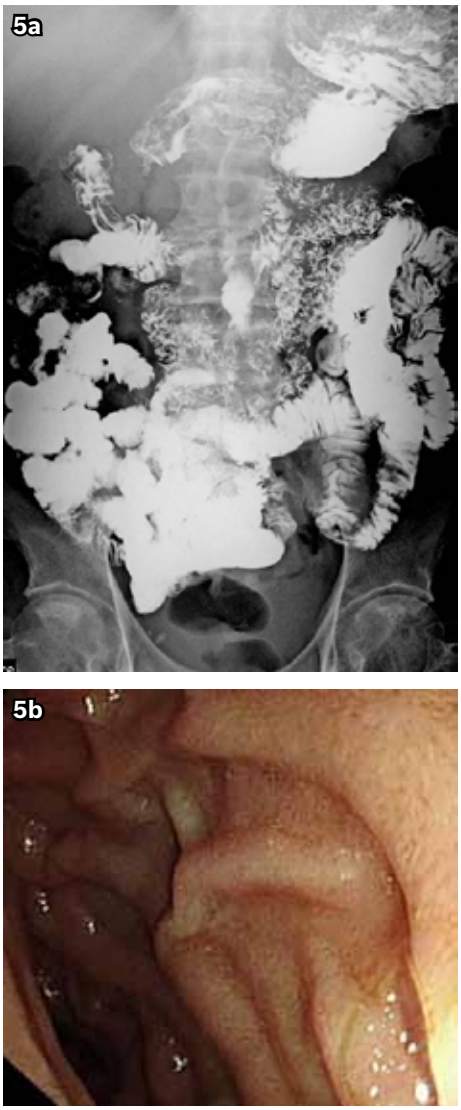

Fig. 5 (a) Radiograph shows repeat barium study at 4 weeks. (b) Photograph of the repeat endoscopy of the D3 at 6 weeks after nutritional therapy shows resolution of the duodenal obstruction.

There are surgical options to treat SMA syndrome, including dissection of the ligament of Treitz, duodenojejunostomy and gastrojejunostomy.[5] However, if the problem is secondary to acute weight loss, many cases of SMA syndrome can be corrected by nutritional therapy, which would lead to weight gain, widening of the aortomesenteric angle and relief of the duodenal obstruction. Non-surgical management of SMA syndrome requires accurate prediction of the energy needs of the patient. Overfeeding and underfeeding are associated with negative effects on the recovery of the patient. Underfeeding can lead to delays in the correction of SMA syndrome, poor wound healing, respiratory muscle weakness, immunosuppression and loss of lean body mass.[10] Overfeeding can cause hyperglycaemia and respiratory failure.[11] The gold standard for the measurement of caloric needs of an individual is indirect calorimetry. However, this method is expensive, not readily available and requires trained technicians for its operation. In place of this, equations have been formulated to estimate the caloric needs of patients. One commonly used equation is the Harris-Benedict formula,

which was used to calculate the basal metabolic rate of the patient. This equation uses variables, such as weight, height, age, gender and activity levels, to assess the caloric consumption of an individual. The Harris-Benedict equation has been found to have an accuracy of 17%–67%.[10] Accuracy implies that the correlation between the measurements obtained using indirect calorimetry and the equation is within 10%.

Refeeding syndrome is associated with electrolyte imbalances and fluid shifts following feeding after a period of malnourishment,[12] which is classically described in patients with anorexia nervosa. Hypophosphataemia is the characteristic feature, although other abnormalities, including hypokalaemia, hypomagnesaemia, disorders of sodium and fluid balances, and alterations of glucose, protein and fat metabolism, have been reported.[12] These electrolyte imbalances may manifest as nausea and vomiting, but may also lead to more serious adverse effects such as respiratory distress, cardiac failure, cardiac arrhythmias, delirium, coma and death.[13] The pathophysiology of refeeding syndrome is postulated to be caused by an increase in serum insulin following a period of fasting.[14] Insulin brings glucose from the extracellular environment into the intracellular environment for metabolism, and in the process, shifts phosphate, magnesium and potassium ions into this intracellular environment as well. This results in the described biochemical abnormalities. The mainstay of management of refeeding syndrome is gradual commencement of nutritional support, especially in patients with substantial weight loss, as well as close monitoring of electrolytes during nutritional therapy and adequate correction of electrolyte abnormalities.[15]

In conclusion, SMA syndrome is a rare problem that affects patients after substantial weight loss. Its diagnosis should be considered in patients when they manifest symptoms of duodenal obstruction. Our patient with SMA syndrome was successfully treated with nutritional therapy, which avoided the risk of another operative intervention. Such nutritional therapy should be carefully titrated according to the patient's caloric and protein requirements, with gradual commencement of therapy and close monitoring of fluid and electrolyte balance.

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