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Superior mesenteric artery syndrome: a single centre experience of laparoscopic duodenojejunostomy as the operation of choice

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ABSTRACT

INTRODUCTION The superior mesenteric artery (SMA) syndrome, or Wilkie's syndrome, is a rare cause of postprandial epigastric pain, vomiting and weight loss caused by compression of the third part of the duodenum as it passes beneath the proximal superior mesenteric artery. The syndrome may be precipitated by sudden weight loss secondary to other pathologies, such as trauma, malignancy or eating disorders. Diagnosis is confirmed by angiography, which reveals a reduced aorto-SMA angle and distance, and contrast studies showing duodenal obstruction.

Conservative management aims to increase intra-abdominal fat by dietary manipulation and thereby increase the angle between the SMA and aorta. Where surgery is indicated, division of the ligament of Treitz, anterior transposition of the third part of the duodenum and duodenojejunostomy have been described.

METHODS We present four cases of SMA syndrome where the intention of treatment was laparoscopic duodenojejunostomy. The procedure was completed successfully in three patients, who recovered quickly with no short-term complications. A fourth patient underwent open gastrojejunostomy (complicated by an anastomotic bleed) when dense adhesions prevented duodenojejunostomy.

CONCLUSIONS The superior mesenteric artery syndrome should be considered in patients with epigastric pain, prolonged vomiting and weight loss. Laparoscopic duodenojejunostomy is a safe and effective operation for management of the syndrome. A multi-speciality team approach including gastrointestinal, vascular and radiological specialists should be invoked in the management of these patients.

KEYWORDS
Superior mesenteric artery syndrome – Wilkie syndrome – Laparoscopic duodenojejunostomy

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Introduction

Superior mesenteric artery (SMA) syndrome is a rare cause of duodenal obstruction. The condition was first described by Rokitansky in 1861,[1] and is occasionally referred to eponymously as Wilkie's syndrome, after D Wilkie's initial description of post mortem findings and early experience in 1921 and his subsequent case series published in 1927.[2,3] The cases were described as 'chronic duodenal ileus'. The third part of the duodenum passes between the aorta and the SMA and is narrowed by the angle between these two vessels in afflicted patients.[4] Compression may be precipitated by progressive weight loss, such as accompanies malignancy, dietary disorders or trauma. It may also result from surgery that reduces the aorto-mesenteric angle; for example, ileoanal pouch surgery producing caudal traction of the small bowel mesentery or scoliosis correcting surgery.[5,6]

Clinical presentation

Patients classically present with a long history of abdominal complaints: prandial epigastric pain, nausea, vomiting, anorexia, weight loss, oesophageal reflux and a feeling of fullness. The discomfort may be relieved by measures that reduce tension on the small bowel mesentery, such as left lateral decubitus or 'knee–chest' positioning, or the Hayes manoeuvre (pressure applied inferior to the umbilicus in a superodorsal direction, which reduces traction of the small bowel on its mesentery). Clinical examination may reveal cachexia, epigastric distension, and a succussion splash. Bowel sounds may be normal or hyperactive. Dehydration and electrolyte abnormalities due to prolonged vomiting are not uncommon.[5,7–9]

Diagnosis

SMA syndrome is diagnosed through a combination of clinical history, examination and radiological imaging.

Currently, contrast-enhanced computed tomography (CT) is the imaging of choice, as this will define the anatomy of the gastrointestinal tract and the relationship of the SMA to the duodenum. It is possible, with multiplanar reconstruction, to accurately measure the aorto-mesenteric angle (AMA) and the distance between the aorta and the SMA (the aorto-mesenteric distance, AMD). Normal values for these measurements are in the ranges 28–65° and 10–34mm, respectively. Angles and distances less than this with evidence of duodenal obstruction proximal to the crossing of the SMA at the level of the third lumbar vertebra would be consistent with a diagnosis of SMA syndrome.[10]

In some cases, colour flow arterial duplex ultrasound is possible. This modality is operator and patient dependent; in some patients, a reduced AMD can be measured accurately.[11] Contrast x-ray imaging of the duodenum may show stenosis or obstruction at the third part of the duodenum (D3), while gastroscopy may reveal a dilated duodenum and abnormal peristalsis.

Management

Conservative

Patients may be managed by gastric and proximal duodenal decompression via a nasogastric tube. Eating should be encouraged and facilitated by the left-lateral or prone position, if tolerated. Feeding via jejunal intubation or total parenteral nutrition can lead to weight regain, with the aim of increasing mesenteric fat and restoring an adequate AMD, thereby reversing the duodenal compression.[4,12,13]

Operative

Several operations have been described for SMA syndrome, aimed at bypassing or relieving the obstruction. Strong's procedure involves dividing the ligament of Treitz and freeing the duodenum by dividing the surrounding peritoneum, allowing the duodenum to occupy a more caudal space, to escape the acute angle between the SMA and the aorta.[6] Strong's procedure has the advantages that it may be performed laparoscopically and does not require an anastomosis; however the failure rate is 25%.[9]

Anterior transposition of the third part of the duodenum at laparotomy is less commonly performed and requires extensive dissection and duodenal reanastomosis but has been found to be effective.[14,15] Duodenojejunostomy has been described at open, laparoscopic, and single-port surgery,[16,17] with or without division of the fourth part of the duodenum.

Our technique is via a laparoscopic approach. The transverse colon is retracted cranially. As the third part of the duodenum is likely to be chronically dilated and readily visible through the avascular part of the colonic mesentery, it may be exposed by dividing the overlying peritoneum with hook diathermy. A loop of proximal jejunum (approximately 30cm from the duodenojejunal flexure) is bought up without tension, and anastomosed to the third part of the duodenum. The side to side anastomosis is formed by creating an enterotomy in each segment of bowel and firing a 45-mm laparoscopic stapling device. The enterotomy is closed with a continuous absorbable V-Loc™ (Covidien) suture. The patency of the anastomosis is confirmed by blue dye instillation through a nasogastric tube.

Case series

Case 1

A 17-year-old male presented as an emergency to the paediatric team with epigastric pain, bloating and a prolonged history of vomiting. His body mass index (BMI) was 14.3. He had a tachycardia and a raised lactate (7mmol/L). CT showed a massively distended stomach and proximal duodenum with an acute calibre change at proximal D3 level (Fig 1). Oesophagogastroduodenoscopy (OGD) revealed grade D oesophagitis and a distended stomach and duodenum, which it was not possible to negotiate. Magnetic resonance angiography revealed an AMA of 34 degrees but an AMD of only 3.5mm. Laparoscopic duodenojejunostomy was performed, with reintroduction of diet and fluids over the next few days. He was discharged without complication after 5 days. At a 40-week follow-up, he had not vomited and was able to tolerate normal diet without symptoms. His BMI had increased to 19.1.

Case 2

A 45-year-old female with a past medical history of smoking, oesophagitis, benign breast disease, caesarean section and hysterectomy presented with epigastric pain and vomiting. Her BMI was 19. CT revealed an AMA of 18 degrees and an AMD of 7mm. A barium meal investigation was normal and OGD initially showed reflux oesophagitis. Subsequent endoscopy revealed an obstruction at D3 with no mucosal abnormality or stricture. Laparoscopic duodenojejunostomy was uncomplicated and the patient was discharged after one

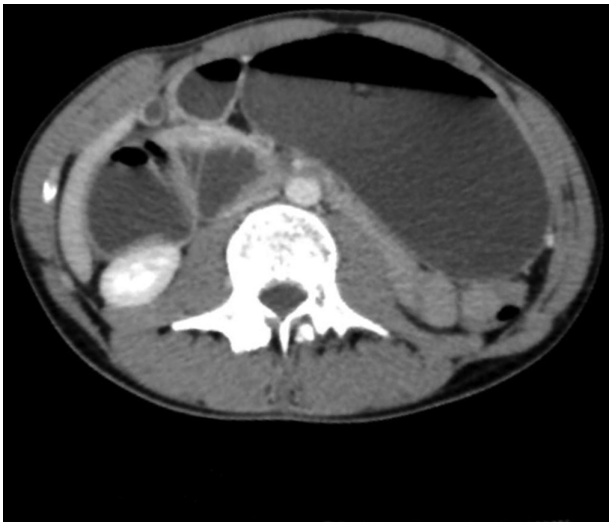

Figure 1 CT showing dilated stomach and obstruction of the third part of the duodenum.

day. She reported improved symptomatology, with no vomiting. However, she did report some postprandial discomfort at clinical follow-up 29 weeks postoperatively. A barium study showed a patent duodenojejunostomy, with no contrast passing D3. Her BMI remained at 19.

Case 3

A 21-year-old female with poorly controlled type 1 diabetes mellitus complained of daily, frequent vomiting and the need to lie on her left side for comfort after eating. Abdominal ultrasound and barium meal investigations were unremarkable. CT showed extrinsic compression at D3. Vascular and upper gastrointestinal surgery multidisciplinary team meetings agreed a diagnosis of SMA syndrome. The AMD was 7mm and her preoperative BMI was 15.7. She underwent laparoscopic duodenojejunostomy, from which she recovered well and was discharged after seven days. Initially, all symptoms resolved and she was well for several months, but after seven months her vomiting returned, accompanied by postprandial diarrhoea. A diagnosis of diabetic gastroparesis was made. It is postulated that an incipient gastroparesis may have led to weight loss, compounded by a resultant SMA syndrome. At 62 weeks follow-up, her BMI was 15.8.

Case 4

A 69-year-old female with an extensive past surgical history of sequential laparotomies for hysterectomy, open cholecystectomy, pyloroplasty and adhesiolysis presented with episodes of postprandial epigastric pain, and nausea without vomiting. Enteroscopy demonstrated extrinsic compression of D3, confirmed on CT, which revealed an AMD of 8mm. A barium meal investigation confirmed significant delayed transit from D3 to D4, with dilatation of the third part of the duodenum to 4.5cm (Fig 2). Mesenteric arterial duplex was normal. Her preoperative BMI was 16.2. The patient underwent laparoscopy with a view to duodenojejunostomy but this proved impossible, due to dense adhesions between colon, small bowel and abdominal wall. Open gastrojejunostomy was undertaken, from which she recovered well, and she was discharged from hospital after seven days. Two days after hospital discharge, she was readmitted with a significant upper gastrointestinal bleed. At endoscopy, a stomal ulcer was identified at the gastrojejunostomy; bleeding was arrested with a single clip placed under vision. At a four-week follow-up, the patient had made a full recovery with resolution of abdominal pain and an appetite much improved. Her BMI was 15.

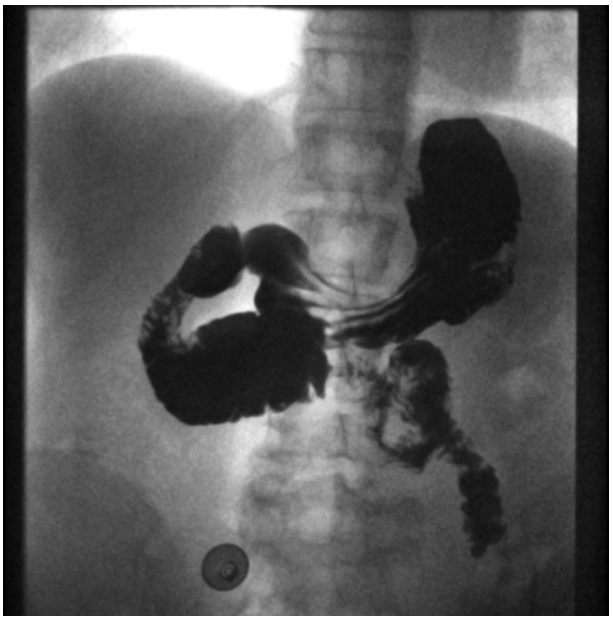

Figure 2 Barium study shows the dilated third part of the duodenum with distal obstruction.

Discussion

Owing to the rarity of SMA syndrome, patients often present after a long illness and may have been misdiagnosed with psychosomatic or malabsorptive conditions, while often being investigated and treated for gastro oesophageal reflux.[9] The diagnosis of SMA syndrome should be considered in all patients presenting with upper abdominal pain, vomiting, weight loss and food intolerance, particularly when they are young and otherwise healthy. A thorough preoperative investigation should be undertaken; upper gastrointestinal endoscopy, contrast imaging and cross-sectional angiography is recommended for both diagnosis and exclusion of important differential diagnoses. We consider that review by a vascular surgeon and radiologist experienced in reporting vascular imaging is crucial to determine the diagnosis.

We have found laparoscopic duodenojejunostomy to be a safe procedure, which achieves satisfactory decompression of the third part of the duodenum and a functional bypass. One case we have included on an 'intention to treat' basis was converted to laparotomy and gastrojejunostomy due to dense colonic adhesions, which made access to the third part of the duodenum unsafe. This patient developed an anastomotic ulcer – a recognised complication of gastrojejunostomy. Functionally, the patient was much improved following the operation, which demonstrates that gastrojejunostomy is a viable rescue option should duodenojejunostomy not be possible.

It is notable that AMA was within normal range in one patient with a reduced AMD. Duodenojejunostomy has provided an excellent result, which supports the logical supposition that compression of the duodenum may be present, even if measurement of the angle alone is not diagnostic. Equally, a reduced AMA should only be acted on in the context of clinical findings.

Conclusion

Duodenojejunostomy is the commonly accepted surgical management of the SMA syndrome. When performed

laparoscopically, it is an effective operation with rapid recovery. We recommend a multidisciplinary team approach to the diagnostic process, including upper gastrointestinal surgeons, vascular surgeons and radiologists with expertise in arterial and abdominal anatomy and pathology.

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