CASE REPORT

Superior mesenteric artery syndrome: A rare etiology of upper intestinal obstruction in adults

Syndrome de la pince aorto-mésentérique : une occlusion haute rare de l’adulte


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Summary The superior mesenteric artery (SMA) syndrome is an atypical cause of high intestinal obstruction in adults. Formerly considered controversial, this syndrome has now been recognized as a real clinical entity which results from extrinsic compression of the third portion of the duodenum by reduction of the angle formed between the SMA and the aorta, usually favoured by rapid and dramatic weight loss. We report a case observed in a 25-year-old female. The abdominal scan provided the diagnosis. Laparoscopic duodenojejunostomy provided cure after failure of initial conservative treatment.

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Case report

A 25-year-old female patient was referred to the emergency unit for an acute abdomen associated with refractory vomiting for 48 h. The most remarkable event in the patient's past history was a brief psychotic disorder 2 months earlier, which was treated with aripiprazole. The patient had lost 10 kg during the last 2 months; her body mass index was 20 at admission. The physical examination revealed a distended abdomen sensitive to palpation of the epigastric region and dull at percussion. A standard X-ray in the upright position showed major gastric distension with an air-fluid level. Routine blood tests were normal. Stomach aspiration produced 3 L of bilo-digestive fluid. A computed tomography of the abdomen and pelvis with contrast injection performed the
Figure 1  Abdominal scan with injection: portal time 60 s after injection. A. Scout view showing the major gastric dilatation. B. Axial slice at the T3 level showing extrinsic compression of the duodenum by the superior mesenteric artery (arrows) due to the small aorto-mesenteric distance (6 mm).

same day confirmed high intestinal obstruction and visualized major dilatation of the stomach and the duodenum. The junction level involved the third portion of the duodenum, which appeared compressed between the aorta and the superior mesenteric artery (SMA) (Fig. 1). Upper gastrointestinal fibroscopy was then performed and showed a normal stomach and duodenal bulb, with an increased diameter of the second portion of the duodenum and extrinsic narrowing of the third portion (Fig. 2). Parenteral nutrition was instituted and a low-pressure aspiration tube was inserted. Abdominal pain and vomiting persisted for 72 h; aspiration produced 1.5 L of bilio-digestive fluid daily. Blood biochemistry revealed progressive global dehydration and acute renal failure by constitution of a third sector. Multidisciplinary discussion led to the decision for a laparoscopic surgical intervention. Laparoscopy revealed a dilated sec-

Figure 2  Gastroduodenal fibroscopic view visualizing the extrinsic compression of the third part of the duodenum (arrow).

Figure 3  Laparoscopic view after exposure of the duodenum revealing the distention of the portion caught in the anatomic vise between the superior mesenteric artery and the posterior structures. The jejunum is normal.
Superior mesenteric artery syndrome

by von Rokitanski, the pathogenic mechanism, as described the third portion of the duodenum between the SMA and tion. The characteristic feature is extrinsic compression of The SMA syndrome is a rare cause of upper intestinal obstruc-

ond portion of the duodenum with severe stenosis of the third portion by extrinsic compression at the level of the SMA (Fig. 3). Laparoscopic side-to-side duodenojejunostomy was performed between the third portion of the duodenum and the first jejunal loop, short circuiting the aorto-mesenteric compression (Fig. 4). The postoperative period was unevent-

Figure 4 Laparoscopic view after the side-to-side duodeno-
jejunal anastomosis created with the endo-GIA stapler inserted via the enterotomy orifices.

Discussion

The SMA syndrome is a rare cause of upper intestinal obstruction. The characteristic feature is extrinsic compression of the third portion of the duodenum between the SMA and the posterior structures. Reported for the first time in 1842 by von Rokitanski, the pathogenic mechanism, as described by Wilkie in 1927 [1], involves a small aorto-mesenteric space, measuring less than 8 mm in height at the level of the third duodenum, associated with a reduced angle of less than 22° between the aorta and the SMA [2,3]. The exact prevalence remains unknown, estimated at 0.013–0.3% in series based on barium studies [1]. The SMA syndrome occurs preferentially in adolescents and young adults, with a discrete female predominance. It is favoured by particular anatomic conditions such as a short or hypertrophic ligament of Treitz, a low origin of the SMA, intestinal malrotation, or lumbar hyperlordosis. Undernutrition or rapid weight loss leads to a constant finding; reduced thickness of the adipose tissue in the aorto-mesenteric space. Many causes have been identified including eating disorders (anorexia nervosa, malabsorption), conditions leading to cachexia (neoplasia, AIDS), situations of hypercatabolism (multiple trauma, burn victims), and surgical causes such as bariatric surgery or correction of spinal malformation. Other causes have been reported including accelerated growth in adolescents with rapid increase in height without weight gain or aneurysm of the abdominal aorta. The anatomic condition leads to a vicious cycle of nausea and vomiting preventing adequate food intake which in turn favours weight loss and aggravation of the syndrome [1,4]. In our patient, the development of the SMA syndrome was probably related to major weight loss over a short period after an acute psychotic episode; arip-

iprazole may have had a potentializing effect on vomiting [5].

There are two clinical expressions of the SMA syndrome. The manifestations of the more common chronic form are non-specific and intermittent with post-prandial epigastric pain, nausea, vomiting and weight loss. In the much more exceptional acute form, as observed in our patient, severe high intestinal obstruction leads to acute potentially life-threatening dilatation of the stomach. Late diagnosis raises the risk of major complications including severe acute fluid-electrolyte imbalance, cardiovascular collapse, gastric rupture, gastric wall pneumatosis and necrosis or portal venous gas [6].

Plain X-rays of the abdomen in the upright and reclining positions contribute little to diagnosis. Significant gastric distention can be seen with an air-fluid level and absence of air in the remainder of the gut. Pneumoperitoneum can be seen after gastric rupture. An upper transit study is not appropriate in this emergency situation. Multi-detector computed tomography with contrast injection is the exploration of choice, confirming the high mechanical obstruction and visualizing the extrinsic compression as the SMA crosses the third portion of the duodenum. Other causes of high obstruction can be ruled out and potential complications (gastric wall pneumatosis or portal venous gas) can be recognized. The aorto-mesenteric angle can be measured on multiplanar and vascular reconstructions [2]. Endoscopy is indicated to search for deleterious effects of the gastric stasis and biliary reflux (gastritis, duodenal ulcer) and to rule out an intrinsic cause of duodenal compression. Endoscopic ultrason can demonstrate the pulsating nature of the compression and provide an objective measurement of the aorto-mesenteric distance without irradiation [7].

First intention treatment is symptomatic. Posture ther-

apy (left lateral decubitus, proctibus) can improve symptoms in half of patients. Continuous low-pressure aspiration together with correction of fluid-electrolyte imbalance is necessary. A weighted gastric tube is inserted under radiographic control to enable optimal enteral nutrition although parenteral nutrition may be needed [1,7]. The different surgical options include side-to-side duo-

denojejunostomy between the duodenum and the first jejunal loop, section of the ligament of Treitz (Strong operation), and gastrojejunostomy. Most surgeons prefer duodenojejunostomy, considered superior to other options with success rates to the order of 80–100% [1]. Du-

denojejunostomy is a simple procedure with low risk of postoperative adhesions. Gersin and Heniford proposed laparoscopic duodenojejunostomy in 1998 [8]. Although this minimally invasive option requires an experienced surgical team, it reduces postoperative pain, shortens the duration of the hospital stay and limits the risk of incision herniation [9].

Conclusion

The SMA syndrome is an unusual cause of potentially life-threatening high mechanical intestinal obstruction in adults. An abdominal scan with injection provides the diagnosis in the acute form. First line treatment is conservative but surgery may be necessary. Minimally invasive laparoscopic duodenojejunostomy is the preferred technique.

Conflict of interest

None.

References