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 duodenojejuno-stomy 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 bilio-digestive fluid. A computed tomography of the abdomen and pelvis with contrast injection performed the
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 1](image1.png)  
**Figure 1** Abdominal scan with injection: portal time 60s 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).

![Figure 2](image2.png)  
**Figure 2** Gastro-duodenal fibroscopic view visualizing the extrinsic compression of the third part of the duodenum (arrow).

![Figure 3](image3.png)  
**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.
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by Wilkie in 1927 [1], involves a small aorto-mesenteric
by von Rokitanski, the pathogenic mechanism, as described
the posterior structures. Reported for the first time in 1842
vation. The characteristic feature is extrinsic compression of
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
pression (Fig. 4). The postoperative period was uneventful.
Intestinal transit was noted on the second postoperative
day with resumption of oral food intake on day 3. The patient
was discharged on the sixth postoperative day. At the follow-up visit at 1 month, the patient had gained 10 kg and was
symptom-free.

Discussion

The SMA syndrome is a rare cause of upper intestinal obstuc-
ction. 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 dis-
crete 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 adi-
pose 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 explo-
ration 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 rec-
ognized. 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. Endo-
scopic ultrasound can demonstrate the pulsating nature
of the compression and provide an objective measure-
ment of the aorto-mesenteric distance without irradiation
[7].

First intention treatment is symptomatic. Posture ther-
apy (left lateral decubitus, proclitus) 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 nutri-
tion although parenteral nutrition may be needed [1,7].
The different surgical options include side-to-side duodo-
enojejunostomy 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].
Duo-
dojejunostomy 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