SAS Journal of Surgery
Abbreviated Key Title: SAS J Surg
ISSN 2454-5104
Journal homepage: https://www.saspublishers.com

OPEN ACCESS

Visceral Surgery

Perforated Gastric Necrosis Secondary to Superior Mesenteric Artery Syndrome: A Rare and Life-Threatening Complication. A Case Report

AZZI Abdenbi[1]*, IBN SBIH Oualid[1], LAZRAQ Farouq[1], JBILOU Aymane[1], YAZOUGH Issam[1], AGOURI Younes[1], AIT LAALIM Said[1]

[1]Department of Visceral Surgery, University Hospital Center of Tangier, Tangier, Morocco

DOI: https://doi.org/10.36347/sasjs.2026.v12i11.002 | **Received:** 19.08.2026 | **Accepted:** 24.10.2026 | **Published:** 03.11.2026

***Corresponding author:** Azzi Abdenbi
Department of Visceral Surgery, University Hospital Center of Tangier, Tangier, Morocco

Abstract **Case Report**

Introduction: Superior mesenteric artery (SMA) syndrome, or Wilkie syndrome, is a rare cause of upper digestive obstruction due to compression of the third portion of the duodenum between the aorta and the SMA. Acute gastric dilatation complicated by gastric necrosis and perforation is an exceptional and life-threatening complication. ***Case presentation:*** We report the case of a 36-year-old woman with no medical history and a very thin body habitus, who presented to the emergency department with a three-day history of profuse, incoercible bilious vomiting, with cessation of stool but preserved passage of gas. Examination revealed abdominal distension with tympany. Laboratory tests showed hypokalemia and functional (prerenal) renal failure. After resuscitation and correction of the biological disorders, the plain abdominal radiograph showed a massively distended stomach, and computed tomography confirmed a voluminous gastric distension with a transition point at the duodenum, consistent with SMA syndrome. At laparotomy, the hugely distended stomach showed areas of necrosis with a perforation on its anterior wall. The necrotic area was resected and an omega-loop gastrojejunostomy was performed. The patient stayed four days in the intensive care unit and was discharged on postoperative day 8. ***Conclusion:*** SMA syndrome should be considered in thin young patients with acute high digestive obstruction. Massive gastric dilatation may lead to gastric necrosis and perforation; early diagnosis, gastric decompression and timely surgery are essential.

Keywords: Superior mesenteric artery syndrome; Wilkie syndrome; Acute gastric dilatation; Gastric necrosis; Gastric perforation; Gastrojejunostomy.

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution **4.0 International License (CC BY-NC 4.0)** which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

1. INTRODUCTION

Superior mesenteric artery (SMA) syndrome, first described in detail by Wilkie in 1921, is a rare cause of proximal intestinal obstruction resulting from extrinsic compression of the third portion of the duodenum in the narrowed angle between the abdominal aorta and the SMA [1,2]. Its reported prevalence ranges from 0.013% to 0.3% in barium studies, and it mainly affects young adults, with a female predominance [2–4]. The main predisposing factor is significant weight loss, which reduces the mesenteric fat pad that normally keeps the SMA away from the aorta [2,3].

The usual presentation is chronic or intermittent, with postprandial epigastric pain, early satiety, nausea, bilious vomiting and weight loss [3,4]. Acute presentations are less common, and massive acute gastric dilatation complicated by gastric ischemia, necrosis or perforation is exceptional, with a high mortality [7,8,10].

We report a rare case of perforated gastric necrosis secondary to SMA syndrome in a young, very thin woman, managed by resection of the necrotic area and gastrojejunostomy. This case is reported in line with the CARE guidelines [11].

2. CASE PRESENTATION

2.1. Patient information

A 36-year-old woman with no significant medical or surgical history and a very thin body habitus presented to the emergency department with profuse, incoercible bilious vomiting that had been progressing for three days. She also reported cessation of stool, while the passage of gas was preserved.

2.2. Clinical findings

Physical examination revealed abdominal distension with tympany on percussion.

2.3. Diagnostic assessment

Laboratory investigations showed hypokalemia and functional (prerenal) renal failure, consistent with dehydration and electrolyte losses from vomiting. Initial management included resuscitation, intravenous rehydration and correction of the biological disorders.

The plain abdominal radiograph showed a massively distended stomach occupying most of the abdomen (Figure 1). Abdominal computed tomography confirmed a voluminous gastric distension with an air-fluid level (Figure 2) and a transition point at the level of the duodenum, consistent with duodenal compression by the superior mesenteric artery.

Figure 1: Plain abdominal radiograph showing a massively distended stomach

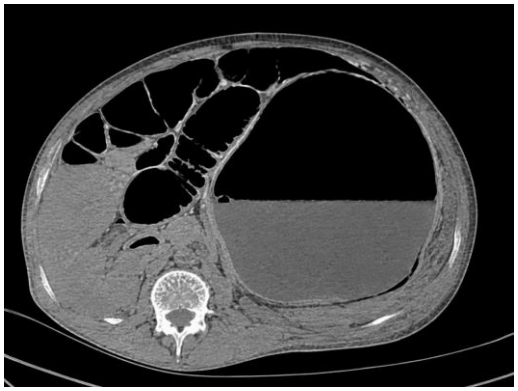

Figure 2: Axial abdominal computed tomography showing a voluminous gastric distension with an air-fluid level

2.4. Therapeutic intervention

After preoperative preparation, the patient underwent surgery. Exploration revealed a hugely distended stomach with areas of necrosis and a perforation on its anterior wall (Figure 3). The procedure consisted of resection of the necrotic area and an omega-loop gastrojejunostomy (Figure 4).

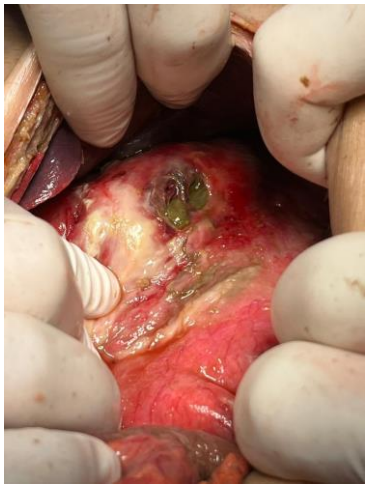

Figure 3: Intraoperative view showing a distended stomach with areas of necrosis and a perforation of the anterior gastric wall

Figure 4: Intraoperative view of the omega-loop gastrojejunostomy being performed

2.5. Follow-up and outcomes

The patient was monitored in the intensive care unit for four days, then transferred to the surgical ward. The postoperative course was favorable, and she was discharged on postoperative day 8.

3. DISCUSSION

SMA syndrome is a rare and often underdiagnosed condition. Normally, the SMA leaves the aorta at an angle of about 25° to 60°, with an aortomesenteric distance of 10 to 28 mm, and the third portion of the duodenum crosses between the two vessels, cushioned by mesenteric fat [2,5]. In SMA syndrome, the angle is reduced, usually to less than 22°–25°, and the distance to less than 8 mm, so that the duodenum becomes compressed [5,6]. The loss of the mesenteric fat pad after rapid or severe weight loss is the main mechanism, and a very thin body habitus, as in our patient, is a classic predisposing factor [2–4]. Other causes include anorexia nervosa, malabsorption, catabolic states, spinal surgery and anatomical variants such as a short ligament of Treitz [2,3,7].

The diagnosis relies on clinical suspicion confirmed by imaging. Computed tomography, especially with angiographic reconstructions, shows the dilatation of the stomach and the proximal duodenum, an abrupt transition at the third portion of the duodenum where it crosses between the aorta and the SMA, and allows measurement of the aortomesenteric angle and distance [3–6]. In our patient, computed tomography showed a massive gastric distension with a transition point at the duodenum, consistent with SMA syndrome.

Acute gastric dilatation is the most dreaded complication. The stomach normally resists ischemia because of its rich, anastomotic arterial supply, which explains why gastric necrosis is rare [8]. However, when intragastric pressure rises above the venous pressure of the gastric wall, venous outflow is impaired, followed by arterial insufficiency, mucosal ischemia and eventually transmural necrosis and perforation [8]. Cases of gastric ischemia, necrosis or perforation complicating SMA syndrome or acute gastric dilatation are rarely reported; they have been described after binge eating and in patients with eating disorders, and some required partial or total gastrectomy [7,9,10]. The duration of symptoms before admission, three days in our patient, probably contributed to the progression from distension to necrosis and perforation.

Management of uncomplicated SMA syndrome is initially conservative, with nasogastric decompression, correction of fluid and electrolyte disorders and nutritional support, enteral beyond the obstruction or parenteral, to restore the mesenteric fat pad [2,3]. Surgery is indicated after failure of conservative treatment; duodenojejunostomy is the most effective procedure, while gastrojejunostomy and division of the ligament of Treitz (Strong procedure) are alternatives [2,3,6]. In the presence of gastric necrosis or perforation, emergency surgery is mandatory. The priorities are to control the perforation, resect all non-viable gastric tissue and restore digestive continuity while bypassing the duodenal obstruction [9,10]. In our patient, the necrosis was limited, which allowed a resection of the necrotic area rather than a total gastrectomy. An omega-loop gastrojejunostomy restored gastric emptying and bypassed the compressed duodenum. Although a gastrojejunostomy may leave residual duodenal stasis compared with a duodenojejunostomy [3], it was the most appropriate option in this emergency setting. Long-term follow-up, together with nutritional rehabilitation and weight gain, is essential to prevent recurrence.

4. CONCLUSION

SMA syndrome is a rare cause of upper digestive obstruction that should be considered in young, thin patients presenting with incoercible bilious vomiting. Massive gastric dilatation may progress to gastric necrosis and perforation, a rare and life-threatening complication. Early diagnosis by computed tomography, prompt gastric decompression and correction of metabolic disorders, and timely surgery in the presence of complications are the keys to a favorable outcome.

Compliance with ethical standards

Acknowledgments

The authors thank the surgical, anesthesia, intensive care and nursing teams involved in the care of this patient.

Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of informed consent

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

REFERENCES

1. Wilkie DPD. Chronic duodenal ileus. Br J Surg. 1921;9(34):204-14.
2. Welsch T, Büchler MW, Kienle P. Recalling superior mesenteric artery syndrome. Dig Surg. 2007;24(3):149-56.
3. Merrett ND, Wilson RB, Cosman P, Biankin AV. Superior mesenteric artery syndrome: diagnosis and treatment strategies. J Gastrointest Surg. 2009;13(2):287-92.
4. Lee TH, Lee JS, Jo Y, Park KS, Cheon JH, Kim YS, *et al.,*Superior mesenteric artery syndrome: where do we stand today? J Gastrointest Surg. 2012;16(12):2203-11.
5. Neri S, Signorelli SS, Mondati E, Pulvirenti D, Campanile E, Di Pino L, *et al.,*Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. J Intern Med. 2005;257(4):346-51.
6. Aranda Escaño E, Perfecto Valero A, Tellaeche de la Iglesia M, Fernández Gómez-Cruzado L, Santidrian Martinez JI. Superior mesenteric artery syndrome (Wilkie syndrome): analysis of a series of 5 cases. Cir Esp. 2020;98(1):48-50.
7. Adson DE, Mitchell JE, Trenkner SW. The superior mesenteric artery syndrome and acute gastric dilatation in eating disorders: a report of two cases and a review of the literature. Int J Eat Disord. 1997;21(2):103-14.
8. Todd SR, Marshall GT, Tyroch AH. Acute gastric dilatation revisited. Am Surg. 2000;66(8):709-10.
9. Sakurai Y, Hirai F, Abe M, Okaya T, Suzuki H, Sugano I. A case of gastric ischemia caused by massive gastric dilatation due to superior mesenteric artery syndrome. Clin J Gastroenterol. 2020;13(6):1066-9.
10. Tse A, Phan N, Ayoubi S, Fenton-Lee D. Necrotic mega-stomach from superior mesenteric artery syndrome. J Surg Case Rep. 2024;2024(7):rjae463.
11. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group. The CARE guidelines: consensus-based clinical case reporting guideline development. Glob Adv Health Med. 2013;2(5):38-43.