SAGE Open Medical Case Reports
Volume 12: 1–5

DOI: 10.1177/2050313X241269593
journals.sagepub.com/home/sco
Sage

Superior mesenteric artery syndrome-induced pancreatitis: Case report

Mejda Zakhama[1], Safa Moussaoui[1], Firas Aissaoui[1], Arwa Guediche[1], Mohamed Hichem Loghmari[1], Marwa Moussaoui[2], Om Kalthoum Sallem[3], Jemni Imen[1], Nabil Ben Chaabene[1], Ahmed Zrig[2] and Leila Safer[1]

Abstract

Superior mesenteric artery syndrome is an acquired vascular compression disorder resulting from the compression of the third portion of the duodenum, which is the first part of the small intestine, leading to a reduction in the space between the aorta and the superior mesenteric artery. Although rare, superior mesenteric artery syndrome-induced pancreatitis has been documented in the literature. This article presents the case of a 20-year-old female patient with a history of colectomy for acute severe colitis, resulting in significant weight loss. She was admitted to the hospital with symptoms of upper bowel obstruction, and the diagnosis of superior mesenteric artery syndrome complicated by acute pancreatitis was made. The patient underwent a nutritional assistance program along with intravenous fluid therapy, resulting in positive outcomes. Superior mesenteric artery syndrome -induced pancreatitis is rarely reported and can be attributed to an occlusive post-papillary syndrome, which causes retrograde reflux of bile into the pancreatic duct, activating inflammation responsible for pancreatitis.

Keywords

Weight loss, superior mesenteric artery syndrome, intestinal obstruction, pancreatitis

Date received: 16 February 2024; accepted: 25 June 2024

Introduction

Superior mesenteric artery syndrome (SMAS) is a rare condition characterized by vascular compression, wherein the third part of the duodenum is compressed between the aorta and the superior mesenteric artery (SMA).[1] Several predisposing factors for SMAS, affecting the aortomesenteric angle, have been identified. SMAS can present with acute manifestations such as proximal small intestinal obstruction or more commonly with chronic manifestations including weight loss, vomiting, decreased appetite, and postprandial abdominal pain.[2] Conservative management, involving parenteral nutrition and nasogastric tube decompression, can be effective. However, SMAS can lead to serious complications due to the close anatomical relationships between D3, the main bile duct, and the Wirsung canal. This report presents a case of SMAS-induced pancreatitis.

Case report

A 20-year-old Caucasian female was admitted to the hospital with abdominal pain and vomiting. The patient had a history of acute severe colitis refractory to intensive medical therapy, resulting in subtotal colectomy with double stomia. One month post-surgery, the patient experienced epigastric pain, bilious vomiting, and a 15-kg weight loss over 3 months. On examination, the patient had weakened with a performance status of 2 and a body mass index (BMI) of 15 kg/m^2. Abdominal tenderness was noted in the epigastric and right upper quadrant areas, with no signs of peritoneal irritation. Stoma openings were functional. Initial blood investigations revealed a normal white count, a hemoglobin level of 8.38 g/dL, slightly low albumin level at 34 g/L, and cytolysis

[1]Department of Gastroenterology, University Hospital Fattouma Bourguiba, Monastir, Tunisia
[2]Department of Radiology, University Hospital Fattouma Bourguiba, Monastir, Tunisia
[3]Department of Nutrition, University Hospital Fattouma Bourguiba, Monastir, Tunisia

Corresponding Author:
Mejda Zakhama, Department of Gastroenterology, University Hospital Fattouma Bourguiba, Monastir 5000, Tunisia.
Email: zakhama.majda@gmail.com

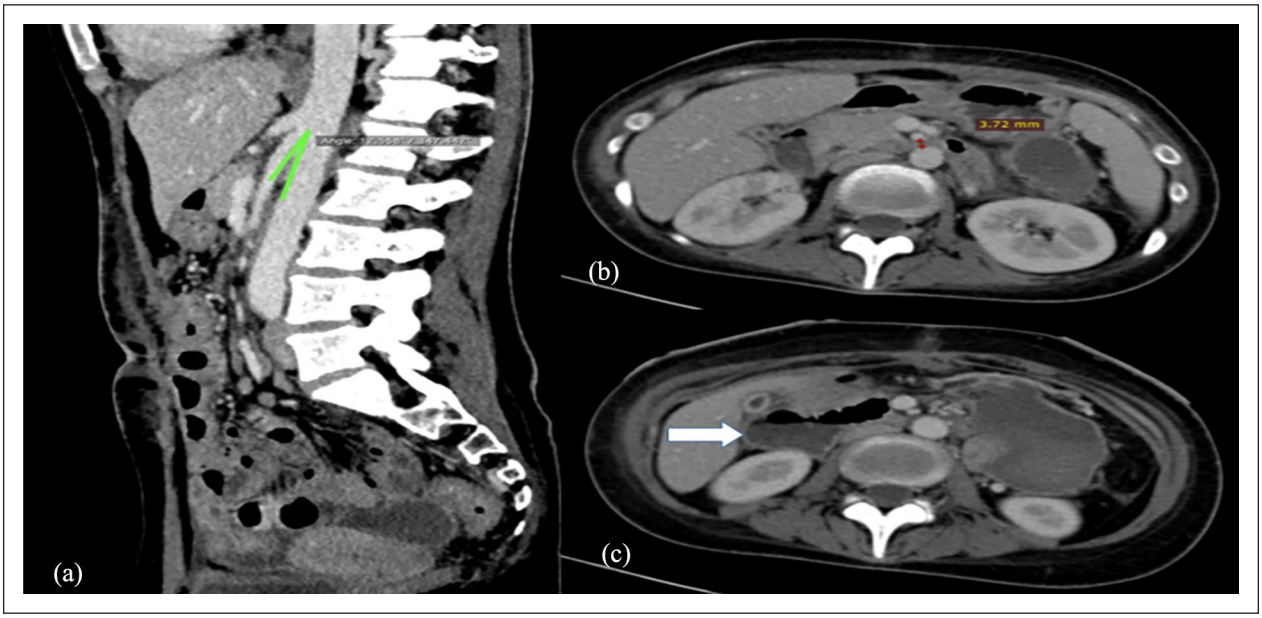

Figure 1. Sagittal (a) and axial (b,c) abdominal CT scan in aortic phase show a SMAS : Acute angulation of the superior mesenteric artery (12 °) (a) , with reduced aortomesenteric distance (3,7 mm) (b) resulted in compression of the third portion of the duodenum, leading to dilatation of the proximal duodenum (arrow in c).

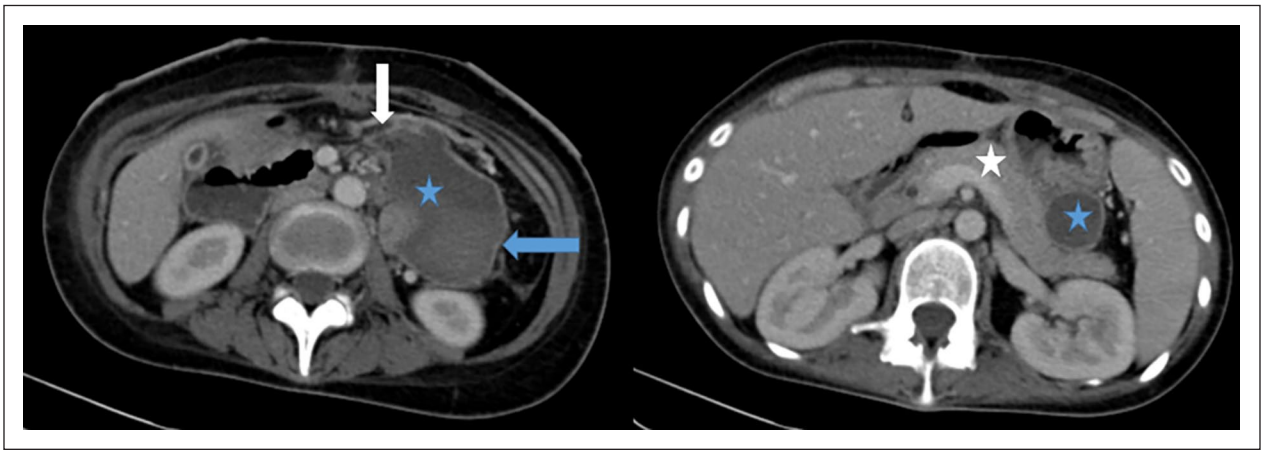

Figure 2. Axial computed tomography scan images in arterial phase show acute necrotizing pancreatitis involving only the peripancreatic tissues CT scan images in arterial phase showing the enhancement of the entire pancreatic parenchyma (white star) associated with heterogeneous peripancreatic collections (blue stars), fully encapsulated, containing area of fat (white arrow), fluid density, and areas with greater attenuation (blue arrow) compatible with walled-off necrosis.

predominant to aspartate aminotransferase and cholestasis on liver enzyme panel. C-reactive protein levels were elevated at 60 mg/L. However, lipasemia and other biological examination results were unremarkable. Abdominal computed tomography (CT) scan showed an acute angle of 16° between the SMA and the aorta (normal range 25°–65°), with compression on the anterior third part of the duodenum. Proximal duodenal dilatation was observed with an SMA-aorta distance of 3.7 mm (Figure 1). Upper endoscopy revealed biliary gastric stasis, confirming the diagnosis of SMAS. Additionally, acute pancreatitis was diagnosed based on acute epigastric pain and Balthazar D grade acute pancreatitis observed on CT scan (Figure 2). However, etiological assessment for acute pancreatitis was negative, with no evidence of alcohol abuse, drug-related causes, hypercalcemia, or hypertriglyceridemia. Ultrasonography and magnetic resonance angiography showed no biliary stone, pancreatic mass, or malformations.

Although idiopathic case could not be denied, the patient was diagnosed with acute pancreatitis resulting from increased intraduodenal pressure due to SMAS.

The patient was initially managed with total parenteral nutrition and a continuous nasogastric aspiration tube for decompression. Subsequently, she commenced a nutritional

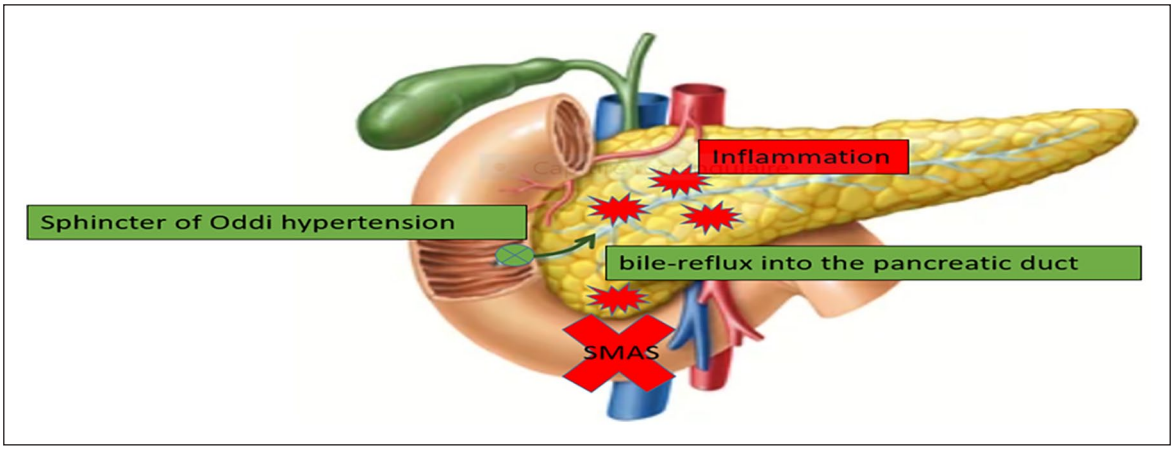

Figure 3. Labeled diagram illustrating the mechanism of superior mesenteric artery syndrome -induced pancreatitis.

assistance program via a nasojejunal tube for enteral alimentation (Nutrison® 1.0 Kcal/ml), starting with an intake of 10 kcal/kg/day, rehydration with Crystalloid Fluids (1000 ml of 0.9% Saline), and antiemetic medication (metoclopramide: 10 mg three times a day for 5 days). After 10 days, the nasogastric tube was removed, and the patient transitioned to an oral hypercaloric diet with good tolerance.

Fifteen days post-admission, the patient was asymptomatic with good tolerance of oral nutrition and had gained weight. Due to the successful medical treatment, surgical intervention was unnecessary, and the patient was discharged home. Three weeks later, she had gained 3 kg. During a 4-year follow-up period, the patient remained asymptomatic with no recurrence of acute pancreatitis or upper bowel obstruction.

Discussion

SMAS was first described by Von Rokitansky in 1861 as a clinical manifestation resulting from vascular compression of the third part of the duodenum between the aorta and the SMA.[3]

Various predisposing factors influencing the aortomesenteric angle have been identified, including excessive weight loss leading to depletion of mesenteric and retroperitoneal fat, spine diseases, congenital conditions like a short Treitz ligament, or acquired anatomical abnormalities post-surgical correction of aneurysms.[3,4] In our case, the patient was a typical thin young female; however, SMAS can also occur in men and across all age groups.[5] Our patient had a BMI of 15 kg/m^2 with a significant weight loss of 15 kg over 3 months following acute severe colitis and subsequent surgery.

The typical clinical presentation of SMAS includes intermittent or postprandial abdominal pain (59%–81%), though acute presentations are rare and characterized by rapid development of upper intestinal ileus followed by vomiting, nausea, and anorexia exacerbating weight loss.[6] Diagnosis is aided by CT scan, which reveals a reduced angle between the SMA and the aorta (normally between 38° and 65°) and a decreased aorto-mesenteric distance (2–8 mm, compared to 10–33 mm in normal cases).[2] Diagnosis is generally confirmed based on symptoms, an aortomesenteric angle $<22°$, and an aortomesenteric distance <8 mm.[2]

In recent years, the treatment approach for SMAS has shifted toward medical management, utilizing advances in both enteral and parenteral nutrition. This involves decompression of the stomach and duodenum through nasogastric tube placement, correction of hydroelectrolyte imbalances, and initiation of enteral hypercaloric nutrition via nasojejunal tube. The success rate of medical treatment is estimated to be around 72%, with a recurrence rate of 30%.[7] Surgical options, such as gastrojejunostomy or duodenojejunostomy, are considered if medical treatment fails.[3]

Persistent obstruction in the third duodenum can lead to complications such as biliary reflux, gastritis, duodenal ulcers, and pancreatitis due to the close anatomical proximity of the third part of the duodenum, the main bile duct, and the Wirsung canal.

The association between acute pancreatitis and SMAS is rare, with very few reported cases.[4,5,8–10] In these cases, the patients presented with an abdominal pain and distension, and repeated vomiting. The diagnosis of SMAS-induced pancreatitis was made in all cases after ruling out the usual causes with a magnetic resonance cholangiopancreatography showing no evidence of pancreatic duct anomaly or biliary stone with no evidence of alcohol abuse, drug-related causes, normal triglyceride and phosphocalcium levels. The diagnosis of SMAS, complicating poor nutritional status, was made based on CT scan data in all cases. The clinical presentation of our patient is similar to reported cases, except that in our case there was no organ failure. Kojima et al.[5] reported the case of a 76-year-old patient who presented in a state of shock. The patient was diagnosed with acute pancreatitis with hypovolemic shock resulting from increased intraduodenal pressure due to SMAS and gas bloat.[5] The association between acute pancreatitis and SMAS may be attributed to retrograde reflux of bile into the pancreatic duct due to a secondary occlusive post-papillary syndrome, leading to activation of pancreatic enzymes and subsequent pancreatitis (Figure 3).[4,11,12]

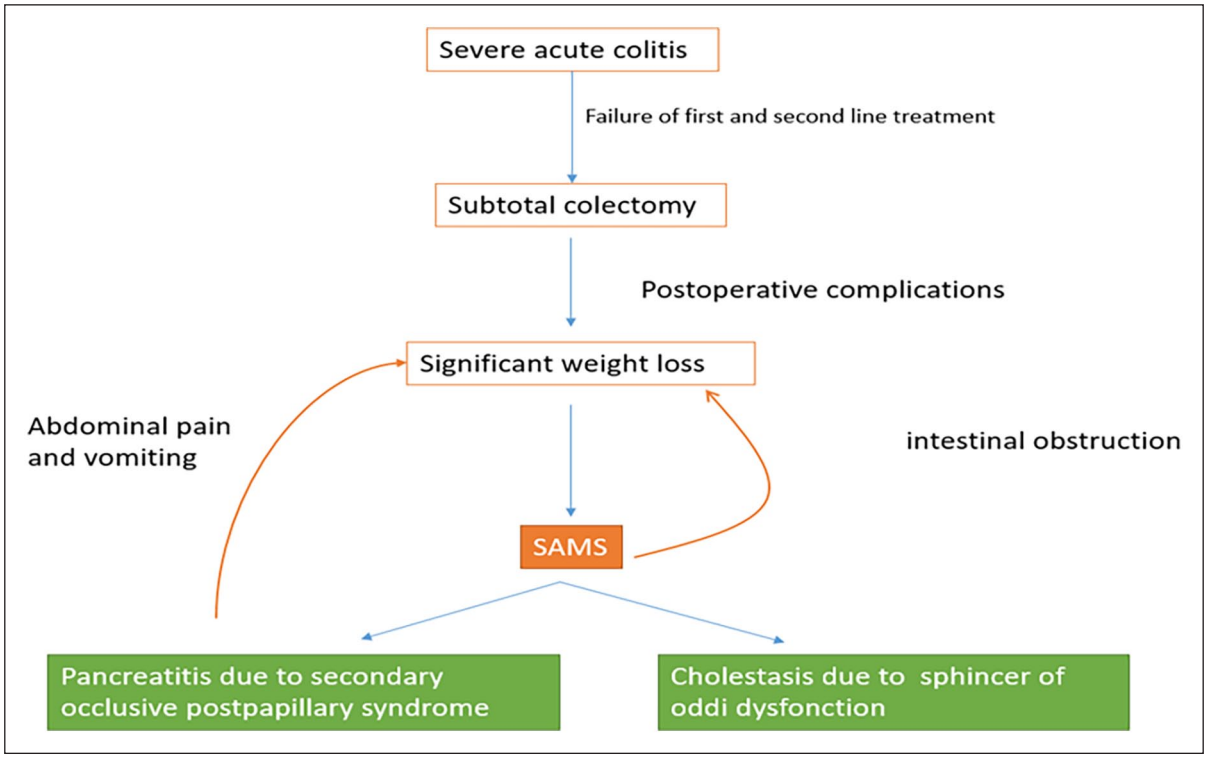

Figure 4. Schematic diagram of the sequence of events in this case report.

In our case, SMAS was considered the cause of acute pancreatitis after ruling out other etiologies such as biliary stones, alcohol abuse, drug intake, hypercalcemia, or hypertriglyceridemia, and the pancreatitis resolved without recurrence, making a genetic etiology unlikely.

Furthermore, the patient also presented with cholestasis, which could be explained by compression of the distal portion of the common bile duct or sphincter of Oddi dysfunction within the framework of SMAS. Imaging did not reveal dilatation of the common bile duct, suggesting sphincter of Oddi dysfunction as the likely cause of cholestasis in our case.[13]

The sequence of events in this case report is illustrated in Figure 4.

Conclusion

SMAS is a multifaceted rare entity with diverse presentations due to the close anatomical relationships between the third part of the duodenum, the main bile duct, and the Wirsung canal. Pancreatitis is a rarely reported complication of SMAS, caused by retrograde reflux of bile into the pancreatic duct, leading to inflammation.

Acknowledgements

A pre-print of the manuscript is available at: https://www.authorea.com/doi/full/10.22541/au.166374368.82841992

Authors contributions

M. Z. Conception; S. M. Writing the manuscript; F. A. Data collection; A. G. Data collection; M. H. L. Literature review; M. M. Interpretation of radiological data; O. K. S. Analysis and interpretation; J. I. Critical review; N. B. C. Design; A. Z. Supervision; L. S. Supervision.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases.

Informed consent

Written informed consent was obtained from the patient for her anonymized information to be published in this article.

ORCID iD

Mejda Zakhama https://orcid.org/0000-0003-3127-527X

References

1. Karki B, Pun B, Shrestha A, et al. Superior mesenteric artery syndrome. *Clin Case Rep* 2020; 8(11): 2295.
2. Oka A, Awoniyi M, Hasegawa N, et al. Superior mesenteric artery syndrome: diagnosis and management. *World J Clin Cases* 2023; 11(15): 3369–3384.
3. Welsch T, Büchler MW and Kienle P. Recalling superior mesenteric artery syndrome. *Dig Surg* 2007; 24(3): 149–156.
4. Sihuay-Diburga DJ, Accarino-Garaventa A, Vilaseca-Montplet J, et al. Acute pancreatitis and superior mesenteric artery syndrome. *Rev Esp Enferm Dig* 2013; 105(10): 626–628.
5. Kojima S, Suzuki K, Katayama N, et al. Superior mesenteric artery syndrome as a cause of acute pancreatitis. *BMJ Case Rep* 2016; 2016: bcr2016217073.
6. Biank V and Werlin S. Superior mesenteric artery syndrome in children: a 20-year experience. *J Pediatr Gastroenterol Nutr* 2006; 42(5): 522–525.
7. Shin MS and Kim JY. Optimal duration of medical treatment in superior mesenteric artery syndrome in children. *J Korean Med Sci* 2013; 28(8): 1220–1225.
8. Gwee K, Teh A and Huang C. Acute superior mesenteric artery syndrome and pancreatitis in anorexia nervosa. *Austr Psychiatry* 2010; 18(6): 523–526.
9. Inoue M, Uchida K, Otake K, et al. Development of acute pancreatitis after Nissen fundoplication. *Pediatr Int* 2015; 57(2): e48–e49.
10. Castro BN, Ferreira AR, Graça S, et al. Combined superior mesenteric artery syndrome and nutcraker syndrome presenting as acute pancreatitis: a case report. *J Vasc Bras* 2023; 22: e20220161.
11. Kamisawa T, Kurata M, Honda G, et al. Biliopancreatic reflux-pathophysiology and clinical implications. *J Hepatobiliary Pancreat Surg* 2009; 16(1): 19–24.
12. Nakamura T, Okada A, Higaki J, et al. Pancreaticobiliary maljunction-associated pancreatitis: an experimental study on the activation of pancreatic phospholipase A2. *World J Surg* 1996; 20(5): 543–550.
13. Jeune F, d'Assignies G, Sauvanet A, et al. A rare cause of obstructive jaundice and gastric outlet obstruction. *World J Gastrointest Surg* 2013; 5(6): 192–194.