



Case Study

Superior Mesenteric Artery Syndrome Associated with Chronic Pancreatitis and Suspected Pancreatic Tail Malignancy in An Elderly Male: A Case Report

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Superior mesenteric artery (SMA) syndrome is a rare but potentially serious cause of proximal intestinal obstruction resulting from compression of the third part of the duodenum between the abdominal aorta and the superior mesenteric artery. The condition is often difficult to diagnose because its symptoms mimic many other gastrointestinal disorders. Delayed diagnosis may lead to severe malnutrition, electrolyte imbalance, and deterioration in quality of life. We report the case of a 72-year-old male who presented with abdominal pain, persistent vomiting, abdominal distension, nausea, and poor oral intake. He had a background history of type 2 diabetes mellitus, chronic obstructive pulmonary disease, and previously treated pulmonary tuberculosis. Imaging studies demonstrated features suggestive of SMA syndrome with acute angulation between the aorta and superior mesenteric artery. Additional investigations revealed chronic calcific pancreatitis, a cystic lesion in the pancreatic tail with suspicion of underlying pancreatic malignancy, gastric food stasis on upper gastrointestinal endoscopy, and significant weight loss. Conservative measures were insufficient because of persistent symptoms and nutritional compromise. The patient subsequently underwent exploratory laparotomy, duodenojejunostomy, and feeding jejunostomy. Following surgery, enteral nutrition was gradually initiated, symptoms improved, and the patient was discharged in stable condition. This case highlights the importance of considering SMA syndrome in elderly patients presenting with chronic vomiting and weight loss, particularly when associated with chronic pancreatic disease and severe nutritional depletion. Early diagnosis using cross-sectional imaging and timely surgical intervention can significantly improve patient outcomes.

Keywords: Superior mesenteric artery syndrome, Wilkie syndrome, duodenal obstruction, chronic pancreatitis, duodenojejunostomy, pancreatic lesion.

INTRODUCTION

Superior mesenteric artery syndrome, also known as Wilkie syndrome, is an uncommon cause of upper gastrointestinal obstruction. The syndrome occurs when the third portion of the duodenum becomes compressed between the abdominal aorta posteriorly and the superior mesenteric artery anteriorly. Normally, a cushion of mesenteric fat maintains an adequate angle and distance between these two vascular structures, allowing the duodenum to pass freely without compression. Any condition that reduces this protective fat pad can narrow the aortomesenteric angle and lead to external

compression of the duodenum. SMA syndrome is rare, with an estimated prevalence ranging from 0.013% to 0.3%. The condition can occur at any age but is more commonly described in adolescents and young adults. Nevertheless, it may also occur in elderly individuals, especially in the presence of chronic illness, severe weight loss, malignancy, prolonged bed rest, malnutrition, or catabolic states. Because of its rarity and non-specific presentation, the diagnosis is frequently delayed. Patients with SMA syndrome typically present with postprandial abdominal pain, early satiety, nausea, vomiting,

abdominal distension, and progressive weight loss. In many patients, the weight loss further aggravates the disease by reducing retroperitoneal fat, thereby worsening the compression. This creates a vicious cycle of obstruction, reduced intake, and continued nutritional deterioration. Several predisposing factors have been reported. Rapid weight loss due to malignancy, chronic infection, eating disorders, trauma, burns, prolonged immobilization, or chronic systemic diseases can lead to reduction of mesenteric fat. Surgical correction of spinal deformities, especially scoliosis surgery, has also been implicated because of changes in the anatomical relationship between the aorta and SMA. Congenital anatomical variations may contribute in certain patients. The diagnosis of SMA syndrome requires a high index of suspicion. Clinical symptoms alone are insufficient because they overlap with peptic ulcer disease, gastric outlet obstruction, pancreatitis, gastroparesis, intestinal pseudo-obstruction, and malignant gastrointestinal obstruction. Crosssectional imaging such as computed tomography (CT) angiography or magnetic resonance imaging plays a crucial role in demonstrating reduced aortomesenteric angle and distance, proximal duodenal dilatation, and evidence of duodenal compression. Management depends on the severity of symptoms and nutritional status. Conservative treatment focuses on nutritional rehabilitation, weight gain, correction of electrolyte imbalance, postural therapy, and enteral feeding. Surgical intervention becomes necessary when conservative measures fail or when there is persistent obstruction. Duodenojejunostomy is considered the preferred surgical procedure because it bypasses the obstructed segment and provides excellent long-term outcomes. The coexistence of SMA syndrome with chronic pancreatitis and a pancreatic tail lesion is uncommon. Chronic pancreatic disease can contribute to malnutrition and severe weight loss, potentially predisposing patients to SMA syndrome. Furthermore, symptoms from pancreatic pathology may overlap with manifestations of SMA syndrome, making diagnosis even more challenging. We present a case of SMA syndrome in a 72-year-old male with chronic calcific pancreatitis, severe weight loss, gastric stasis, and a suspected pancreatic tail malignancy who required surgical treatment with duodenojejunostomy and feeding jejunostomy.

CASE PRESENTATION

A 72-year-old male presented with complaints of abdominal pain, recurrent vomiting, nausea, abdominal distension, and inability to tolerate adequate oral intake. His symptoms had progressively worsened, leading to reduced food consumption and significant nutritional compromise. The vomiting was persistent and occurred predominantly after meals. He also reported generalized weakness and reduced appetite.

The patient had a known history of type 2 diabetes mellitus. He was also diagnosed with chronic obstructive pulmonary disease and had a previous history of pulmonary tuberculosis that had been treated in the past. There was no evidence of active pulmonary infection at the time of admission. Because of the persistent gastrointestinal symptoms, the patient underwent extensive evaluation. Physical examination revealed abdominal discomfort and distension. Repeated vomiting and poor intake raised concern regarding proximal intestinal obstruction. The patient had experienced substantial weight loss, although exact previous body weight records were unavailable. Nutritional depletion was clinically evident. Routine laboratory investigations and radiological studies were performed. Upper gastrointestinal endoscopy demonstrated food stasis within the stomach, particularly involving the fundus and body. The persistence of food residue despite fasting suggested impaired gastric emptying or distal obstruction. Cross-sectional abdominal imaging was subsequently obtained. Computed tomography and magnetic resonance imaging demonstrated narrowing of the angle between the superior mesenteric artery and the abdominal aorta, raising suspicion for SMA syndrome. Associated dilatation of the proximal bowel supported the diagnosis of duodenal compression. Additional findings complicated the clinical picture. Imaging revealed chronic calcific pancreatitis with pancreatic duct dilatation and a lesion involving the pancreatic tail. A cystic component was also identified. The radiological appearance raised concern regarding possible underlying pancreatic malignancy. The patient also had minimal ascites and diffuse mesenteric fat stranding.

MRI with MRCP demonstrated diffuse pancreatic atrophy involving the head and body with complete fatty replacement. Multiple intraductal calculi were present, and the main pancreatic duct was dilated. A heterogeneous lesion involving the distal pancreatic tail was identified with associated restricted diffusion, warranting concern for neoplastic pathology. However, definitive histopathological confirmation was not available. Imaging also identified acute angulation between the superior mesenteric artery and the abdominal aorta, supporting the diagnosis of SMA syndrome. This finding, together with severe weight loss, gastric food stasis, persistent vomiting, and proximal bowel obstruction, strongly suggested clinically significant duodenal compression.

Chest CT demonstrated hyperinflation of both lungs, mild centrilobular emphysematous changes, and fibrotic lesions related to previous pulmonary tuberculosis. No active pulmonary infection was identified. The patient's symptoms persisted despite supportive treatment. Nutritional compromise and inability to maintain adequate oral intake increased the risk of further deterioration. Given the severity of symptoms and evidence of persistent obstruction, surgical management was considered. After multidisciplinary evaluation, the patient underwent exploratory laparotomy. Intraoperatively, duodenojejunostomy was performed to bypass the obstructed duodenal segment. A feeding jejunostomy was also created to facilitate postoperative nutritional support. Following surgery, enteral nutrition was gradually initiated. Tube feeding was continued according to nutritional requirements. Oral intake was advanced slowly as tolerated. The patient received antibiotics, supportive care, glycaemic control, and nutritional rehabilitation during the postoperative period. His symptoms improved progressively after surgery. Episodes of vomiting decreased, oral intake improved, and nutritional support was continued. The patient remained haemodynamically stable and was discharged with dietary advice, medications, and instructions for follow-up.

DISCUSSION

Superior mesenteric artery (SMA) syndrome, also known as Wilkie syndrome, is a rare cause of proximal intestinal obstruction resulting from

compression of the third part of the duodenum between the abdominal aorta and the superior mesenteric artery. The condition is frequently difficult to diagnose because its clinical manifestations are nonspecific and overlap with several gastrointestinal disorders. The present case is particularly noteworthy because SMA syndrome occurred in a 72-year-old man with chronic calcific pancreatitis, severe nutritional depletion, persistent vomiting, and a pancreatic tail lesion suspicious for malignancy. The combination of advanced age and significant pancreatic pathology made the clinical presentation and diagnosis more complex. The pathophysiology of SMA syndrome is closely related to loss of the retroperitoneal fat pad surrounding the superior mesenteric artery. Normally, this fat pad maintains an adequate angle and distance between the abdominal aorta and SMA and allows the third part of the duodenum to pass freely. Reduction in this fat pad can result in narrowing of the aortomesenteric angle and distance, leading to external compression of the duodenum. In the present patient, persistent vomiting, poor oral intake, chronic pancreatic disease, and substantial weight loss may have contributed to depletion of the protective mesenteric fat. Imaging demonstrated acute angulation between the SMA and aorta with associated proximal bowel dilatation, supporting clinically significant duodenal compression. An important comparison can be made with the case reported by Kulkarni, in which a patient with chronic pancreatitis developed SMA syndrome following severe weight loss and anorexia. The patient presented with recurrent postprandial vomiting and was found to have gastric and duodenal dilatation up to the level of the SMA. The authors proposed that weight loss associated with chronic pancreatitis resulted in loss of mesenteric fat and subsequent duodenal compression. This mechanism is highly relevant to our patient, who also had chronic calcific pancreatitis, pancreatic atrophy, poor oral intake, persistent vomiting, and weight loss. However, our case differs because the patient was substantially older and had an additional pancreatic tail lesion with radiological suspicion of malignancy. Kulkarni's case therefore supports the nutritional mechanism in our patient, although the exact contribution of the pancreatic lesion cannot be confirmed because histopathological evidence of malignancy was unavailable.

The relationship between chronic pancreatitis and nutritional deterioration is clinically important. Chronic pancreatitis can lead to reduced food intake because of abdominal pain and anorexia and may also contribute to malnutrition through pancreatic exocrine dysfunction and malabsorption. In the present case, the imaging findings of chronic calcific pancreatitis, pancreatic duct dilatation, intraductal calculi, and pancreatic atrophy indicate longstanding pancreatic disease. The additional pancreatic tail lesion raised concern for malignancy, which could potentially contribute to further weight loss. Nevertheless, because histopathological confirmation was not available, malignancy should be described as suspected rather than established. The age of the patient represents another distinctive feature. SMA syndrome is often associated with younger individuals, particularly adolescents and young adults, but it can occur in older adults when significant weight loss or chronic illness results in depletion of mesenteric fat. Tsukayama and colleagues reported SMA syndrome in a man in his eighties who presented with severe vomiting. CT, endoscopy, and gastroduodenography supported the diagnosis. Conservative treatment consisting of gastric decompression, fasting, and nutritional therapy was unsuccessful, and the patient underwent laparoscopic duodenojejunostomy with an uncomplicated postoperative course. This case, together with our patient's presentation, demonstrates that advanced age should not exclude SMA syndrome from the differential diagnosis of persistent vomiting and nutritional deterioration. A similar observation was reported by Jain and colleagues, who described SMA syndrome in an elderly woman. The diagnosis was established through radiological demonstration of vascular compression of the duodenum, and the patient was successfully managed surgically. Their report emphasized that SMA syndrome should be considered when an elderly patient presents with symptoms of duodenal obstruction and radiological evidence of an abnormally narrow aortomesenteric relationship. Our case supports this observation because persistent vomiting and abdominal distension initially suggested a more common gastrointestinal or pancreatic cause, while cross-sectional imaging subsequently demonstrated the vascular compression. The clinical presentation in our patient was consistent with previously reported cases. He had abdominal

pain, nausea, recurrent predominantly postprandial vomiting, abdominal distension, poor oral intake, and significant nutritional deterioration. Similar symptoms have been described by Mangano et al. in patients treated with laparoscopic duodenojejunostomy and by other authors reporting SMA syndrome in adults. In these cases, vomiting, abdominal pain, early satiety, food intolerance, and weight loss were major presenting features. The similarity of these symptoms highlights why SMA syndrome can be overlooked, particularly when the patient has another established gastrointestinal disease such as chronic pancreatitis. The diagnostic process in our case illustrates the importance of combining endoscopic and cross-sectional investigations. Upper gastrointestinal endoscopy demonstrated significant gastric food stasis despite fasting. Although endoscopy cannot directly establish SMA syndrome, it is valuable for excluding intraluminal causes of gastric outlet or duodenal obstruction. Cross-sectional imaging subsequently demonstrated narrowing of the aortomesenteric angle, proximal bowel dilatation, and compression at the third portion of the duodenum. At the same time, CT and MRI provided important information about the patient's chronic pancreatitis and pancreatic tail lesion. Therefore, imaging was essential not only for confirming the suspected vascular compression but also for identifying coexisting pathology. The importance of cross-sectional imaging has also been demonstrated in other reported cases. Mangano and colleagues described patients in whom CT demonstrated extrinsic compression of the third part of the duodenum and reduced aortomesenteric measurements. Their experience emphasized that cross-sectional imaging should be integrated with endoscopic and clinical findings when evaluating suspected SMA syndrome. Similarly, Tsukayama et al. used enhanced CT together with endoscopy and gastroduodenography to establish the diagnosis in an elderly patient. These reports are consistent with the diagnostic approach used in our patient. Weight loss is not merely a consequence of SMA syndrome but can also be an important factor in its development. This creates a potentially self-perpetuating cycle in which vomiting and food intolerance cause reduced caloric intake and weight loss, while further loss of retroperitoneal fat increases vascular compression and worsens obstruction. The present patient appeared to

have several factors contributing to this cycle, including chronic pancreatitis, poor appetite, persistent vomiting, and suspected pancreatic pathology. Similar weight-loss-related mechanisms have been described in patients with chronic pancreatitis-associated SMA syndrome and in other reported cases of SMA syndrome. Overall, comparison with previously reported cases demonstrates that the present case shares the classical features of SMA syndrome, particularly persistent vomiting, weight loss, proximal gastrointestinal obstruction, and radiological evidence of duodenal compression. However, it is unusual because of the patient's advanced age and the coexistence of chronic calcific pancreatitis and a pancreatic tail lesion suspicious for malignancy. The case further demonstrates the potential relationship between chronic pancreatic disease, nutritional depletion, loss of mesenteric fat, and development of SMA syndrome. Early recognition is important because delayed diagnosis can result in progressive malnutrition and worsening obstruction. When conservative treatment fails, duodenojejunostomy provides an effective means of bypassing the obstruction and restoring enteral nutrition.

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