

# Recurrent Pancreatitis in the Setting of Superior Mesenteric Artery Syndrome

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## Abstract

Various causes of Recurrent Acute pancreatitis have been identified in the past. This report presents a novel case of “recurrent” acute pancreatitis in the setting of superior mesenteric artery syndrome (SMAS). Thus, highlighting SMAS as a rare yet possible cause of recurrent pancreatitis facilitating early diagnosis and prompt treatment.

## Introduction

Superior mesenteric artery syndrome (SMAS) is a clinical condition that is characterized by compression of the third part of duodenum between the aorta posteriorly and the superior mesenteric artery (SMA) anteriorly. Von Rokitsansky described the syndrome for the first time in 1861. Later, in 1927, pathophysiology of SMAS was described in detail by Wilkie, giving this syndrome another name “Wilkie’s syndrome”.<sup>1</sup> This syndrome can present with very non-specific symptoms and thus making it difficult for prompt diagnosis and differentiating it from other conditions such as cholelithiasis, peptic ulcer disease, and abdominal angina. Therefore, the key to diagnose this condition is a high index of suspicion. In this report, we present an unusual yet possible case of “recurrent” acute pancreatitis as a result of superior mesenteric artery syndrome.

## Case presentation

A 45-year-old man presented to the hospital with a 24-hour history of worsening upper abdominal, bilateral flank pain and nausea without emesis. His past medical history consists of congestive heart failure, history of acute myocardial infarction, lower urinary tract symptoms, benign prostatic hyperplasia, vitiligo, congenital utero pelvic junction obstruction status post left pyeloplasty. He experienced similar symptoms months back when it resolved on its own. On presentation, he was hemodynamically stable with history significant for unintentional weight loss of 35lbs. The patient denied fever, chills or loose stools. Physical examination was significant for epigastric tenderness. Laboratory data demonstrated normal kidney function, glucose 100 (70-99 mg/dL), lipase 1552 (73-393U/L), unremarkable urinalysis, albumin 3.1 (3.4-5.4 g/dL) and calcium 8.3 (8.6-10.3 mg/dL). Diagnostic imaging included contrasted-computed tomography (CT) of the abdomen and pelvis, which showed inflammation in the tail of pancreas without necrosis. In addition, dilation of the second and third portion of the duodenum with an abrupt change in caliber (Figures 1-2) along with narrow aortomesenteric angle of 18° (Figure 3) was also observed. The patient was managed conservatively with intravenous fluids, intravenous morphine and nothing per mouth status. Gastric decompression was not performed because of incomplete obstruction. Diet was advanced gradually. Other potential causes of acute pancreatitis like gall bladder stones or biliary obstruction, alcohol or drug induced pancreatitis were ruled out. Ultrasound showed no gall stones or biliary obstruction. Based on the patient’s clinical presentation, laboratory and radiographic findings, he was diagnosed with SMAS -associated acute pancreatitis. Patient

was readmitted again two months later with a similar presentation. CT of the abdomen and pelvis showed acute pancreatitis complicated by splenic vein thrombosis with resolution of duodenal dilation. During the recurrent episode, he was again conservatively treated as well with subsequent excellent recovery.

## Discussion

SMAS is considered a rare but serious condition with an estimated incidence of 0.1-0.3%.<sup>2</sup> Though SMAS can occur at any age, it has been mostly reported in adolescent or young adults. Anatomically, SMA rises from the aorta at L1-2 level forming an angle with the aorta called aortomesenteric angle, normally 45–60°, while the third part of duodenum passes between the aorta and SMA at L3 level.<sup>3</sup> Normally, a retroperitoneal fat pad sitting at the angle between the aorta and SMA maintains adequate aortomesenteric angle, and thus prevents duodenal compression.<sup>3,4</sup> Hence, factors that result in a loss of retroperitoneal fat and a decrease of aortomesenteric angle and aortomesenteric distance can potentially cause compression of the third part of the duodenum, so called SMAS.

Factors that lead to an acute angulation of SMA can be either congenital or acquired. Congenital anomalies such as short Treitz ligament, malrotations and peritoneal adhesions have been described in association with this clinical condition.<sup>5,6</sup> On the other hand, most common risk factors that cause a decrease in aortomesenteric angle are acquired and related to a loss of retroperitoneal fat following severe weight loss. Causes of excessive weight loss include but not limited to eating disorders, cachexia, malabsorption, polytrauma, malignancy and acquired immunodeficiency syndrome, large extensive burns, and major surgeries.<sup>7</sup> Additionally, SMAS has been reported following orthopedic correction of spinal deformities, so called cast syndrome.<sup>8</sup> According to previous studies, pronounced lumbar lordosis in females makes them more prone to develop this condition particularly in the age range from 10-39 years.<sup>6</sup>

Furthermore, acute pancreatitis complicated by SMAS is an extremely rare presentation, and only very few cases, describing this association, have been reported in the literature.<sup>9,10,11</sup> This is the first report to describe SMAS in association with “recurrent” acute pancreatitis, to the best of our knowledge. In our patient, we believe that the recent excessive and rapid weight loss had resulted in a loss of retroperitoneal fat and, subsequently, a decrease in aortomesenteric angle, which lead ultimately to the duodenal compression. This was evident on the CT scan of the abdomen and pelvis, which was notable for the narrow aortomesenteric angle in addition to the compressed third part of duodenum leading to small bowel obstruction and dilation along with acute pancreatitis. The exact mechanism of pancreatitis in SMAS is still unknown, but it is proposed that the secondary occlusive postpapillary syndrome due to duodenal compression could cause reflux of bile into pancreas causing inflammation. On the other hand, inflammation of the pancreas could also cause paralytic ileus or peripancreatic edema thus reducing the aortomesenteric angle.<sup>9</sup>

Importantly, before SMAS diagnosis is made, other causes of GI obstruction should be ruled out, utilizing various imaging modalities. Traditionally, upper gastrointestinal barium swallow study has been used to diagnosis SMAS, which shows distended stomach and proximal duodenum along with abrupt vertical or oblique external compression of the third part of the duodenum.<sup>12</sup> Also, conventional mesenteric angiography with hypotonic duodenography used to be the most efficient diagnostic tool for this syndrome in the past.<sup>12</sup> Nevertheless, other noninvasive imaging modalities are more routinely utilized nowadays to diagnosis SMAS with high diagnostic accuracy including, multidetector CT scan, CT angiography and magnetic resonance angiography.<sup>4,13</sup> These imaging techniques can accurately demonstrate duodenal obstruction due to SMA compression along with a narrow aortomesenteric angle and distance seen in this syndrome.<sup>13,14,15</sup> Furthermore, in patients with proximal duodenum obstruction, an aortomesenteric angle of less than 22° along with an aortomesenteric distance less than 8mm on imaging corresponds strongly to SMAS.<sup>7,16</sup>

As it was shown in our patient, SMAS complicated by acute pancreatitis is usually conservatively treated by correcting electrolyte disturbances, gastroduodenal decompression and nutritional support, with an ultimate goal of weight gain to allow for retroperitoneal fat pad reinforcement.<sup>5</sup> Surgery is indicated for patients where conservative management fails.<sup>18</sup> Available surgical options include gastro jejunostomy, duodenojejunostomy and lysis of the ligament of Treitz with mobilization of the duodenum.<sup>19,20</sup> Duodenojejunostomy is the

preferred surgical approach due to lower rates of surgical complications in addition to lower failure rate.<sup>21</sup>

## Conclusion

Timely diagnosis and treatment of SMAS is pivotal to avoid serious complications such as fatal electrolyte abnormalities, gastric rupture, gut perforation, upper gastrointestinal bleeding, hypovolemic shock, oliguria, or sudden cardiovascular collapse.<sup>17</sup> Since recurrent pancreatitis due to SMAS is a rare condition, increased awareness of its existence to facilitate early diagnosis and prompt treatment that is paramount.

## Conflict of Interest

No conflict of interest to declare.

## Author Contribution Statement

Rajshri Joshi: Conceptualization, Investigation, Writing-Original draft, Methodology, Resources, Visualization, Supervision. Zafar Alzubi: Editing, Figure analysis, Correspondence. Mo'tasem Alkhayat: Figure analysis. Amar Gupta: Editing, Figure analysis. Daniel Mauro: Editing.

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