Case Report

Uncommon abdominal pain: A case report on spontaneous superior mesenteric artery dissection

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ABSTRACT

Spontaneous isolated superior mesenteric artery dissection (SISMAD) is a rare cause of abdominal pain, with symptoms ranging from incidental discovery to severe complications, such as acute bowel ischemia or a potentially fatal rupture of a superior mesenteric artery aneurysm. First reported in 1946, SISMAD is an uncommon vascular condition, accounting for <0.06% of acute abdominal pain cases. This case involves a 52-year-old male presenting with abdominal pain, diagnosed with SISMAD through imaging without evidence of bowel ischemia. Non-invasive imaging techniques, such as computed tomography angiography or magnetic resonance angiography, are crucial for diagnosing SISMAD. In this instance, a conservative treatment approach, including bowel rest and anticoagulation, was successful. The increased detection of SISMAD due to advanced imaging techniques necessitates its consideration when common causes of acute abdomen are excluded. Management strategies, in the absence of standardized guidelines, range from conservative care and anticoagulation to endovascular stenting or open surgical repair. Despite its rarity, SISMAD poses significant risks for morbidity and mortality.

Key words: Acute abdominal pain, Computed tomography angiography, Conservative management, Endovascular stenting, Superior mesenteric artery dissection

Spontaneous isolated superior mesenteric artery dissection (SISMAD) presents a notable diagnostic challenge in the emergency department (ED) due to its extreme rarity and diagnostic difficulty. First documented in 1946, SISMAD is an uncommon vascular disorder, with an estimated incidence of <0.06% among acute abdominal pain cases [1,2]. Typically occurring in individuals aged 50–70 years, SISMAD is more frequently observed in men than women [3]. Although factors, such as hypertension, connective tissue disorders, vasculitis, atherosclerosis, and aortic trauma can contribute to SISMAD, many cases arise without any identifiable risk factors [1,4]. Clinically, SISMAD can manifest in a spectrum of presentations, from asymptomatic incidental findings to severe outcomes, such as acute bowel ischemia or rupture of an aneurysmal superior mesenteric artery (SMA) [5]. Diagnosis relies heavily on non-invasive imaging techniques, such as computed tomography angiography (CTA) or magnetic resonance angiography (MRA), which can reveal characteristic features like a false lumen within the SMA, intramural hematoma, thrombosis, and possible pseudoaneurysm formation [6,7]. Treatment strategies for SISMAD vary based on the dissection's severity and the patient's symptoms, ranging from conservative management with anticoagulation to endovascular stenting or surgical intervention [8].

SISMAD is an exceptionally rare cause of acute abdominal pain, often under-recognized due to its non-specific presentation and lack of awareness. This case report aims to highlight the importance of advanced imaging techniques, such as CTA and MRA, in diagnosing SISMAD and discusses a successful conservative management approach in the absence of standardized guidelines. By raising awareness of this uncommon condition, we hope to improve diagnostic accuracy and inform management strategies to enhance patient outcomes.

CASE REPORT

A 58-year-old male presented to the ED with sudden epigastric abdominal pain persisting for 12 h. He had no fever, nausea, vomiting, or other gastrointestinal symptoms and denied any history of abdominal trauma or surgeries. The patient had a medical history of hypertension for 1 year.

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On examination, his blood pressure was 170/110 mm Hg, his heart rate was 96 beats/min, and his oxygen saturation was 95% on room air. His abdomen was soft and mildly tender with normal bowel sounds, and the rest of the physical exam was unremarkable.

A complete blood count revealed polycythemia with a peripheral blood smear showing normocytic and normochromic red blood cells (Table 1). To investigate potential underlying causes, a myeloproliferative neoplasm mutation panel was performed using the polymerase chain reaction method, including tests for the BCR/ABL fusion transcript, JAK2 V617F mutation, CALR gene mutations, and MPL mutations, all of which returned negative results. Serum erythropoietin level was measured at 7.05 mIU/mL, which was within the normal range.

Acute pancreatitis was initially suspected based on the patient's symptoms and physical findings, prompting a CTA of the abdomen. The CTA identified a dissection of the SMA without true lumen compromise and noticeable atherosclerotic changes in the abdominal aorta and other visceral branches.

Axial plain CT abdomen at the level of SMA shows significant fat stranding and cuffing was noted around the SMA (Fig. 1a). The SMA exhibited a prominent linear filling defect approximately 5 mm distal to its origin, causing 50–60% luminal occlusion and extending distally up to around 27 mm (Fig. 1b). 3D reconstructed images show dissected SMA (Fig. 2). An intimal flap originating from the thrombus area divided the distal SMA into two lumens, forming a double-barrel appearance. The anterior lumen, larger and continuous with the thrombus, represented the false lumen, while the smaller posterior lumen, well-attenuated, represented the true lumen (Fig. 3). Eccentric long segmental non-opacification was noted in the ileocolic and right colic branches on the right side and the jejunal branches on the left side, suggesting partial arterial thrombus of the false lumen. The false lumen was patent for a short segment before becoming thrombosed, while the true lumen appeared normal without significant compression. The aorta and its remaining visceral branches were normal, except for multifocal areas of atheromatous intimal calcifications in the aorta. There was no abnormal bowel wall thickening or bowel dilatation.

Table 1: Complete blood picture of patient

Test name	Result (%)
Hemoglobin	19.1 g/dL
Hematocrit	55.3
RBC count	5.94 million/uL
MCV	93.1fl
MCH	32.2 pg
MCHC	34.5 g/dL
RDW	13.0
Platelet count	165 10^3/mm^3
MPV	9.7 fl
PDW	16.2
TLC count	7.86 103/mm^3
Differential count	
Neutrophils	61
Lymphocytes	31
Monocytes	05
Eosinophils	03
Basophils	00

RBC: Red blood cell, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red cell distribution width, MPV: Mean platelet volume, PDW: Platelet distribution width, TLC: White blood cell

DISCUSSION

Isolated superior mesenteric artery dissection (SISMAD) is a rare condition with an unclear etiology. Yasuhara *et al*. reported in a study involving 22 patients with SISMAD that potential etiologic factors included atherosclerosis (18%), medial necrosis or degeneration (14%), fibromuscular dysplasia (5%), and idiopathic causes (63%), though the precise cause remains unknown [8]. Hypertension is prevalent among SISMAD patients, but a direct causal relationship has not been established [4,9]. SISMAD typically presents with sudden epigastric pain and may be accompanied by nausea, vomiting, melena, and abdominal distention. In some cases, patients may be asymptomatic, and the condition is discovered incidentally. The increase in reported

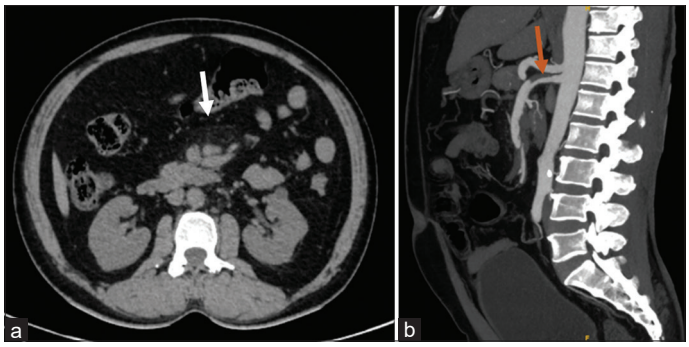

Figure 1: (a) Axial plain computed tomography abdomen at the level of a superior mesenteric artery (SMA) level shows subtle mesenteric fat stranding around SMA; (b) Sagittal reformatted images show linear filling defect approximately 5 mm distal to its origin, causing 50–60% luminal occlusion and extending distally

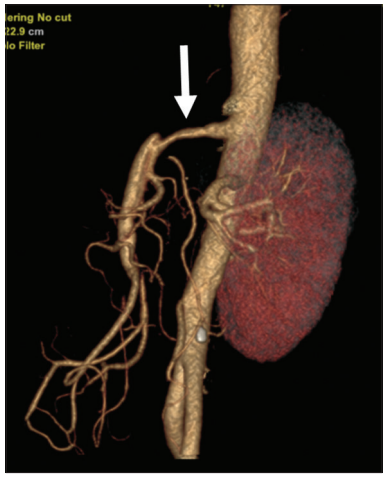

Figure 2: 3D reconstructed images show dissected superior mesenteric artery

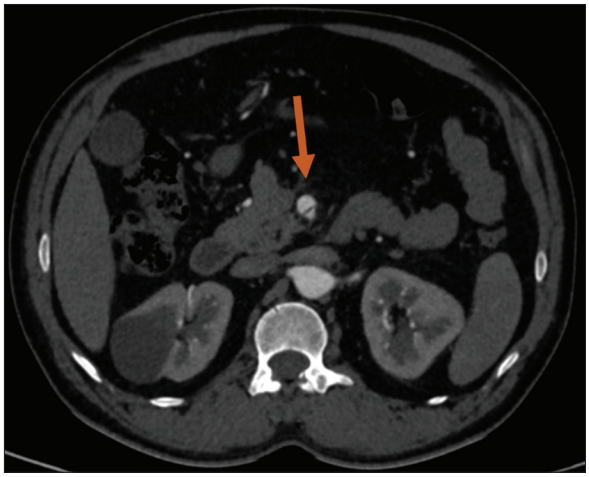

Figure 3: Axial contrast-enhanced computed tomography abdomen shows dissection of superior mesenteric artery into true and false lumen

cases over the years is likely due to the broader use and improved quality of CT imaging for undifferentiated abdominal pain [2,10].

The pathogenesis of SISMAD is not fully understood, but the dissection generally starts 1.5–3 cm from the SMA origin, avoiding the origin itself. The shearing forces between the fixed retropancreatic segment and the more mobile distal mesenteric segments cause a tear in the intima or primary hemorrhage in the media. This results in blood accumulating between the medial and adventitial layers or within the medial laminae, propagating the dissection through the artery. The dissection may be limited by thrombosis of the false lumen or extend to involve distal branches, narrow or obliterate the true lumen, or rupture through the adventitia [6]. Computed tomography (CT) angiography is effective in demonstrating the dissection flap, true and false lumens, and thrombosis. Sakamoto *et al*. and Yun *et al*. categorized SMA dissection into types: Type I (patent true and false lumina with entry and re-entry sites), type II (patent true lumen without re-entry flow from the false lumen), type IIa (visible false lumen without re-entry site), type IIb (no visible false luminal flow, causing true luminal narrowing), and type III (SMA dissection with SMA occlusion) [11,12].

Complications of SISMAD include bowel infarction, acute peritonitis, and late-stage pre-renal uremia. Rupture into the peritoneal cavity from an SMA aneurysm or dissecting artery can lead to intra-abdominal hemorrhage and hemorrhagic shock, necessitating close follow-up. In this patient, no signs of bowel ischemia were observed [10]. There is no consensus on the optimal treatment for SISMAD. Management options include conservative care, anticoagulation, endovascular stenting, and open surgery [5,8]. Surgical intervention with endovascular stent placement is recommended for patients at risk of bowel ischemia or those with aneurysmal enlargement or rupture. This patient was managed conservatively with anticoagulants, resulting in symptomatic improvement.

Isolated SISMAD is a rare vascular condition with an unclear etiology. The rarity of SISMAD has resulted in limited awareness among clinicians, which contributes to diagnostic challenges. However, several case reports have added valuable insights into its presentation, diagnosis, and management. A recent case report by Takach *et al*. described a patient with SISMAD presenting with acute abdominal pain, successfully managed conservatively with anticoagulation, similar to our case. The report underscored the role of non-invasive imaging techniques, such as CTA, in timely diagnosis and management decisions [13]. Another case by Park *et al*. highlighted the variability in clinical presentations, ranging from asymptomatic findings to severe complications like bowel ischemia, advocating for the inclusion of SISMAD in differential diagnoses for acute abdomen when other causes have been excluded [14]. In addition, a case report by Joo *et al*. detailed a patient with symptomatic SISMAD managed with endovascular stenting due to progressive symptoms and risk of ischemia, illustrating that management strategy must be tailored to the severity of symptoms and the patient's overall condition [15]. Such reports emphasize the diverse presentations and outcomes associated with SISMAD and reinforce the need for heightened clinical suspicion and individualized management approaches.

In our case, the use of advanced imaging modalities allowed for the accurate diagnosis of SISMAD and the patient was managed conservatively with anticoagulation, resulting in a favorable outcome. This aligns with reports suggesting that conservative management can be effective in stable patients without signs of bowel ischemia [13,14]. However, in cases where there is evidence of ischemia or aneurysmal complications, more invasive strategies, such as endovascular repair, may be required [15].

CONCLUSION

Isolated spontaneous dissection of the SMA, without aortic involvement, is a rare and potentially fatal condition, with limited cases reported in the literature. CT angiography is essential for prompt detection and management. Conservative management with anticoagulants can be a viable option, especially for stable patients without evidence of bowel ischemia.

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