

Superior Mesenteric Artery Thrombosis and Partial Dissection Managed Medically

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Abstract A 44-year old male presented to the ED complaining of abdominal pain. CT angiography of the abdomen showed thrombus extend from the proximal superior mesenteric artery (3 cm distal to the origin), with partial dissection of the superior mesenteric artery. He was given intravenous fluid and was started on intravenous heparin. All hypercoagulable workup was done to rule out the causes of thrombosis or dissection was negative. The patient's abdominal pain was decreasing, and heparin was bridged with Coumadin. The patient was discharged to home, and advised to check INR regularly and follow up with the vascular surgery department. Conservative management is generally the preferred treatment. For more serious cases, aggressive approaches such as percutaneous endovascular stent placement or surgery would be considered.

Keywords: vascular, surgery, superior mesenteric artery, thrombosis, dissection

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1. Introduction

Acute superior mesenteric artery thrombosis (AMAT) is a rare disease which commonly occurs secondary to trauma, atherosclerotic disease, or cardiogenic source [4]. It can cause acute mesenteric ischemia (AMI), which is a life-threatening with mortality rates as high as 93% [6]. In 10% of cases, the cause of AMI is AMAT [6,8].

Spontaneous isolated superior mesenteric arterv dissection (SIDSMA) is a superior mesenteric artery dissection without aortic dissection [9,13,15]. First reported by Bauersfelt, this disease has been reported as a rare cause of acute abdominal pain [7]. The reported cases of SIDSMA in an autopsy series was 0.06% [11]. Diagnostic imaging studies such as multi-detector row computed tomography, ultra sounds, MRI, CTA, and reconstruction imaging have placed the spotlight on SIDSMA in recent years [10,13,15]. CT imaging is usually capable of displaying the thrombosis and disintegration of the false lumen, intramural hematoma, and dissecting aneurysm over time [13,15]. SIDSMA occurs mainly in male patients (91.7%) in their fifties (median age is 57 years), and 52.9% of them have hypertension [11,13,15]. Potential risk factors include hypertension, smoking, hyperlipidemia, coronary heart disease, atherosclerosis, diabetes mellitus, and trauma [19]. The main symptom of SIDSMA is abdominal pain of acute onset, and major complications include arterial rupture with bleeding and bowel infarction [12,15,16]. The natural course and ideal management is yet to be established for this rare but potentially fatal disease [6,9].

2. Case Presentation

This is a case of a 44 year old male who presented to the ED with complaints of abdominal pain. He noted his pain to be located in his left upper quadrant and radiating to the epigastrium. He characterized the pain as sudden, intense, and progressive; associated with nausea but no vomiting. He denied any fever, chills, diaphoresis, shortness of breath, or chest pain. He felt lightheaded after the onset of pain but did not lose consciousness. He also denied any recent melena, hematochezia, and recent NSAID use.

His past surgical history was significant for laproscopic sleeve gastrectomy and hernia repair performed 11 months prior for his morbid obesity. His other medical history included OSA. His only home medication included was sildenafil for erectile dysfunction. He denied any history of using tobacco products or recreational drugs, and drank alcohol occasionally.

At presentation, his blood pressure was 120/82, heart rate of 78 beats per minute, respiratory rate of 16 beats per minute, and body temperature of 98 fahrenheit. His abdomen was soft, though he expressed diffuse tenderness, which was more pronounced in his left upper quadrant. However, there was no rebound tenderness or rigidity. Bowel sounds were normoactive, and the rest of his physical examination was within normal limits.

His labs on admission showed no leukocytosis, and amylase, lipase, lactic acid level all were normal with no basic metabolic profile abnormality. Computed tomography scan of abdomen and pelvis with IV contrast showed Proximal Superior Mesenteric Artery Thromboses (e.g. Figure 1).

The patient was initially treated with famotidine, intravenous morphine, and normal saline. A11 hypercoaguable blood tests were found to be negative. The patient was started on unfractionated heparin. Serial abdominal examination was done to monitor the patient's abdominal pain for acute abdomen and bowel ischemia. Once the patient did display worsening syptoms, he was started on enteral feeding as tolerated. A transesophageal echography was performed and showed no evidence of emboli. On the third day, repeated CT angiography of the abdomen and pelvis showed thrombus extend from the proximal superior mesenteric artery (3 cm distal to the origin), with partial dissection of the superior mesenteric artery (e.g. Figure 1).



Figure 1. CT angiography of thrombus within the superior mesenteric artery and dissection flap

However, the patient's abdominal pain decreased significantly, and he was tolerating oral diet. No surgical intervention was done. Warfarin was bridged with heparin, and then heparin was discontinued after reaching the target INR of 2-3. Implantable recorder did not detect any arrhythmia and concluded that the thrombus was not cardiac in origin. The patient was discharged to home, with continuous follow up of INR. Three months later, repeated CT angiography showed thrombus had resolved, with no signs of bowel ischemia or bowel necrosis. Symptoms had not recurred in this period, however dissection had persisted.

3. Discussion

Acute mesenteric ischemia is a condition in which mortality may reach from 64 to 93% if not diagnosed early [6]. Various conditions leading to ischemia include: heritable thrombophilia, acquired thrombophilia, cardiac arrhythmia, congestive heart failure, recent myocardial infraction, advanced atherosclerosis, hypotension, sepsis, intraabdominal pathology or idiopathic [2,6]. There are two types of types of mesenteric ischemia: venous and arterial. Mesenteric venous thrombosis is more common than arterial [6]. Of the peripheral arteries, the superior mesenteric artery is the second most common artery [16]. Acute thrombosis of the superior mesenteric artery (SMA) can lead to intestinal infarction and is associated with a mortality rate of around 65% [3,6,7].

Causes of mesenteric artery occlusion may be secondary to thrombi and/or abnormal cardiac activity, including atrial fibrillation, cardiac arrhythmia, and myocardial infarction [4]. Laproscopic sleeve gastrectomy has been identified as one of the possible cause of portomesenteric venous thrombosis but its association with SMA thrombosis or dissection has not been established [17]. Bariatric surgery patients can be susceptible to thromobotic events because of potential hypercoaguable state. The hypercoaguability is likely because of increased release of coagulation factors and fibrinogen associated with metabolic syndrome [18].

Clinical presentation and duration of symptoms help to differentiate the management of the disease. However, it may be difficult with elderly patients, since their symptoms can be masked or not clearly present. Biochemical examination and imaging are important tools in establishing a proper diagnosis of AMI by identifying signs of elevated WBC, amylase, LDH, etc. [3].

Plain X-ray of the abdomen may show thickened bowel and ground glass appearance, but it is only significant when the cause of the abdominal pain is bowel perforation [3]. Ultrasound may be useful in assessing intestinal viability, but it is difficult to assess the whole intestine in a short amount of time [3]. In most settings, CT angiography is the preferred choice when diagnosing AMI (sensibility 0,96, specificity 0,94) [5, 8]. A normal CT exam may show a thick intestinal wall, internal hematoma, enlarged intestines with liquid, airy portal vein, and other nearby obstructions inside the abdomen. SMA thrombosis usually develops at or near the ostium, with atherosclerosis in the background [8].

Treatment of a disease depends on its severity. Options for acute mesenteric ischemia include: anti-platelet agents, anticoagulation, intravenous fluid, optimizing blood pressure and bowel rest [2,4]. Other modalities are plain or mechanical aspiration, percutaneous endovascular reconstruction with bare stent, and surgery [14]. Thrombolytic therapy and plain or mechanical aspiration can only be performed in the early stage of the disease for patients with short vascular occlusions and who do not have clinical evidence of bowel necrosis [2,3,6]. If there is clinical suspicion or evidence of bowel necrosis or upcoming arterial rupture, then surgery is preferred [2,3,4]. Additionally, it is important to note whether the patient is hemodynamically stable or not, and responding to conservative management [5,6].

Spontaneous dissection of peripheral arteries is rare [15,16]. Arteriosclerosis, fibromuscular dysplasia, and cystic medial necrosis may be causes of SIDSMA. For treating SIDSMA, there is no consensus on the best treatment modality. Treatment options have not been uniform, ranging from conservative therapy to endovascular stenting or open surgical revascularization [6,13,14,16]. The non-surgical treatment options for SIDSMA-including conversion or anticoagulation therapy-have been successful and used to achieve normal flow of the SMA. However, there is no consensus on the duration of anticoagulation and/or antiplatelet therapy [1,9]. Surgery is further classified into many categories depending upon various complications and presentation [3]. Surgical techniques include aortomesenteric bypass, thrombo-intimectomy with optional angioplasty, endo-aneurysmorrhaphy, patch SMA interposition and right gastro-epiploic artery-to-SMA bypass [11]. Some studies concluded that stent placement in the SMA is a safe and feasible therapeutic option, but its long-term results are still undetermined [9,13,15,16]. In

addition, conservative treatment has also been reported to have favorable outcomes—especially for patients without signs of acute bowel ischaemia or SMA rupture [8,16].

Conservative management is the preferred approach in these types of cases, including bowel rest to reduce need for mesenteric blood flow, intravenous fluids, and proper nutrition [8,11]. Usually abdominal pain diminishes within a week with conservative therapy [11]. Aggressive approaches such as percutaneous endovascular stent placement or surgery would be considered for patients with ruptured arteries, continuous abdominal pain, or aneurysmal false lumen dilation [4,5,9]. The goal of these interventions was to prevent further extension of dissection and to increase blood flow into the intestine by annihilating the false lumen [4]. Those who had undergone conservative management should undergo follow-up CT scan in 15 days, 1 month, 6 months, and yearly. An additional CT scan can be done if there is a progression of symptoms, but this no strict criteria for surveillance, as it varies from case to case. Patients who undergo endovascular stent or surgery also need imaging surveillance [13].

4. Conclusion

This is a case of superior mesenteric artery (SMA) thrombosis with partial dissection. All hypercoagulable work was done to rule out the causes of thrombosis or dissection, which were negative. The patient was discharged home, and advised for strict follow-up and repeated imaging. Three months later, repeated CT angiography showed thrombus had resolved, with no signs of bowel ischemia or bowel necrosis. Conservative management is the best approach if there is no evidence of bowel ischemia or necrosis, arterial rupture, or extension of dissection. There is always a risk of stenosis in stent in the long term after endovascular stent placement.

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