

Case Report

A rare cause of acute abdominal pain: spontaneous isolated superior mesenteric artery dissection

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Received: 07 May 2018

Accepted: 31 May 2018

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ABSTRACT

Spontaneous isolated superior mesenteric artery dissection is an extremely rare nosological entity, usually occurring with acute abdominal pain. Authors present the case of a 56 - year - old female with spontaneous isolated SMA dissection who was admitted to the hospital with epigastric pain of acute onset. The patient was successfully managed nonoperatively, with anticoagulation starting immediately after diagnosis. Patient symptoms resolved after a few days. There is a discrepancy concerning the treatment of isolated SMA dissection. Generally, if there is no intestinal necrosis or SMA rupture, conservative treatment is safe and effective as an initial approach.

Keywords: Abdominal pain, Dissection, Superior mesenteric artery

INTRODUCTION

Spontaneous isolated superior mesenteric artery dissection (SMA), without concomitant aortic dissection, is an infrequent nosological entity, with an incidence of 0.06% in a series of 6666 autopsies.¹

There is a wide spectrum of clinical presentation of the disease: in some cases it is completely asymptomatic and is detected incidentally during abdominal imaging for other causes, while in other cases it may be the cause of acute abdomen due to bowel necrosis or even the cause of hemorrhagic shock due to arterial rupture. Because of the limited number of cases reported in the literature, the etiology and natural course of the disease are not fully clarified.² For the same reason, there are no guidelines on the management of symptomatic dissection of the SMA.

Herein, we report a case of isolated spontaneous dissection of the SMA, treated conservatively.

CASE REPORT

A 56 - year - old female with a 24 - hour history of epigastric pain of acute onset and vomiting, presented to the emergency department. There were no symptoms of fever, constipation or diarrhea. There was no history of trauma. On admission her blood pressure was normal. Her past medical history was significant for obesity, osteoporosis and cigarette smoking.

On physical examination the abdomen was soft, but there was mild tenderness to deep palpation over the epigastrium. No bruit was audible. No rebound tenderness was present. Laboratory studies showed only

mild leukocytosis (12,080/ μ l) and all other data were within the normal range.

An abdominal color doppler examination revealed a superior mesenteric artery of 10 mm in diameter with a double lumen. Afterwards, we performed a contrast enhanced computed tomography (CT) which confirmed the presence of an isolated dissection of the SMA (Figure 1) beginning at 24 mm from its origin and extending distally for 28 mm. The true lumen was slightly compressed. The CT scan revealed no signs of bowel ischemia.



Figure 1: CT scan shows isolated dissection of the SMA.

The patient underwent conservative treatment consisting of low molecular weight heparin (LMWH) (therapeutic dose), intravenous fluid administration with bowel rest until the pain resolved. Nasogastric suction was also used in combination with strict blood pressure control. The abdominal pain completely resolved after three days and acenocoumarol was administered in conjunction with LMWH. Once therapeutic range of International Normalized Ratio (INR) was obtained, LMWH was discontinued.

Follow - up CT scans revealed a restoration of the caliber of the SMA main trunk with significant improvement of the true lumen narrowing. The patient discharged from hospital 10 days later in good general condition and with full commencement of oral food intake. Acenocoumarol was discontinued at 3 months. The patient continues to be in good condition after 41 months from the initial event.

DISCUSSION

Spontaneous dissection of peripheral arteries is a rare condition. The internal carotid artery is the more frequent

spontaneously dissected peripheral artery, followed by the SMA.³ Yun et al. reported a male predilection of the disease in their fifth decade.²

The etiology of the disease is largely unknown. Several authors support that atherosclerosis, fibromuscular dysplasia, cystic medial necrosis, connective tissue disorders, arteritides, trauma and pregnancy may play a role.⁴ Some others report smoking and hypertension as risk factors, although in a recent series of 32 patients with spontaneous SMA dissection only a third of these patients were hypertensive or smokers.^{2,4}

SMA dissection may be completely asymptomatic, especially when there is limited progression, with thrombosis of the false lumen. In other clinical presentation include abdominal pain of acute onset, intestinal angina and weight loss because of chronic dissection and a compromised true lumen.⁵ Progression of the dissection to distal branches of the SMA or expansion of the false lumen, at the expense of the true one, can be the cause of intestinal necrosis. Severe compression or even complete obstruction of the SMA main trunk does not always coincide with intestinal necrosis due to the collateral blood supply from the marginal artery of Drummond.⁶ Rupture of the dissected SMA is one of the most feared complications, possibly leading to hemorrhagic shock and even death.

Due to the rarity of the disease, a high index of suspicion is needed for a prompt diagnosis and treatment. Computed tomography angiography (CTA) is the most precious diagnostic tool in order to confirm the clinical suspicion, locate the entry point and evaluate the extension of the dissection. CTA aims to decide on the treatment modality, conservative or operative. It is noteworthy that in most cases the entry point is located within 1,5 - 3 cm from the origin of the SMA.⁷ This is the zone where the relatively fixed retropancreatic SMA becomes mobile at the mesenteric root.⁷ Hemorheologic factors and forces applied in this zone may play a role. The dissection can sometimes extend proximally.⁸

Sakamoto et al. and subsequently Yun et al. classified the morphology of isolated SMA dissection based on angiographic appearance.^{2,9} So, currently there are 3 types of spontaneous isolated SMA dissection. In the type I, both false and true lumens are patent with entry and re - entry points. In the type IIa, both lumens are patent without re - entry point, while in the type IIb, the false lumen is thrombosed, but the true one is patent and narrowed. Finally, in the type III there is total occlusion of the dissected SMA.²

It seems that type II lesions are more frequent than the others. On the other hand, type I lesions are more common in patients with incidentally discovered dissections.² There is some evidence that the length of the dissection is correlated with the presence and intensity of symptoms. The dissected SMA triggers an inflammatory

periarterial response which causes visceral nerve plexus irritation.¹⁰ This irritation is the cause of abdominal pain which is a common finding in symptomatic patients. Consequently, long dissections are associated with more intense inflammatory response and as a result more severe pain. Obviously, the intense pain presented in a long dissection is not only a result of the inflammation but may also be the result of obstruction of distal branches, causing bowel ischemia.

From a histopathological point of view scarce elements are known, such as cystic medial necrosis or myxoid degeneration of the dissected SMA wall.^{11,12}

Taking into account the rarity of the disease there are not guidelines for the treatment of these patients. According to the clinical and CTA findings a different treatment modality may be followed. In case of absence of malperfusion of the bowel or when rupture seems unlikely, conservative treatment must be followed. There is currently no consensus as to the use of anticoagulants or antiplatelets in patients undergoing conservative treatment. Some authors raise doubts over the use of any of these drugs.^{13,14} On the other hand, others suggest immediate administration of anticoagulants in order to prevent thrombosis of the true lumen. The mainstay of conservative treatment is bowel rest, in association with nasogastric suction, intravenous fluid administration and nutritional support in case of prolonged fasting. Duration of conservative therapy is guided by symptom relief. When the patient becomes completely asymptomatic, oral intake could start gradually. If during conservative therapy a deterioration of patient symptoms is noted, there must be a turn into operative approach. Operative treatment is indicated in case of acute dissection with malperfusion or chronic dissection with recurrent symptoms. Park et al. advocate for abandonment of conservative therapy in the case of persisting abdominal pain for more than 7 days.¹¹ Additionally, in case of continuous expansion of the false lumen or in the rare occurrence of rupture, operative therapy is mandatory. If operative management is decided, endovascular approach seems to be a safe and valuable option. Stent placement and thrombolytic therapy could be useful especially in patients at high risk for open surgery.^{3,15} The aim of stent placement is to cover the intimal tear and interrupt the blood inflow to the false lumen. A self - expandable stent is adequate in the majority of the cases. Obviously, endovascular repair can be administered in case of failure of the conservative treatment. Open surgery may play a role especially when endovascular treatment is not feasible, with higher morbidity and mortality. Several procedures can be done, such as aortomesenteric or iliomesenteric revascularization or thromboectomy with patch closure.⁸ In case of intestinal necrosis and perforation there is no place for conservative or even endovascular treatment. Open surgery is undoubtedly necessary. In the literature, conservative treatment comprises the most common initial therapeutic approach, with a low failure rate. In a recent review 172 out of 219

patients with SMA dissection, were treated conservatively. The failure rate of this treatment modality was 6,8%. These failures were treated successfully by endovascular means.¹⁶

Follow-up is another question which is largely subjective. It is commonplace that a close lifelong monitoring is mandatory in case of both conservative and operative treatment. The frequency of this monitoring is based upon recommendations of some authors and of course does not comprise guidelines. Jia et al. in a recent series of 17 patients recommend close follow-up through a CT scan, first at 15 days, then at 1 and 6 months and yearly thereafter in case of conservative treatment.¹⁴ If endovascular approach was adopted Jia recommends follow-up CTs at 1 and 6 months and then yearly.¹⁴ CT or duplex scanning with the same frequency are also recommended by the latter in all cases treated successfully as a lifelong surveillance.¹⁴

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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Cite this article as: Galanopoulos G, Konstantopoulos TP, Christakis IA, Antonopoulos PA, Papavassiliou VG. A rare cause of acute abdominal pain: spontaneous isolated superior mesenteric artery dissection. *Int Surg J* 2018;5:2623-6.