

TRANSIENT ISCHEMIC JEJUNITIS DUE TO SYMPTOMATIC ISOLATED SUPERIOR MESENTERIC ARTERY DISSECTION: CASE REPORT AND REVIEW OF LITERATURE

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Abstract

Acute abdominal pain is one of the most common conditions encountered in the emergency department. The differential diagnosis of acute abdominal pain is extensive and identifying the underlying etiology can be challenging. We report a case of acute transient ischemic jejunitis due to symptomatic isolated superior mesenteric artery dissection in a patient with no cardiovascular risk factors or autoimmune diseases. Symptomatic isolated superior mesenteric artery dissection is a rare cause of acute abdominal pain usually treated in the surgical department. The patient had criteria for conservative treatment and rapidly recovered. We highlight a rare condition which should be taken into account for the differential diagnosis of acute abdominal pain.

Keywords: acute abdominal pain, transient ischemic jejunitis, symptomatic isolated superior mesenteric artery dissection, conservative treatment

Introduction

Acute abdominal pain is one of the most common conditions encountered in the emergency department (ED). The clinical diagnosis of acute abdominal pain is unreliable, resulting in both negative laparotomies, as well as ill-advised surgical delay in a large number of patients. New and emerging imaging techniques offer a non-invasive way to decrease both false-negative and false-positive diagnoses in this category of patients [1].

Usually ischemic enteritis is a diagnosis of surgical nature rarely cited in the literature. Symptomatic isolated superior mesenteric artery dissection (SISMAD) may cause segmental ischemic enteritis. Until recently SISMAD in the absence of abdominal

aorta dissection has been considered an exceptionally rare pathology, with only 26 cases published before 2000 [2]. In such cases modern diagnostic imaging techniques can identify SISMAD, which seems to be more frequent than previously thought and has gained more space in medical literature.

Case Report

A 57-year-old male patient, with no medical history or cardiovascular risk factors presented to the Emergency Department (ED) in December 2015 with moderate (on Verbal Rating Scale) diffuse abdominal pain, repeated vomiting and two non-bloody watery stools. The clinical complaints started 72 hours prior to presentation, after a heavy meal.

The physical examination revealed blood pressure of 130/70 mmHg, heart rate of 80, respiration rate 18,

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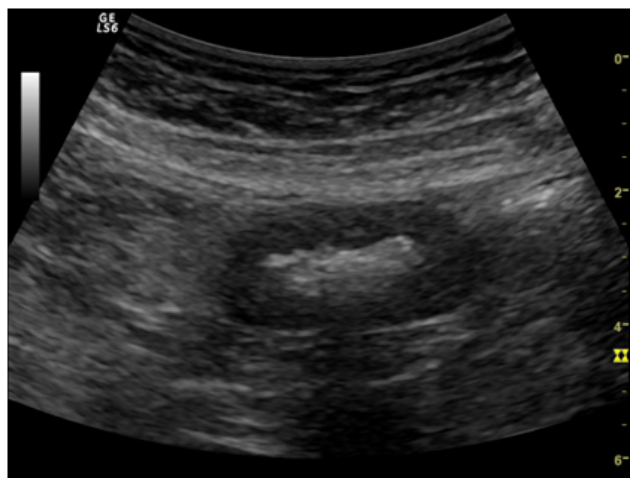
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temperature 36.6°C. The abdomen was slightly distended with epigastric and left middle quadrant tenderness without peritonitic signs.

Blood test showed inflammatory syndrome (CRP 11.98 mg/dl, ESR 65 mm/h, Fbg 759.1 mg/dl), the white blood cell count was $8.1 \times 10^9/L$, hemoglobin was 14 g/dl, neutrophils were at 92%, and amylase/lipase/lactate/liver function tests were within normal limits. Thus, in this case of a middle-age man suffering from moderate abdominal pain, with no fever or leukocytosis the Current Guidelines

for Diagnosis and Management of Abdominal Pain were applied, and further investigation was guided using American College Radiology Appropriateness Criteria [3].

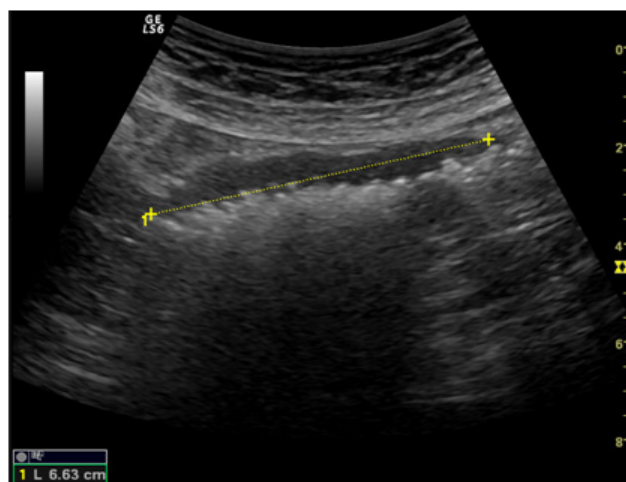
The plain abdominal X-ray was not modified. Ultrasound of the abdomen (GE Logiq S6 with convex transducers of 3.5–6 MHz and linear ones of 7–12 MHz) revealed a jejunal wall thickening of 7.5 mm, with a length of 6.6 cm (Figures 1 A, B, C) and preserved bowel movements. Around the thickened wall mesenteric inflammatory lymph nodes were identified.



A



B



C

Figure 1. Abdominal ultrasound: circumferential thickening of the small bowel wall (A), maximum thickness 7.5 mm (B), length 6.6 cm (C)

The color duplex sonography examining abdominal vascularization showed a superior mesenteric artery (SMA) with normal root and physiological flux Vmax 0.86m/sec. There were no turbulent areas or dissection images. The abdominal aorta was not dilated. The thickened jejunal wall was well vascularized.

The acute onset of the clinical complaints and the ultrasound findings were suggestive for an acute enterocolitis of infectious or inflammatory origin. Stool test for bacterial infections (Salmonella, Shigella) and parasites were negative. Tumoral markers (CEA, CA19-9) were

within physiological range. The fecal occult blood test was positive. In order to exclude a neoplasia or an ischemic process contrast-enhanced computed tomography (CT) was ordered. Contrast-enhanced CT scan confirmed the small bowel circumferential thickening with no free fluid or gas and a normal appendix. CT scan also showed a dissection of the SMA starting at approximately 1.8 cm from root and extending to the origin of the first branches; three of the jejuno-ileal branches and the right colic arteries had a contrast filling defect and were slightly dilated suggesting the presence of intraluminal thrombosis (Figure 2).

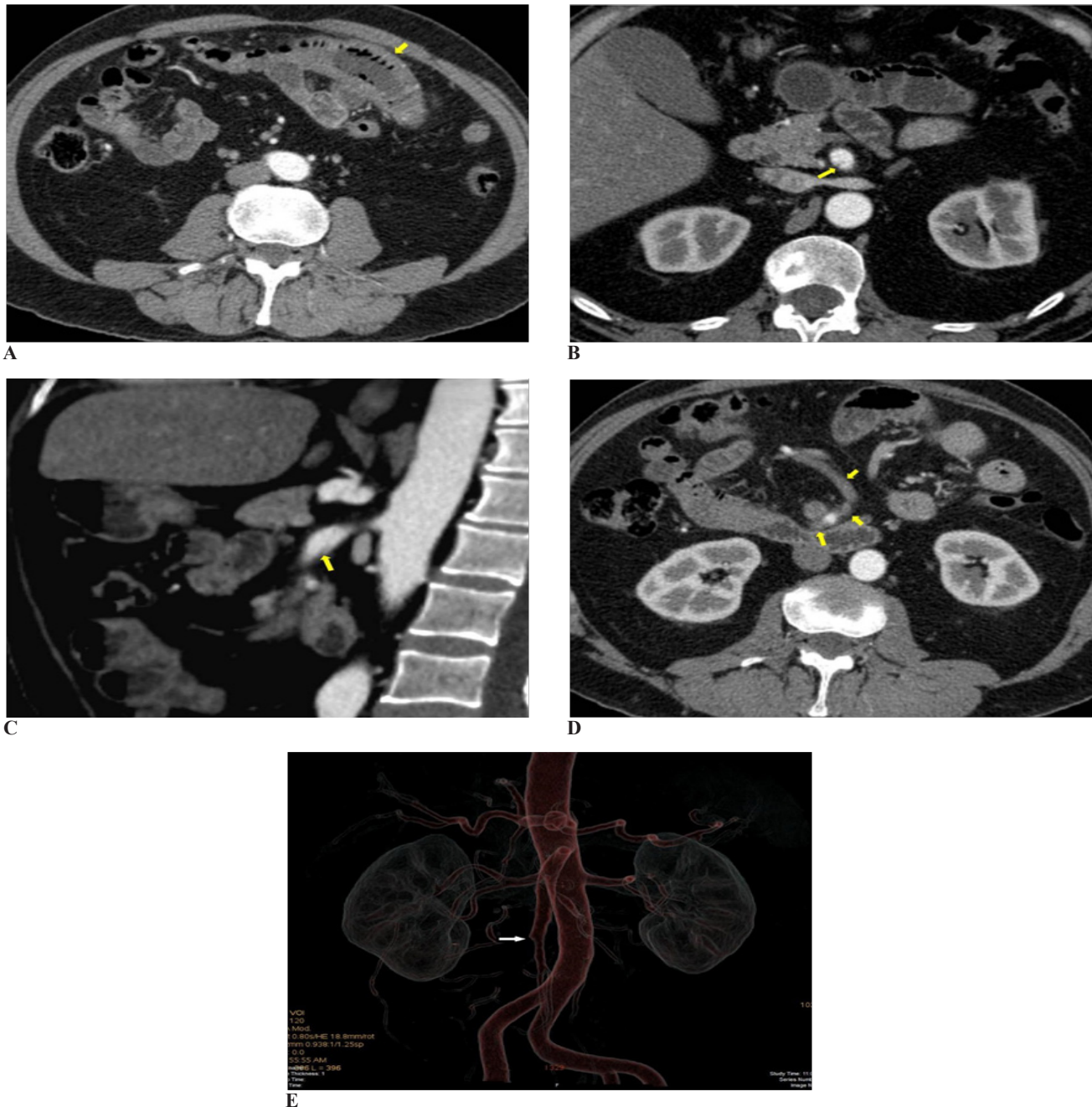


Figure 2. Contrast-enhanced CT scan of abdomen and pelvis: A – the thickening of jejunal walls; B – dissection fold of SMA, axial section; C – dissection fold of SMA, sagittal section; D – thrombosis of the ileo-colic and jejunal arterial branches of SMA; E – vascular reconstruction, the right branch of SMA is missing.

We established the diagnosis of segmental ischemic jejunitis due to SISMAD. In the absence of peritonitis, we decided for a conservative management consisting of bowel rest with fasting until abdominal pain relief, surveillance of blood pressure, and antiplatelet therapy (Aspirin 75 mg/day). Given the segmental enteritis we associated a short term treatment with Rifaximin (2x200 mg/day) and 5-ASA derivate (Sulfasalazine 2x1g/day).

The patient's evolution was carefully monitored clinically, biologically and by daily abdominal ultrasound scan. During the next couple of days, the abdominal pain was relieved and endovascular or surgical treatment was postponed. The antibiotic and 5-ASA treatment were discontinued after 7 days. Abdominal ultrasound scan revealed the decrease of the jejunal wall thickness and physiological arterial flux in SMA.

After 12 days of conservative therapy the leucocytes dropped to $4.1 \times 10^9/L$ with 70% neutrophils and inflammatory syndrome (CRP=0.53 mg/dl, ESR=43 mm/h, Fbg=552.7 mg/dl) decreased significantly, occult stool blood test was negative. A month later CT angiography (CTA) was performed showing a permeable SMA until bifurcation with a non-permeable right branch (Fig 3). The walls of the small bowel were thin with no ischemic or inflammatory signs. The antiplatelet therapy be continued for 1 year.



Figure 3. CTA of abdomen and pelvis with vascular reconstruction: the right branch of SMA without contrast-enhanced.

Discussion

Our case highlights the importance of correct clinical and imagistic evaluation of abdominal pain in emergency. Ischemic segmental enteritis could not have been diagnosed in the absence of abdominal ultrasound evaluation and SISMAD could not have been identified without contrast-enhanced CT scan to visualize the vascular pathology.

So both the accurate diagnosis and correct therapeutic intervention depend on the imaging techniques performed in the early stages of acute abdominal pain.

The dissection of SMA associated with dissecting aortic aneurysm was described for the first time in 1947 by Bauersfeld in autopsy reports [4]. Since then, new reports focusing on epidemiology, risk factors, clinical characteristics and treatment of this special entity have been published [4–8] mostly for Asiatic population.

If in 2013, Luan et al. identified 296 published cases of SISMAD all over the world [5], three years later 622 cases were found in China only. From these reports we have learned that SISMAD most commonly occurs in middle aged (mean age 55.4 years, ranges from 30 to 80 years) males (88%) presenting in emergency for the sudden onset of abdominal pain [7].

No specific underlying cause of SISMAD was identified in the majority of reports, even though some authors suggested arterial wall pathology such as cystic medial necrosis, arterial mediolysis [9] or adventitial inflammation, disruption of the internal elastic lamina [10] might cause SISMAD. Park et al. hypothesized that SISMAD develops due to abnormal hemodynamic force caused by convex curvature of the SMA. A computer simulation model identified abnormal mechanical stresses on the anterior wall of the SMA, in the transition zone from a fixed to a relatively mobile portion of the artery [11].

The abdominal pain resembles the one found in intestinal ischemia (acute or chronic), but in the absence of cardiovascular risk factors. Most clinicians would assume that persistent abdominal pain is a result of bowel ischemia. Yun has suggested that the distention of periarterial nerve fibers induces pain, independently of bowel ischemia. A direct correlation between severity of pain and length of dissection has been observed [12]. In our case, marked improvement of clinical symptoms simultaneous to the reduction of jejunal wall thickness incriminates the bowel ischemia as the cause of abdominal pain.

In the emergency department, ultrasound imaging is important to establish the etiology of abdominal pain. In our case the ultrasound revealed a thickened small bowel wall with preserved color-flow Doppler signals which were suggestive for an acute enteritis. The differential diagnosis included neoplasia and ischemic bowel disease. The ultrasound examination of the ischemic bowel reveals a thickened wall and the absence of color-flow Doppler signals, and a dilated lumen. High velocity flow in SMA indicates significant stenosis [13,14]. Doppler ultrasound can demonstrate a proximal occlusion in the main superior mesenteric artery or vein, but is limited in assessing branch vessels, particularly in the presence of distended, gas filled and/or aperistaltic bowel loops [15]. Acute infectious enteritis, on the other hand, rarely requires diagnostic imaging, but if performed, the ultrasound findings demonstrate a thickened small bowel wall, with preserved

stratification, increased peristalsis and hypervascularity on color Doppler imaging [13]. The wall thickening associates enlarged regional mesenteric nodes, different from mesenteric inflammatory changes seen in Crohn's Disease [15]. As for the possible diagnosis of neoplasia, the great majority of the small bowel tumors are localized in the duodenum and terminal ileum; they are characterized by loss of stratification and sometimes dilatation of the intestine in the affected area [16].

Although ultrasound imaging is a powerful tool to diagnose acute mesenteric ischemia, contrast-enhanced CT is the investigation of choice for SISMAD. Characteristic CT findings include thrombosis of the false lumen, intramural hematoma, and/or intimal flap [6]. In our case, contrast enhanced CT and CTA were concordant showing a dissection of the SMA from the root to the origin of the first branches associating thrombosis of the three jejuno-ileal branches and of the right colic arteries. Arteriography is recommended only when endovascular treatment is required [5].

How to determine which symptomatic patient can be treated conservatively and which requires intervention? To ease the therapeutic decision two classifications of SISMAD: Yun [12] and Luan [7] and a therapeutic algorithm were proposed [17]. These classifications and algorithms are based on clinical and imagistic findings.

Thus, conservative treatment is recommended in the absence of rebound or peritoneal signs. The aim of treatment for SISMAD is to limit the progression of the dissection, to prevent the rupture of the false lumen, and to preserve the distal blood perfusion through the true lumen [8]. The conservative measures include fasting, parenteral nutrition, blood pressure control, and antiplatelet therapy [18]. In terms of anticoagulation therapy in SISMAD, some authors have advocated its use [19,20] to prevent thrombus propagation, while others have not [12,21]. There has been no firm evidence to support anticoagulation therapy in SISMAD. Anticoagulation can prevent false lumen thrombosis at the dissected SMA, and thus promote further propagation of dissection. Moreover, Yun et al showed no significant difference between the patients who received anticoagulants and those who didn't, in terms of treatment outcomes [12]. Thus, we decided to adopt a conservative treatment based on antiplatelet therapy continued after discharge, without the use of anticoagulants.

The patients with SISMAD who fail medical therapy require intervention primarily for persistent pain [17]. Endovascular intervention involves stent placement [18]. In clinical settings, the contrast-enhanced CT and the CTA revealing aneurysmal dilation of the SMA larger than 2 cm or severe compression of the true lumen (more than 80% compared with adjacent normal SMA diameter) are firm indications for endovascular stenting [22]. Arterial rupture and bowel infarction are absolute indications for emergency surgical intervention [22].

Our patient had a favorable evolution with the relief of the symptoms within two days, explained by the presence of intestinal collateral circulation. The collateral circulation of the gastrointestinal system can compensate for approximately a 75% acute reduction in mesenteric blood flow for up to 12 h, without substantial injury [23]. The presence of arterial Doppler signal in the intestinal wall and the absence lactate increase and/or metabolic acidosis are witnesses of a mild ischemic jejunitis.

Conclusions

In conclusion, in a man in his sixth decade with acute abdominal pain and transient segmental jejunitis (diagnosed by clinical and ultrasound criteria) SISMAD should be taken into account as possible diagnosis. Contrast-enhanced CT and/or CTA should be considered in the early stage for an accurate diagnosis. We observed a benign clinical course after conservative treatment even without anticoagulant treatment, with no progression of SISMAD on follow-up CT.

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