ISSN: 2755-015X

Journal of Surgery & Anesthesia Research

SCIENTIFIC Research and Community

Case Report

Open Access

Refractory Chronic Abdominal Pain: An Unusual Manifestation of Superior Mesenteric Artery Thrombosis

Víctor Raúl Luraschi Centurión[1], Montserrat Almada Ruiz Diaz[2], Roun Kim[3*], Victor Daniel Orue Mequer[4], Jorge Oscar Benitez Ramos[5] and Héctor Daniel Del Puerto Peula[5]

[1]Medical Director Head of the General Surgery Department, General Hospital of Luque, Paraguay

[2]Emergency Coordinator, General Surgery Department, General Hospital of Luque, Paraguay

[3]General Surgery Specialist, General Hospital of Luque, Paraguay

[4]3rd-Year Resident, General Surgery, General Hospital of Luque, Paraguay

[5]University Lecturer, General Surgery, General Hospital of Luque, Paraguay

ABSTRACT

Mesenteric ischemia is an uncommon but potentially life-threatening condition, associated with high morbidity and mortality when diagnosis is delayed. Thrombosis of the Superior Mesenteric Artery (SMA) is one of the main causes of arterial mesenteric ischemia and is typically characterized by sudden onset of severe abdominal pain. However, in selected cases, particularly when the obstruction is partial or develops progressively, the clinical presentation may be insidious, mimicking more common gastrointestinal disorders.

Chronic postprandial abdominal pain, progressive weight loss, nausea, vomiting, and intolerance to oral intake are typical features of chronic or subacute mesenteric ischemia. The coexistence of dyspeptic symptoms or upper gastrointestinal bleeding may further obscure the diagnosis and lead to delays in appropriate management. Contrast-enhanced computed tomography currently represents the diagnostic modality of choice, allowing early detection of vascular involvement before the development of irreversible intestinal ischemia.

We report a case of partial mural thrombosis of the superior mesenteric artery presenting as chronic epigastric pain and upper gastrointestinal bleeding, highlighting the diagnostic challenges and the importance of considering vascular etiologies in patients with refractory abdominal symptoms.

***Corresponding author**

Roun Kim, General Surgery Specialist, General Hospital of Luque, Paraguay.

Received: July 09, 2026; **Accepted:** July 13, 2026; **Published:** July 23, 2026

Keywords: Superior Mesenteric Artery Thrombosis, Mesenteric Ischemia, Chronic Abdominal Pain, Upper Gastrointestinal Bleeding

Introduction

Mesenteric ischemia is an uncommon but potentially fatal condition, whose clinical presentation ranges from fulminant acute episodes to chronic or subacute forms that are often difficult to recognize. Thrombosis of the Superior Mesenteric Artery (SMA) is one of the leading causes of arterial mesenteric ischemia, generally associated with atherosclerosis, hypercoagulable states, or underlying systemic disease [1,2].

Classically, arterial mesenteric ischemia presents with severe abdominal pain of sudden onset, often disproportionate to the findings on physical examination. However, in some patients—particularly when the obstruction is partial or develops progressively—the condition may present as chronic postprandial abdominal pain, weight loss, and intolerance to oral intake, mimicking benign gastrointestinal disorders [3].

The coexistence of dyspeptic symptoms and upper gastrointestinal bleeding can delay diagnosis and misdirect the initial clinical approach. In this context, contrast-enhanced computed tomography is the diagnostic tool of choice for identifying mesenteric vascular involvement [2,4]. We present a case of partial mural thrombosis of the SMA with an atypical presentation, highlighting the diagnostic and therapeutic challenges it posed.

Case Presentation

A 63-year-old male patient presented to the emergency department with chronic abdominal pain of approximately three years' duration. The pain had an insidious onset, was oppressive in character, and was located in the epigastrium, with progressive radiation to the entire upper abdomen. Initially mild, the pain gradually intensified, with clear worsening over the preceding month and marked exacerbation in the 48 hours before admission.

The pain worsened after food intake and was associated with recurrent nausea and vomiting; in the two days prior to consultation, the patient developed episodes of hematemesis. He reported an approximate weight loss of 10 kg over the course of the illness

and, in the preceding weeks, progressive intolerance to oral intake, with vomiting occurring approximately one hour after ingesting any food or liquid.

He had previously been evaluated by gastroenterology and started on proton pump inhibitors (omeprazole) and analgesics, without clinical improvement.

On admission, the patient was hemodynamically stable, with vital signs within normal limits. Abdominal examination revealed a soft, depressible abdomen, tender to palpation throughout the upper abdomen and most pronounced in the epigastrium, without signs of peritoneal irritation, with normal bowel sounds. Digital rectal examination showed stool of normal color.

Laboratory studies showed hemoglobin 16.7 g/dL, hematocrit 49%, leukocytosis of 18,190/mm³ with neutrophil predominance (83%), platelets 348,000/mm³, and a prothrombin time of 100%. Serum electrolytes were within normal limits. Liver function tests showed AST 43 U/L, ALT 26 U/L, and alkaline phosphatase 149 U/L. Serum amylase was 78 U/L. Urinalysis showed proteinuria (++), urobilinogen (+++), and hematuria (+).

Abdominal ultrasound showed a distended gallbladder with thin walls and no evidence of gallstones; the bile ducts were not dilated, with a common bile duct of normal caliber. The remainder of the study showed no relevant findings.

Contrast-enhanced computed tomography of the abdomen was performed and revealed partial mural thrombosis in the proximal segment of the superior mesenteric artery, without tomographic signs of acute intestinal ischemia (Figure 1).

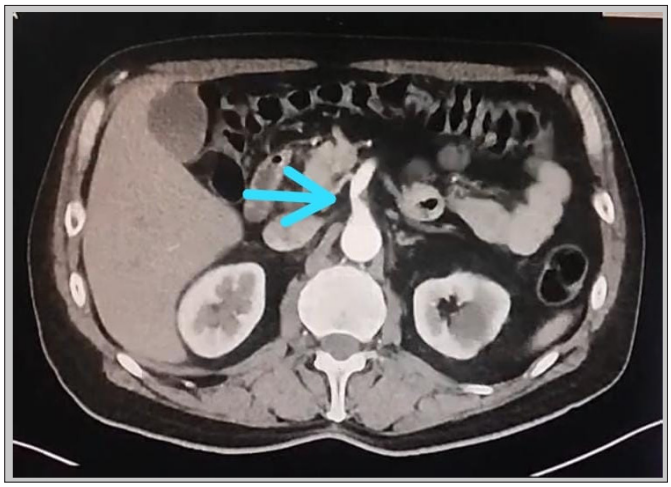

Figure 1: Contrast-enhanced abdominal CT, axial section at the level of the origin of the Superior Mesenteric Artery (SMA), showing the proximal SMA segment (arrow) with partial mural thrombosis. Reference image obtained from a video capture of the PACS workstation; a native high-resolution export is recommended as a substitute, if available, for the final published version.

Upper gastrointestinal endoscopy revealed gastropathy of undetermined etiology, thickening of the duodenal mucosa, and a Forrest III duodenal ulcer, prompting gastric and duodenal biopsies. The biopsy report showed moderate, active chronic antral and body gastritis, with marked edema and vasocongestion of the lamina propria. No intestinal metaplasia, glandular atrophy, or epithelial dysplasia was observed in the examined sections.
Given the finding of mesenteric thrombosis, additional studies were requested for etiological evaluation, including arterial blood gas analysis, serum lactate, protein C and S, antithrombin III, factor V Leiden, quantitative C-reactive protein, and echocardiography. The patient was evaluated by the hematology service, which indicated initiation of oral anticoagulation with apixaban at a loading dose of 10 mg every 12 hours for 7 days, followed by a maintenance dose of 5 mg every 12 hours, with discontinuation of the previously prescribed low-molecular-weight heparin. A vascular surgery consultation was also requested to determine definitive management.

Follow-Up

The initial thrombophilia work-up (February), obtained while the patient was already anticoagulated, showed a prothrombin time of 111% (normal range 70–130%), protein S 135.8% (normal range 73–130%), protein C 155.1% (normal range 70–140%), antithrombin 132.5% (normal range 83–118%), lupus anticoagulant AL1 with a ratio of 1.5 (positive), AL2 with a ratio of 1.18 (negative), and a total ratio of 1.27, with a positive lupus inhibitor reported. Given the recognized potential for direct oral anticoagulants to interfere with phospholipid-dependent coagulation assays used to detect lupus anticoagulant, the study was repeated three months later (May) under the same anticoagulation regimen, showing a prothrombin time of 108%, AL1 with a ratio of 1.11 (negative), a negative lupus inhibitor, anticardiolipin IgG antibody below 2.6 (negative), and anticardiolipin IgM antibody 4 (negative). The reversion to negative of the lupus anticoagulant and anticardiolipin antibodies on follow-up testing precludes fulfillment of the classification criteria for antiphospholipid syndrome, which require persistent positivity on two determinations at least 12 weeks apart, and is consistent with an initial false-positive result attributable to apixaban interference with the assay. The elevated (not decreased) values of protein C, protein S, and antithrombin likewise do not indicate an inherited hypercoagulable state, since only deficiency of these factors is associated with thrombophilia. Histopathological analysis of the gastric and duodenal biopsies remained pending.

On clinical follow-up, the patient remained on oral anticoagulation, was asymptomatic, and showed a favorable clinical course with adequate tolerance of oral intake.

Discussion

Thrombosis of the superior mesenteric artery is an infrequent cause of abdominal pain but carries substantial morbidity and mortality when it progresses to established intestinal ischemia [1,2]. Unlike mesenteric embolism, which typically presents acutely and dramatically, arterial thrombosis can develop progressively, allowing chronic or subacute symptoms to emerge [3].

In this case, the prolonged course of abdominal pain, its relationship to food intake, and the associated weight loss initially directed the clinical approach toward a chronic gastrointestinal disorder. The concomitant presence of a duodenal ulcer and gastropathy further obscured the underlying vascular etiology, delaying the definitive diagnosis. This scenario has previously been described in the literature as a common cause of diagnostic delay in chronic mesenteric ischemia [4].

Contrast-enhanced computed tomography is currently the diagnostic method of choice, offering high sensitivity and specificity for detecting mesenteric thrombosis and evaluating signs of intestinal ischemia [2,5]. In this patient, it identified partial mural thrombosis of the SMA in the absence of intestinal necrosis, which allowed for initial conservative management.
Treatment of arterial mesenteric thrombosis without established ischemia is based on systemic anticoagulation, aimed at preventing

thrombus progression and promoting vascular recanalization [3,6]. In recent years, direct oral anticoagulants such as apixaban have emerged as a therapeutic alternative in selected patients, offering a favorable safety profile and reduced need for monitoring, although specific evidence in arterial mesenteric thrombosis remains limited [7].

The initial etiological work-up reported a positive lupus anticoagulant, a finding that raised the possibility of antiphospholipid syndrome as an underlying cause of the thrombosis. However, this determination was performed while the patient was already anticoagulated with apixaban, a condition recognized to interfere analytically with phospholipid-dependent coagulation assays (such as the dRVVT) used to detect lupus anticoagulant, potentially generating false-positive results [8, 9]. The reversion to negative of the lupus anticoagulant and anticardiolipin antibodies on repeat testing at 12 weeks, under the same anticoagulation regimen, is consistent with this interference and, according to current classification criteria for antiphospholipid syndrome which require persistent positivity on at least two determinations 12 weeks apart rules out this diagnosis [10]. Likewise, the elevated levels of protein C, protein S, and antithrombin are not indicative of thrombophilia, since only their deficiency is associated with a prothrombotic state. Taken together, these findings point toward an atherosclerotic or degenerative etiology of the SMA mural thrombosis rather than an acquired thrombophilia, which is consistent with the patient's age and underscores the importance of not overinterpreting thrombophilia work-ups obtained during acute anticoagulation without subsequent confirmatory testing.

This case underscores the importance of maintaining a high index of clinical suspicion in patients with chronic abdominal pain refractory to conventional treatment, particularly when associated with weight loss and food intolerance, even in the absence of classic signs of acute mesenteric ischemia.

Conclusion

Partial mural thrombosis of the superior mesenteric artery can present as an insidious, prolonged clinical picture that mimics common gastrointestinal disorders. Persistent symptoms, lack of response to standard medical treatment, and the presence of alarm signs should prompt an expanded diagnostic evaluation. Contrast-enhanced computed tomography is essential for timely diagnosis. The etiological thrombophilia work-up did not demonstrate antiphospholipid syndrome or any other inherited thrombophilia; the initial positivity of the lupus anticoagulant, obtained during apixaban anticoagulation, was not confirmed on follow-up testing at 12 weeks, underscoring the need for cautious interpretation of these assays when performed during active anticoagulation. Early initiation of anticoagulation in selected patients can favorably alter the clinical course and prevent serious complications, as demonstrated by the favorable clinical course and sustained tolerance of oral intake observed in this patient's follow-up.

Declarations

Informed Consent

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Conflict of Interest

The authors declare that they have no conflicts of interest.

References

1. Acosta S (2015) Mesenteric ischemia. Curr Opin Crit Care 21: 171-178.
2. Clair DG, Beach JM (2016) Mesenteric ischemia. N Engl J Med 374: 959-968.
3. Björck M, Koelemay M, Acosta S, Bastos FG, Kölbel T, et al. (2017) Editor's Choice Management of the diseases of mesenteric arteries and veins. Eur J Vasc Endovasc Surg 53: 460-510.
4. Oldenburg WA, Lau LL, Rodenberg TJ, Hope JE, Charles DB (2004) Acute mesenteric ischemia: a clinical review. Arch Intern Med 164: 1054-1062.
5. Endean ED, Barnes SL, Kwolek CJ, Minion DJ, Schwarcz TH, et al. (2001) Surgical management of thrombotic acute intestinal ischemia. Ann Surg 233: 801-808.
6. Brandt LJ, Feuerstadt P, Longstreth GF, Scott JB (2015) ACG Clinical Guideline: Epidemiology, risk factors, patterns of presentation, diagnosis, and management of colon ischemia. Am J Gastroenterol 110: 18-44.
7. Valerio L, Riva N, Di Nisio M, Omri C (2019) Splanchnic vein thrombosis treated with direct oral anticoagulants: a systematic review and meta-analysis. Thromb Res 174: 43-49.
8. Favaloro EJ, Mohammed S, Curnow J, Pasalic L (2019) Laboratory testing for lupus anticoagulant (LA) in patients taking direct oral anticoagulants (DOACs): potential for false positives and false negatives. Pathology 51: 292-300.
9. Favaloro EJ, Pasalic L, Lippi G (2022) Lupus anticoagulant testing during anticoagulation, including direct oral anticoagulants. Res Pract Thromb Haemost 6: e12676.
10. Miyakis S, Lockshin MD, Atsumi T, Branch DW, Brey RL, et al. (2006) International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). J Thromb Haemost 4: 295-306.