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Case Report

Asymptomatic Intestinal Ischemia Secondary to Thrombosis of the Spleno-Mesenteric Portal Axis: Usefulness of Laparoscopic Approach for Diagnosis and Therapeutical Decisions—Case Report and Review of the Literature

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Abstract: Mesenteric Venous Thrombosis (MVT) is a rare but often lethal form of ischemia. The diagnosis may be difficult for the clinician due to its mild symptoms with a great discrepancy between the pain suffered by the patients and the clinical observation. The non-specificity of the laboratory tests and the challenge in computed tomography (CT) scan in distinguishing when an ischemia is occurring lead to the risk of an unrecognized diagnosis, especially considering that an early onset of treatment is largely fundamental for better outcomes. Also, the choice between the surgical approach and the anticoagulation may be a pitfall for the clinicians. The treatment choice requires a multidisciplinary approach. Emergency exploration by laparoscopy allows clinicians to reach the correct diagnosis and the real-time assessment of the intestinal infarction extension, playing a double diagnostic and therapeutic role in those doubtful and tricky cases. Here, a 77-year-old male with a completely asymptomatic bowel ischemia secondary to a Mesenteric Venous Thrombosis was submitted to a laparoscopic approach.

Keywords: mesenteric thrombosis; ischemia; laparoscopy

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1. Introduction

Mesenteric Venous Thrombosis (MVT) is an uncommon but often lethal form of ischemia. It results from a thrombus or blood clot developed in one of the veins returning blood from the bowel. This condition was first described by Elliot in 1895 [1]; in 1926, Cokkins stated that "Occlusion of the mesenteric vessels is apt to be regarded as one of those conditions of which the diagnosis is impossible, the prognosis hopeless and the treatment almost useless" [2]. This was true in the pre-anticoagulant era when medical management was primitive. Today, however, we have improved our understanding of the disease process and its therapy. Only in 1935, Warren and Eberdard [3] recognized it as being distinct from arterial occlusion, defining its clinical presentation.

A mesenteric venous thrombosis is a deep venous thrombosis that occurs due to coagulation problems interfering with bowel venous return [4–7]. It represents 0–16% of all cases of intestinal infarction and 5–15% without mechanical obstruction [8]. Although it is rare, it is potentially deadly with a mortality rate of 20–30% [8,9]. Life-threatening risks include infarction, following the thrombosis.

Three main patterns of presentation of MVT are described: the acute one, the subacute and the chronic form. The diagnosis is challenging because of the discrepancy between the abdominal pain suffered by the patient and the physical examination observed [1]. Etiologically, we can count primary mesenteric venous thrombosis, spontaneous and

idiopathic. A history of peripheral venous thrombosis and migratory thrombophlebitis is often present in these patients. A secondary mesenteric venous thrombosis instead arises from underlying diseases or risk factors [3].

The development of computed tomography (CT) and the introduction of CT angiography have improved the early detection of Mesenteric Venous Thrombosis, improving survival, decreasing the risk of recurrences and preventing thrombus propagation [9]. Early diagnosis is necessary to prevent the complications of this disease that can lead to serious complications and death.

When bowel ischemia is suspected, surgeons might choose between operative management or conservative treatment with anticoagulation [8]. Exploratory laparotomy in acute stages is often complicated due to undefined borders between the ischemic bowel and the viable bowel. Since the bowel viability is difficult to determine, especially in early diagnosis, overly aggressive bowel resection may result in consequent short bowel syndrome [10]. Starting from a peculiar case report, the aim of this paper is to review the present literature to better understand and define the most useful approaches for a correct diagnosis and subsequent therapeutical decisions.

2. Case Report

A 77-year-old male complaining of constipation for five days without signs of abdominal pain was evaluated by the Emergency Department.

His medical history showed a previous pulmonary thromboembolism and a benign prostatic hyperplasia. At that time, the patient was not taking any medication despite the previous thrombosis.

His vital signs were as follows: heart rate 67 bpm, SpO2 (oxygen saturation) 94%, P (pressure) Max 199 mmHg, P Min 100 mmHg, and T (temperature) 36. The abdomen was globular but tender, not painful, without any signs of peritonism; peristalsis was torpid but present. All laboratory studies including arterial blood gases, metabolic profile, complete blood count, urinalysis and coagulation profile were normal. A mild leukocytosis (white blood cell 14.000 [Range 4.000–10.000 U/μL]) and a rise of CRP (C-Reactive Protein) (64 mg/dL [Normal Value < 10 mg/dL]) were the only relevant data in the blood tests.

Subsequently, an X-Ray of the abdomen showed enterocolic distension in the right quadrant with several air-fluid levels. So, in order to exclude a form of mechanical ileus, a CT scan of the abdomen was requested for diagnostic completion.

The CT scan highlighted meteoric distension of jejunal loops and middle proximal ileum with thickened walls and poor contrast enhancement, markedly edematous adipose tissue, occlusion on a thrombotic basis of the entire spleno–mesenteric–portal axis up to its more distal branches; hepatic parenchyma in segments VI-VII-VIII inhomogeneously hypodense due to hypoperfusion phenomena; and perisplenic, pelvic and right iliac effusion and along the mesenteric fan (Figure 1).

The radiological findings were indicative of jejunal ischemia with diffuse visceral hypoperfusion secondary to complete occlusion of the spleno–mesenteric–portal axis in an asymptomatic patient. As the etiology of this clinical scenario was a complete thrombotic occlusion conditioning small bowel ischemia, the surgery team assumed immediate operative management and performed exploratory laparoscopy.

A laparoscopic approach has been chosen with the intention of using exploratory laparoscopy as a last diagnostic investigation, for direct visualization of the abdominal condition in case a widespread ischemic picture incompatible with survival had been detected. By opting for a laparoscopic approach, the aim was to minimize surgical time and avoid unnecessary surgical procedures in cases where a severe and non-survivable abdominal condition was present.

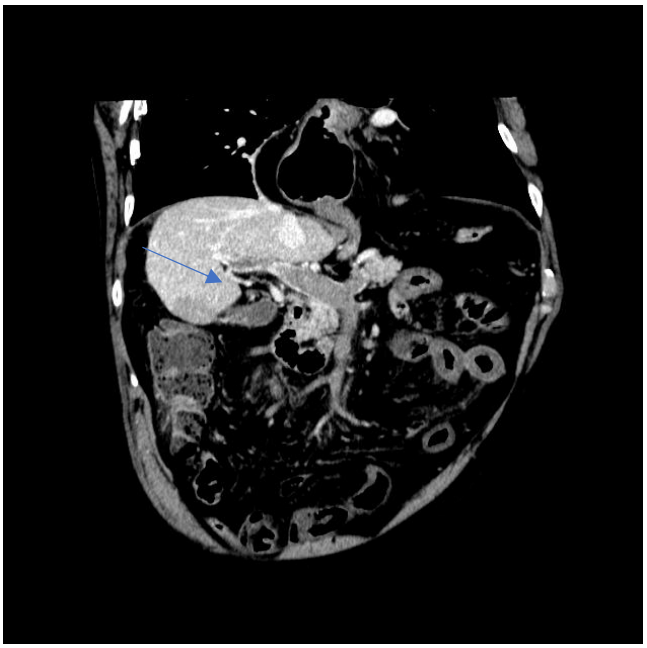

Figure 1. CT scan image showing the thrombosis of the spleno–mesenteric–portal axis (blue arrow).

During this procedure, a turbid exudate was observed in the abdomen and an ischemic pattern involving a jejunal and meso-jejunal segment extended for 120 cm from the Treitz's ligamentum. Due to these findings, the surgeons converted the surgical procedure from laparoscopy to open laparotomy for proceeding to the enteric resection. The patient was left with an open abdomen, according to Barker's technique, to evaluate the case within 48 h to better define any further extension of the ischemia (Figure 2).

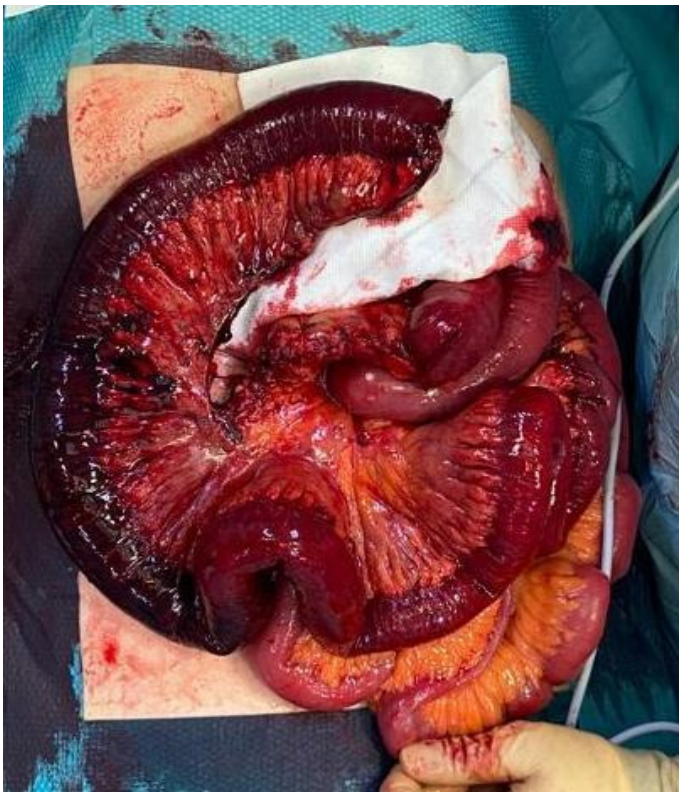

Figure 2. Length of the Jejunal-ileal necrosis.

The patient was moved, sedated and intubated in the Intensive Care Unit where Low Molecular Weight Heparin (LMWH) 20,000 IU 2 mL/h was administered.

In the perioperative period, the patient was afebrile and was hemodynamically stable, with no onset of multi-organ failure or anemia.

At 48 h after the first intervention, the surgeons proceeded with a surgical revision. The exploration of the abdominal cavity showed no further extension of the ischemia with normal perfusion of the viscera, so an isoperistaltic later-lateral jejunal-ileal anastomosis was executed followed by the abdominal wall closure.

The patient remained in the ICU for 2 days and was then transferred to the Surgery Department on the 5th postoperative day for further care and rehabilitation.

Given the normal hemoglobin value (10.9 g/dL [Range 12–14 g/dL]), the normal platelet count (PLT 214,000 [Range 150,000–450,000 U/µL]) and a renal function with Creatinine of 1.03 [Range 0.8–1.2 mg/dL], continuous heparin was suspended 5 days after the operation, first prescribing LMWH 4000 IU twice daily and subsequently modulated to 6000 IU plus 8000 IU die.

Oral feeding was regularly and gradually reintroduced, showing no complications during hospitalization. Before discharge, a new CT-angiography was performed with evidence of recanalization of the portal vein and the spleno–mesenteric–portal confluence; minimal residual thrombosis remained near its confluence, and the superior mesenteric vein was recanalized with no signs of ischemic intestinal distress.

The patient was regularly discharged 14 days after the first surgical procedure with LMWH twice daily, then switched to direct-acting oral anticoagulants (DOAC) 1 month after the discharge. The histological examination of the bowel described that the intestinal section suffered a consistent transmural hemorrhagic infarction.

No post-discharge complications were noted at a one- and six-month follow-up. Within the first week of post-discharge, the patient was recovering normal bowel habits, and he has been able to restart his daily physical activities without any significant symptoms.

During regular follow-up visits, the patient continued to improve and was able to control his bowel habits, eat a regular diet and maintain an active lifestyle. He continues to visit the Surgical Department for regular follow-ups and is able to maintain a normal activity level without experiencing any symptoms related to thrombotic vascular obstruction.

3. Discussion

MVT is an unusual but often lethal form of ischemia. The incidence of Mesenteric Venous Thrombosis is 2.7/100,000 patient years and accounts for 5–15% of mesenteric ischemia in the population. This is likely to be an underestimation of its true incidence since the autopsy diagnoses represent 1.5% of the population. Most of the patients are older than 70 years [1,10,11]. As predictors of mortality, we can consider colon involvement, fortunately, colon ischemia occurs only in 5–13% of MVT, failure of anticoagulation therapies, extensive bowel resection leaving less than 100 cm of bowel [12], age, cardiac illness and acute renal failure [13].

According to the literature, it can be described as a Primary Mesenteric Venous Thrombosis and a Secondary form. MVT is considered primary in lacking etiology or predisposing factors, and it represents up to 49% of the cases. Secondary Mesenteric Venous Thrombosis takes into consideration as risk factors all of the conditions involved in hypercoagulation disorders and prothrombotic states. The most common are Thrombocythemia, post-splenectomy thrombocytosis, polycythemia, neoplastic disorders, abdominal inflammatory conditions (inflammatory bowel disease, diverticulitis, pancreatitis), genetic defects affecting antithrombin III, Protein C and S [4–6], atrial fibrillation and aneurysms of the aorta and the visceral arteries [11,14]. It has been estimated that 60% of patients suffering from MVT had a history of deep venous thrombosis in the past [1].

Generally, thrombosis of the superior mesenteric vein involves the ileum and jejunum segments of the bowel. MVT is characterized by generalized abdominal cramps nonproportional to the clinical findings [1]. Nonspecific symptoms include nausea, vomiting, diarrhea,

hematemesis and hematochezia. The nonspecific features of the pain and the possibility of initially normal laboratory and clinical findings may delay the diagnosis [7]. This delay contributes to the 15–40% mortality rate indicated in the literature [1]. As affirmed by Clavien, clinical signs of peritonitis (tenderness, rebound pain and guarding) were noted less frequently in patients with MVT than in those with mesenteric infarction of other etiologies. Patients often complain that pain started days before admission [15]. Signs like fever, rebound tenderness and guarding are often shown when a progression to bowel infarction is occurring.

Leukocytosis and metabolic acidosis are the most specific laboratory findings [7]. Other tests such as phosphorus, alkaline phosphatase, aspartate aminotransferase, creatinine phosphokinase and lactate dehydrogenase levels have low sensitivity and specificity.

Abdominal radiographs of the case may demonstrate nonspecific dilated ileus and fluid-filled loops of the bowel. It is generally accepted that computer tomography is the best diagnostic test for acute MVT. With a 91% accuracy, CT-angiography of the abdominal vessels with 3D reconstruction is the gold standard for diagnosing acute mesenteric ischemia [1]. Mesenteric ischemia or infarction due to acute MVT manifests as continuous bowel wall thickening to greater than 20 mm with layered and/or inhomogeneous enhancement, mesenteric haziness, mesenteric vascular engorgement and ascites [1,16]. It was Bradbury's [17] idea that thrombosis in the SMV, thickening of the bowel wall and peritoneal fluid suggest bowel infarction secondary to MVT, requiring laparotomy. The findings are confirmed in Salem et al.'s case series, where a previous history of thromboembolism is associated with small bowel edema, dilation and ascites, which indicate resection risk [11]. Performing a selective mesenteric angiography can establish a definitive diagnosis demonstrating a thrombus in the mesenteric or portal vein, absent filling of mesenteric veins, arterial spasm, prolongation of arterial phase and prolonged blush in the involved segment [1].

Besides the use of CT for diagnosis, Reuttimann stated the feasibility of the measurement of the gastric intramucosal pH for monitoring the gut perfusion in these thromboses. The affection of thrombosis in gastrointestinal perfusion is critical for the disruption of the gastrointestinal mucosal barrier and the development of sepsis. This rapidly affects the gastric pH, even in cases of normal arterial lactates. This allows for observing the evolution of ischemia, avoiding unnecessary laparotomies, and the effect of the nonsurgical therapies [18]. Treatment of MVT depends on the stage of presentation; the options include anticoagulation therapies, local or systemic thrombolysis and interventional or surgical thrombectomy [8]. When bowel infarction is present, resection is mandatory [1]. Early diagnosis allows noninvasive therapies such as intravenous fluids, nasogastric decompression and anticoagulation. When surgery or interventional therapy is delayed for more than 12 h after initial symptoms, mortality increases significantly [11]. The management of acute MVT patients has two potential difficulties. There are two issues to consider: one is whether to explore surgically or treat conservatively, and the second is whether to confirm a viable bowel when exploring surgically. Systemic anticoagulation for the prevention of thrombus propagation is a well-established treatment modality and a mainstay of MVT treatment. This reduces recurrence rates and improves survival rates. Surgical exploration is indicated when peritoneal irritation is present. There is no clear correlation between peritoneal signs and bowel ischemia severity. So new criteria have been proposed, such as bowel wall thickness and bowel wall enhancement on the arterial phase. Limited areas of infarcted bowel can be surgically resected and anastomosed without significant morbidity; however, surgeons should try to conserve as much bowel as possible. Unfortunately, the border between the ischemic bowel and viable bowel is often diffused. In many cases, it is initially unclear whether the bowel is viable or nonviable, with a dusky presentation. In those situations, surgeons might prefer avoiding extensive resection that may lead to complications such as short gut syndrome, intestinal failure, undernutrition and hydroelectrolytic abnormalities. This supports a damage control approach with an open abdomen and surgical re-exploration within 48 h of continuous monitoring and resuscitation [19].

Obviously when available, indocyanine green is a powerful instrument to guarantee a direct view of the viable borders of a bowel suffering from ischemia. Using indocyanine green and other fluorescence techniques applied to laparoscopy allows us to view the vascularization of the bowel not only to identify better ischemic areas but also bowel segments macroscopically normal but lacking sufficient blood flow that might evolve into further necrosis, perforations and deadly complications [10,19]. During ischemia, the necrosis of enteral mucosa occurs in 3 h, while the trans-mural, full-thickness wall necrosis requires 6 h. In this range of time, even the CT scan may lead to a misdiagnosis without recognizing the real situation. These possible mistakes enhance the use of indocyanine green that, with its deep tissue penetration even in low blood-supplied cases, allows for the identification of patchy enteric segments that are not found by other diagnostic tools [20].

Primary anastomosis is proposed by some surgeons; however, recurrent infarction usually involving anastomosis develops in up to 60% of cases and is usually caused by incomplete resection of the ischemic segments. As a result, an open abdomen or a second-look laparotomy has been also advocated in the literature [3,15]. The second-look technique improves survival rates by up to 70% [19]. If during surgical re-exploration at 48 h, the macroscopic evaluation of the intestine is not sufficient, even in these cases the use of indocyanine green can be applied to identify a further extension of necrosis [21]. Rongwei et al. performed a hybrid approach combining surgical strategies with intraoperative fluoroscopic-assisted balloon thrombectomy. In their series, they described a 100% effectiveness in removing the thrombi, preserving venous splanchnic outflow, relieving bowel congestion and limiting ischemia [8]. The present case has some limitations due to the difficult diagnosis. For this patient, a prompt diagnosis has been made avoiding quoad vitam sequelae. Unfortunately, in real-life practice, it does not always happen due to the possible asymptomatic presentation of this condition.

4. Conclusions

Bowel ischemia secondary to Mesenteric Venous Thrombosis is a rare but not-to-be-missed diagnosis. The main difficulties for clinicians are the non-specificity of physical examinations and laboratory signs. Even the CT scan can be useless in determining the onset and the extension of bowel ischemia secondary to Mesenteric Venous Thrombosis. Despite that, an early diagnosis is mandatory to perform timely treatment avoiding worsening of the outcome. The choice between conservative management and a surgical approach requires a multidisciplinary evaluation. Emergency exploration by laparoscopy, like in our case, allows for reaching the correct diagnosis plus the real-time assessment of the intestinal infarction extension, thus playing a double-diagnostic and therapeutic role in those doubtful and tricky cases.

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