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MESENTERIC VENOUS THROMBOSIS LEADING TO SMALL BOWEL NECROSIS IN A VIETNAMESE PATIENT WITH PROTEIN C DEFICIENCY: A CASE REPORT

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ABSTRACT

Mesenteric venous thrombosis (MVT) is a rare but life-threatening cause of acute abdominal pain, particularly when associated with congenital protein C deficiency. We present a 65-year-old Vietnamese man with acute epigastric and periumbilical pain and loose stools. Though initially hemodynamically stable, he showed signs of peritonitis. Laboratory findings revealed leukocytosis and elevated D-dimer. Contrast-enhanced CT confirmed complete thrombosis of the superior mesenteric vein, partial portal vein thrombosis, and bowel wall edema. Protein C activity was reduced to 50%. The patient underwent emergency laparotomy with resection of one meter of necrotic bowel and creation of a double-barrel ileostomy. He received postoperative anticoagulation with enoxaparin, later switched to apixaban. Discharged in stable condition on postoperative day 14, he showed resolution of portal vein thrombosis at one-month follow-up. This case underscores the importance of early imaging, recognition of inherited thrombophilia, and timely surgical and anticoagulant therapy in managing MVT.

Keywords: Protein C deficiency; Mesenteric venous thrombosis; Small bowel necrosis; Inherited thrombophilia.

1. INTRODUCTION

Mesenteric venous thrombosis (MVT) is defined as the formation of a thrombus within the superior or inferior mesenteric veins or their branches, often leading to impaired venous return, bowel ischemia, and potential necrosis. This condition accounts for 5–15% of acute mesenteric ischemia cases and may arise from inherited thrombophilias, such as protein C deficiency, which heightens the risk of recurrent thrombosis, or acquired prothrombotic states. It can also be triggered by or associated with inflammatory processes [1]. Contributing factors include trauma, venous stasis, malignancy, infections, physical injury, and systemic inflammation. Although over 25% of MVT cases initially appear idiopathic, comprehensive evaluation frequently uncovers identifiable etiologies. The thrombosis may originate in small venules or the main vein and can extend to the portal vein. The superior mesenteric vein is involved in over 90% of cases, while the inferior mesenteric vein is affected in approximately 11% [2,3]. MVT is a rare yet potentially fatal condition characterized by nonspecific symptoms, such as abdominal pain, distension, and diarrhea, which complicates timely diagnosis. Contrast-enhanced computed tomography (CT) is pivotal for prompt identification, enabling urgent interventions like surgical resection of necrotic bowel and anticoagulation therapy to improve outcomes.

This case report describes a patient with small bowel necrosis due to complete superior mesenteric vein thrombosis and partial portal vein thrombosis, precipitated by congenital protein C deficiency.

2. CASE

A 65-year-old Vietnamese male patient with a body mass index of 24 presented with a one-day history of epigastric and periumbilical abdominal pain, occurring in episodes, accompanied by two episodes of loose stools without fever. He self-presented to the hospital in stable condition, with clear consciousness, a respiratory rate of 22 breaths per minute, oxygen saturation of 98% on room air, pulse of 90 beats per minute, and blood pressure of 120/70 mmHg. Physical examination revealed a distended abdomen with positive peritoneal signs. His past medical history included a right coronary artery stent placement 25 years prior, treated with aspirin 81 mg daily for two years post-procedure, which was subsequently discontinued. One year ago, he was diagnosed with left lower limb deep vein thrombosis and

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treated with rivaroxaban 20 mg daily, discontinued two months before presentation. He reported no allergies, smoking, alcohol, or recreational drug use. Family history was unremarkable for inheritable conditions, and he lived independently in a private residence, with no reported mobility limitations.

Laboratory investigations showed leukocytosis (white blood cell count 14 G/L, neutrophils 86%, lymphocytes 7.5%), elevated hemoglobin (179 g/L), hematocrit (0.51), and normal platelet count (254 G/L). Coagulation studies revealed elevated D-dimer (2128 ng/mL, reference <500 ng/mL). Renal function tests showed elevated urea (12 mmol/L) and creatinine (138 µmol/L) (Table 1). CT of the abdomen with contrast revealed no free intraperitoneal air, but demonstrated small bowel wall edema (up to 14.5 mm thickness) with poor contrast enhancement (pre–contrast 45 HU, post–contrast 52 HU), surrounding fat stranding, partial portal vein thrombosis, and complete superior mesenteric vein thrombosis (Figure1A, 1B). The superior mesenteric artery and celiac trunk were patent. Peritoneal fluid was noted around the liver, spleen, and pelvis, with normal spleen size and structure.

The patient's symptoms began one day prior to admission, with no delays in presentation or diagnosis. The clinical and imaging findings led to a diagnosis complete superior mesenteric vein thrombosis and partial portal vein thrombosis. Following the diagnosis of superior mesenteric vein thrombosis, a workup for underlying prothrombotic conditions was promptly initiated. Coagulation assays revealed plasma protein S activity at 118% (reference range 77–143%), significantly reduced plasma protein C activity at 50% (reference range 70–140%), and plasma antithrombin III activity at 114% (reference range 77–143%) as determined by chromogenic testing (Table 1). Based on these findings, a definitive diagnosis of superior mesenteric vein thrombosis secondary to protein C deficiency was established. Genetic testing for PROC mutation was not available due to resource limitations; however, the persistently low functional protein C levels in absence of secondary causes strongly supported a diagnosis of inherited deficiency. Differential diagnoses included acute appendicitis, perforated peptic ulcer, and ischemic colitis, which were excluded due to the absence of free intraperitoneal air, lack of localized right lower quadrant findings, and CT evidence of small bowel involvement with thrombosis. The prognosis was guarded due to the extent of bowel necrosis and underlying thrombophilia.

The patient underwent urgent surgical intervention with no specific pre-operative optimization due to the acute presentation. Intraoperative findings revealed a 1-meter segment of small bowel, approximately 1 meter proximal to the ileocecal junction, with necrosis, purple-black discoloration, rigidity, edema, and absent peristalsis. The proximal 2 meters of small bowel showed moderate dilation (3–4 cm), mild congestion, but preserved perfusion. The peritoneal cavity was relatively clean, with clear fluid and no pseudomembranes. A segmental small bowel resection was performed, followed by a double-barrel ileostomy (Figure 2). General anesthesia was used, with the patient positioned supine, and standard sterile preparation applied. No novel techniques or devices were employed. The procedure was performed in a tertiary hospital by a senior surgical team with extensive experience in emergency abdominal surgery. Post-operatively, enoxaparin 60 mg was administered subcutaneously twice daily starting 12 hours after surgery for venous thromboembolism prophylaxis and treatment of thrombosis. On postoperative day 6, the patient transitioned to apixaban 5 mg twice daily, intended for long-term use.

Table 1. Laboratory Investigations Summary

Parameter	Result	Reference Range	Parameter	Result	Reference Range
Hematology			**Coagulation Profile**		
White Blood Cell Count	14 G/L	4–10 G/L	Prothrombin Time	113%	70–120%
Neutrophils	86%	40–75%	Activated Partial Thromboplastin Time	25 sec	25–35 sec
Lymphocytes	7.5%	20–45%	Fibrinogen	4.6 g/L	2–4 g/L
Hemoglobin	179 g/L	120–160 g/L	D-dimer	2128 ng/mL	<500 ng/mL
Hematocrit	0.51	0.40–0.54	Protein S activity	118%	77–143%
Platelets	254 G/L	150–400 G/L	Protein C activity	50%	70–140%
Arterial Blood Gas (ABG)			Antithrombin III activity	114%	77-143%
pH	7.37	7.35–7.45	**Renal & Liver Function Tests**		
PaO_2	138 mmHg	75–100 mmHg	Urea	12 mmol/L	2.5–7.5 mmol/L

Parameter	Result	Reference Range	Parameter	Result	Reference Range
$PaCO_2$	40 mmHg	35–45 mmHg	Creatinine	138 µmol/L	60–110 µmol/L
HCO_3	22 mmol/L	22–26 mmol/L	AST (Aspartate Aminotransferase)	54 IU/L	<40 IU/L
Base Excess	-2 mmol/L	-2 to +2 mmol/L	ALT (Alanine Aminotransferase)	8 IU/L	<40 IU/L
Lactate	1.5 mmol/L	<2.0 mmol/L	Total Bilirubin	18 µmol/L	<20 µmol/L

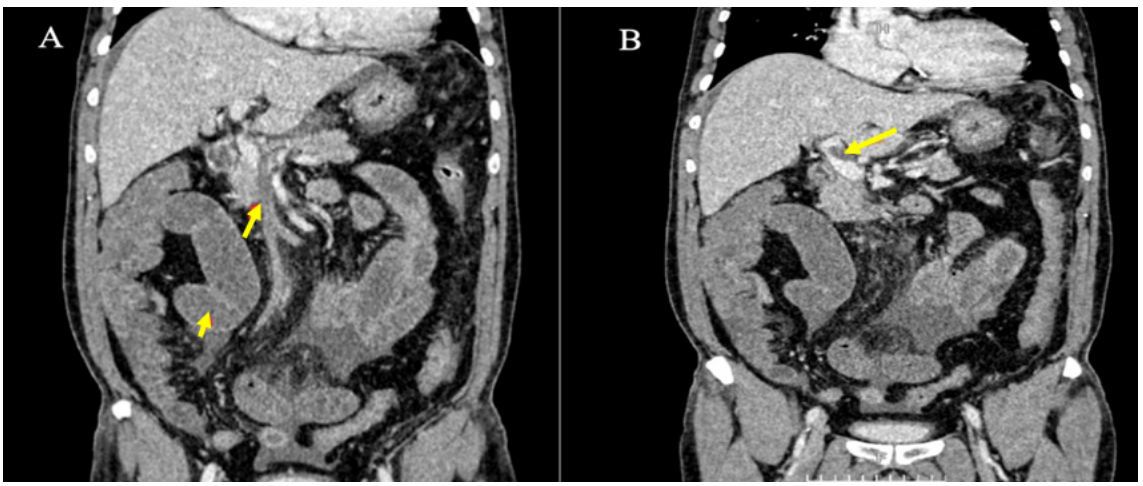

Figure 1.

Contrast-enhanced computed tomography (CT) of the abdomen shows small bowel wall edema with poor contrast enhancement, perienteric fat stranding, partial thrombosis of the portal vein (Figure 1A, red arrow), and complete thrombosis of the superior mesenteric vein (Figure 1B, orange arrows)

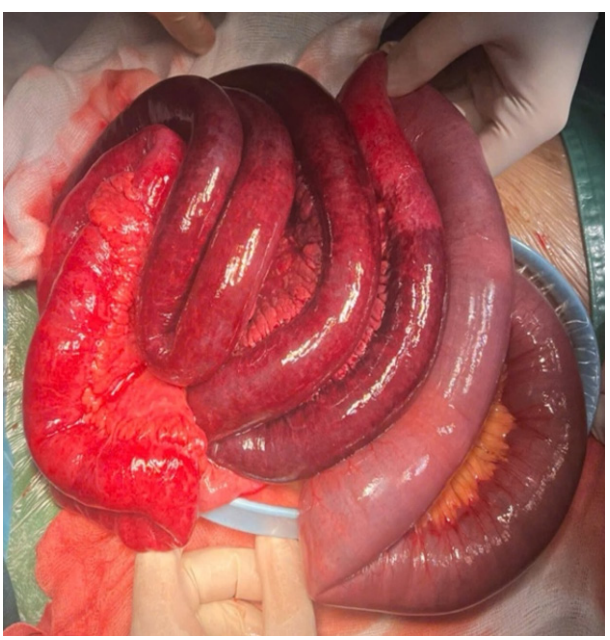

Figure 2.

Intraoperative findings showing a necrotic segment of small bowel approximately 1 meter proximal to the ileocecal junction, characterized by purple-black discoloration, edema, rigidity, and absence of peristalsis. The proximal small bowel is moderately dilated with preserved perfusion. Segmental small bowel resection and double-barrel ileostomy were subsequently performed

The patient's postoperative course was stable, with discharge on day 14. Follow-up at one month included clinical examination and abdominal ultrasound, which showed resolution of portal vein thrombosis. Coagulation test results: PT 102%, APTT 30 seconds, fibrinogen 4.5 g/L, and notably, protein C activity remains low at 45%. The patient adhered well to the anticoagulation regimen, with no reported adverse effects, and tolerance was assessed via clinical follow-up and patient-reported compliance. The expected outcome, based on literature regarding mesenteric venous thrombosis with prompt surgical and anticoagulant therapy, was resolution of thrombosis and restoration of bowel function, which was achieved. No long-term surveillance imaging was planned, but lifelong anticoagulation was recommended due to protein C deficiency. The patient reported no significant quality-of-life impairments at follow-up. Written informed consent was obtained from the patient for publication of this case report and accompanying images. The patient understood that all personal identifiers would be removed to protect privacy.

5. DISCUSSION

This case report of a 65-year-old Vietnamese male with small bowel necrosis secondary to mesenteric venous thrombosis (MVT) due to congenital protein C deficiency provides valuable insights into the diagnosis and management of a rare and potentially life-threatening condition.

Protein C is a vitamin K-dependent anticoagulant protein synthesized in the liver. It has a molecular weight of approximately 62 kilodaltons (kDa) and consists of two polypeptide chains connected by a disulfide bond [4]. Protein C circulates in the blood as a zymogen (an inactive precursor) and exerts its anticoagulant function after being activated into activated protein C (aPC), a serine protease. This activation process occurs when thrombin binds to thrombomodulin, a specific receptor on the surface of vascular endothelial cells. The synthesis of gamma-carboxyglutamic (Gla) acids in protein C requires the presence of vitamin K [5]. The Gla domain binds to calcium, inducing a conformational change that facilitates protein C's binding to phospholipids, a critical step for its anticoagulant function. The primary role of aPC is to inactivate coagulation factors Va and VIIIa, which are essential for efficient thrombin generation and factor X activation . The inhibitory effect of aPC is enhanced by protein S, another vitamin K-dependent anticoagulant protein. Hereditary protein C deficiency can lead to a thrombophilic state (increased propensity for blood clot formation) [4].

Most patients with hereditary protein C deficiency are heterozygous. Over 195 different mutations in the protein C gene (PROC) have been described. This gene is located on chromosome 2 (2q13-14) [6]. Individuals with protein C deficiency may experience venous thromboembolism (VTE) at any site; deep vein thrombosis (DVT) of the legs, mesenteric vein thrombosis, and pulmonary embolism (PE) are the most common. VTE can also affect other sites, including cerebral veins, portal veins, superficial veins, or other unusual locations. The risk of thrombosis with heterozygous protein C deficiency is increased approximately sevenfold [7]. In this case, although genetic testing for PROC mutations was not available due to resource limitations, the diagnosis of inherited protein C deficiency was supported by multiple clinical and laboratory findings. Notably, protein C activity remained consistently low (45%) at one-month follow-up, well beyond the acute phase, which reduces the likelihood of transient suppression due to illness or inflammation. Common secondary causes of acquired protein C deficiency—including liver dysfunction, vitamin K deficiency, disseminated intravascular coagulation, and warfarin use—were ruled out based on normal liver function tests, absence of coagulopathy, and no history of anticoagulant therapy at the time of admission. In addition, the patient had a prior episode of unprovoked deep vein thrombosis, which further supports the presence of a prothrombotic predisposition. According to established clinical guidelines, persistently reduced functional protein C levels in the absence of secondary causes, combined with a history of thrombosis at an unusual site, are strongly indicative of congenital deficiency [8]. Therefore, in this context, a working diagnosis of inherited protein C deficiency was made.

The literature supports the management strategy employed in this case. Lev N. Korovin, et al. (2016) reported that MVT accounts for 5–15% of acute mesenteric ischemia cases, with inherited thrombophilias, such as protein C deficiency, being significant risk factors [1]. This aligns with the patient's presentation, where protein C activity was significantly reduced (50%, reference range 70–140%), confirming the underlying prothrombotic state. Similar published cases highlight the rarity and complexity of MVT associated with protein C deficiency. A case reported by H Orozco et al. (1988) described two patients with MVT and portal vein thrombosis due to protein C deficiency, managed with bowel resection and lifelong warfarin, achieving resolution of thrombosis [9]. Khalid Al Sulaiman, et al. (2022) emphasize lifelong anticoagulation for patients with protein C deficiency to prevent recurrent thrombosis, consistent with the transition to apixaban 5 mg twice daily in this case [10]. The American College of Gastroenterology (ACG) guidelines (Simonetto et al., 2020) recommend Doppler ultrasound as the initial noninvasive modality for diagnosing portal vein thrombosis (PVT), followed by contrast-enhanced CT or MRI to assess thrombus extension into mesenteric veins, which mirrors the diagnostic approach taken here with CT imaging confirming complete superior mesenteric vein thrombosis and partial portal vein thrombosis. The ACG guidelines further advocate anticoagulation for all noncirrhotic patients with acute symptomatic MVT in the absence of contraindications, supporting the use of enoxaparin and apixaban in this case [8].

This case generate hypotheses for future research. The underreporting of inherited thrombophilias in Vietnamese populations, as noted in the limited literature on MVT in Vietnam, suggests a potential higher prevalence of conditions like protein C deficiency than currently recognized. This warrants population-based studies to assess the incidence of thrombophilias in patients with unexplained venous thrombosis in this region.

Several limitations should be acknowledged in this case. The tertiary care setting with advanced resources may limit applicability to facilities with diagnostic constraints. While clinical and laboratory findings strongly suggested hereditary protein C deficiency, genetic confirmation of PROC mutation was unavailable. The one-month follow-up was insufficient to evaluate long-term outcomes such as recurrent thrombosis or stoma complications.

4. Conclusion: Key take-away lessons include the importance of considering inherited thrombophilias in patients with atypical abdominal pain and a history of thrombosis, as well as the critical role of CT imaging in confirming MVT. Early multidisciplinary intervention is essential to optimize outcomes in such cases. In future similar cases, the authors would prioritize genetic testing to confirm protein C deficiency, enhancing diagnostic precision, and consider earlier hematology consultation to tailor anticoagulation regimens. Additionally, extended follow-up with periodic imaging could be implemented to monitor for recurrence, given the lifelong risk of thrombosis in protein C deficiency. This case underscores the need for heightened awareness of rare thrombotic etiologies in acute abdominal presentations and the value of integrated care in achieving successful outcomes.

CONFLICT

The authors declare that they have no conflicts of interest relevant to this manuscript.

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The authors have nothing to report.

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