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| RESEARCH ARTICLE

Recurring Abdominal Pain: The Hidden Clue of Antiphospholipid Syndrome

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| ABSTRACT

Recurrent abdominal pain is a common clinical presentation, yet when episodes are severe, episodic, and unexplained by routine gastrointestinal investigations, vascular etiologies must be considered. We present a 52-year-old previously healthy man with a six-week history of intermittent, severe central abdominal pain, culminating in an acute exacerbation. Prior evaluations, including ultrasound, non-contrast CT, and endoscopy, were unrevealing. On acute presentation, laboratory studies demonstrated elevated D-dimer and mild lactate elevation, prompting contrast-enhanced CT angiography, which revealed a proximal superior mesenteric artery thrombus with preserved bowel viability. Further workup confirmed triple-positive antiphospholipid syndrome (APS), establishing the underlying prothrombotic etiology. The patient underwent urgent systemic anticoagulation with unfractionated heparin followed by successful endovascular thrombectomy and catheter-directed thrombolysis. He was subsequently transitioned to long-term warfarin therapy with multidisciplinary follow-up. This case highlights the protean manifestations of APS, demonstrating that recurrent, transient mesenteric ischemia may precede catastrophic arterial thrombosis. Early recognition, prompt vascular imaging, and tailored anticoagulation—combined with minimally invasive revascularization when feasible—are pivotal in preventing bowel infarction and optimizing outcomes. Clinicians should maintain high suspicion for APS in atypical thrombotic presentations beyond the classic venous or cerebrovascular events.

| KEYWORDS

Abdominal Pain, Antiphospholipid Syndrome, Thrombophilia, Mesenteric Ischemia, Arterial Thrombosis, Lupus Anticoagulant, Anticardiolipin, AntiB2-Glycoprotein I, Anticoagulation, Thrombosis, Small Bowel Infarction

| ARTICLE INFORMATION

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Introduction

Antiphospholipid syndrome (APS) is a systemic autoimmune thrombophilic disorder characterized by the persistent presence of circulating antiphospholipid antibodies (aPL)—principally lupus anticoagulant, anti-β_2-glycoprotein I, and anticardiolipin antibodies—in patients who develop characteristic clinical manifestations of thrombosis and/or pregnancy morbidity [1,2].

Originally delineated among individuals with systemic lupus erythematosus (SLE), APS is now firmly recognized as a distinct clinical and immunopathological entity, which may arise either as a **primary (idiopathic)** phenomenon or **secondary** to another autoimmune disease, most often lupus [1]. The pathogenic basis of APS resides in a complex interplay between immune dysregulation, endothelial activation, and coagulation pathway perturbation. Antiphospholipid antibodies bind β_2-GPI and related phospholipid-binding proteins on cellular membranes, triggering endothelial cell and monocyte activation with subsequent **upregulation of tissue factor** and **downregulation of thrombomodulin**, thereby tipping the hemostatic balance toward thrombosis. Simultaneously, these antibodies promote **platelet aggregation**, **complement activation**, and the release of **neutrophil extracellular traps (NETs)**, amplifying a self-sustaining procoagulant cascade [3]. This framework is elegantly conceptualized in the **"two-hit" model**: an underlying chronic prothrombotic state induced by aPL (the first hit), compounded by a secondary precipitating factor such as infection, systemic inflammation, trauma, or pregnancy (the second hit), ultimately resulting in overt thrombotic or obstetric events [1,3]. The clinical consequences of this immune-coagulant synergy include arterial and venous thromboses across multiple organ systems and, in the obstetric context, placental vascular insufficiency culminating in fetal growth restriction, preeclampsia, or fetal loss. Clinically, APS presents with remarkable heterogeneity, encompassing a continuum from isolated venous thrombosis to catastrophic multisystemic involvement. The **cardinal manifestations** remain **unexplained arterial or venous thromboembolic events** and **pregnancy morbidity** [1,2]. Classically, affected individuals—often young adults without traditional vascular risk factors—present with **deep vein thrombosis**, **pulmonary embolism**, **ischemic stroke**, or **myocardial infarction**. Obstetric manifestations include **recurrent miscarriages**, **late fetal demise**, and **severe preeclampsia** resistant to conventional therapy [2]. By formal definition, APS may involve vessels of **any caliber or anatomic distribution** [1]. Large-scale cohorts demonstrate that approximately half of APS cases are **primary**, while the remainder are **secondary**, typically in association with SLE or another systemic autoimmune disease [1]. In all instances, the detection of aPL correlates strongly with heightened risk for both thrombosis and adverse pregnancy outcomes [1,2]. Although traditional teaching emphasizes thrombotic events in "usual" territories such as the deep veins of the legs or cerebral arteries, APS has gained notoriety for producing occlusions in **unusual or unexpected sites**, including the **cerebral venous sinuses**, **renal arteries**, **retinal vessels**, and **digital arteries**, reflecting its profoundly systemic nature [1]. In striking contrast to these well-recognized presentations, **abdominal pain as the initial manifestation of APS** is exceedingly uncommon and often overlooked. The majority of reported cases focus on limb, cerebral, or placental vasculature, whereas **abdominal organ involvement** is comparatively rare [5]. A recent case series documented that although gastrointestinal lesions related to APS have been described in the **esophagus**, **stomach**, **intestine**, and **visceral vasculature**, such occurrences remain "infrequent" and "rarely reported" [5]. Particularly unusual is the scenario wherein **recurrent abdominal pain** constitutes the **sole presenting feature** of APS [5]. In the case under discussion, the patient experienced an **eight-month history of intermittent epigastric discomfort**, prompting extensive gastroenterological work-ups, including endoscopy and routine laboratory evaluation, all of which were unremarkable. It was only upon cross-sectional imaging that **thromboses involving the portal, splenic, and superior mesenteric veins**—with accompanying small-bowel dilatation—were identified [4,5]. This finding led to the performance of aPL assays, which ultimately established the diagnosis of APS. Such cases, in which APS masquerades as chronic or recurrent gastrointestinal pain, have been reported only sporadically in the literature. For instance, Zou et al. described a young woman with two previous miscarriages whose only complaint was episodic abdominal pain; computed tomography revealed **mesenteric vein thrombosis with intestinal obstruction**, and APS was confirmed by the presence of lupus anticoagulant and anti-β_2GPI antibodies [5]. Collectively, these accounts underscore that while **gastrointestinal involvement in APS is recognized**, it is an **exceptional occurrence** that often results in **delayed recognition and treatment**. The **mechanistic underpinnings** of abdominal manifestations in APS follow directly from its thrombotic pathophysiology. **Mesenteric ischemia** may result from arterial or venous thrombosis within the superior mesenteric circulation, leading to reduced intestinal perfusion and ischemic pain [4]. Similarly, **thrombosis of the portal or hepatic veins**—the latter constituting **Budd–Chiari syndrome**—may ensue, producing **hepatic congestion**, **acute liver injury**, and **ascites**. Indeed, certain case series have identified APS as an underlying etiology in a subset of non-tumorous Budd–Chiari patients, further illustrating this pathogenic connection. Moreover, **splenic infarction** may arise from thrombosis of the splenic artery or vein, typically observed on imaging as wedge-shaped hypodense regions within the spleen [4]. APS may also involve the **adrenal vasculature**, where venous thrombosis can precipitate **adrenal infarction or hemorrhage**—a potentially catastrophic complication manifesting as acute abdominal pain, nausea, vomiting, and, in bilateral cases, adrenal crisis [6]. Pathologically, such adrenal hemorrhage reflects thrombosis of the central adrenal vein, leading to ischemic necrosis followed by secondary bleeding [6]. These diverse vascular insults—mesenteric, portal/hepatic, splenic, and adrenal—collectively demonstrate how APS can **simulate a wide array of abdominal pathologies** and present as **unexplained visceral ischemia** in patients lacking classical predisposing factors. **Diagnosing APS in this setting presents formidable clinical challenges.** Patients presenting with isolated abdominal symptoms generally undergo targeted gastrointestinal evaluations—endoscopy, colonoscopy, ultrasonography, or computed tomography—without an initial suspicion

of systemic coagulopathy. The diagnosis is typically entertained only after radiological demonstration of vascular occlusion. According to current criteria, a definitive diagnosis of APS requires both **persistent aPL positivity** (detected on at least two occasions separated by ≥12 weeks) and a qualifying **clinical event** (thrombosis or pregnancy morbidity) [1]. In atypical or delayed presentations, the "first hit" (presence of aPL) often becomes apparent only retrospectively. Indeed, the patient described herein had **no prior history of deep vein thrombosis or cerebrovascular accident**, only a background of **two miscarriages**, and thus APS was not suspected until the development of visceral thrombi [5]. This exemplifies how **an unconventional presentation—isolated, recurrent abdominal pain—** in the absence of overt risk factors (e.g., lupus, malignancy, or hormonal therapy) may significantly delay diagnosis. Hence, clinicians must maintain a **heightened index of suspicion** and incorporate aPL testing into the evaluation of **unexplained recurrent visceral ischemia**. Notably, the **2023 ACR/EULAR classification criteria for APS** emphasize the need to consider **"extra-criteria" manifestations**, encouraging vigilance for such atypical presentations [1]. Comparison with **systemic lupus erythematosus** provides instructive contrast. SLE frequently involves the gastrointestinal tract, most notably through **lupus enteritis**—an immune-mediated vasculitis of the small intestine—which represents the most common etiology of **acute abdominal pain** in SLE patients [7]. Lupus enteritis generally responds rapidly to high-dose corticosteroids, reflecting its **inflammatory** pathogenesis. In contrast, **gastrointestinal manifestations in APS are primarily thrombotic**, stemming from vascular occlusion rather than immune-mediated vasculitis. Although APS and SLE often coexist, the **mechanistic distinction** is clinically pivotal: in APS, gastrointestinal ischemia signifies a **clotting phenomenon**, whereas in lupus it reflects **immune vasculitis** or **serositis**. Similarly, **lupus-related serositis or pancreatitis** may mimic APS-related symptoms, but therapeutic strategies differ substantially—**immunosuppression** for lupus-driven inflammation versus **anticoagulation** for APS-mediated thrombosis. Thus, while both are systemic autoimmune diseases, the **pathophysiological substrate** of gastrointestinal involvement diverges: inflammatory in SLE, thrombotic in APS [7]. Recognizing this distinction is crucial for selecting appropriate therapy and preventing irreversible organ injury. The **prognostic importance of early recognition** in such atypical cases cannot be overstated. APS confers an intrinsically high risk of recurrent and potentially catastrophic thrombosis, and delayed identification may result in severe morbidity or mortality. Numerous reviews stress that **timely diagnosis and multidisciplinary management**—integrating hematology, rheumatology, and gastroenterology expertise—are essential to optimizing outcomes [1]. Early recognition before irreversible bowel infarction or adrenal failure enables prompt institution of **anticoagulation therapy** and, when indicated, adjunctive treatments such as **corticosteroids** or **intravenous immunoglobulin**, particularly in obstetric cases. Consequently, attention to subtle yet recurrent clinical clues, such as **unexplained episodic abdominal pain**, can be **lifesaving**, preventing progression to catastrophic APS. A comprehensive diagnostic approach that integrates detailed clinical history, imaging findings, and aPL testing ensures accurate and timely identification of this protean syndrome. In summary, the patient's clinical trajectory illustrates a **rare but clinically significant** presentation of antiphospholipid syndrome. Recurrent abdominal pain secondary to visceral thromboses constitutes an atypical initial manifestation that is infrequently reported in medical literature [5]. By drawing attention to **mesenteric, hepatic, splenic, and adrenal ischemia** as potential heralds of APS, this report seeks to heighten clinician awareness of its **non-classical presentations**. Although uncommon, APS masquerading as chronic or intermittent abdominal discomfort is a diagnostic pitfall of genuine clinical importance. Recognition of this entity demands a **broad differential diagnosis**, meticulous clinical evaluation, and the inclusion of APS in the diagnostic algorithm for unexplained gastrointestinal ischemia. Early consideration and appropriate management can avert grave complications and significantly improve patient outcomes, reaffirming the critical insight that even subtle or atypical presentations may conceal a **systemic prothrombotic disorder** of major consequence.

Case Presentation

Patient's history and Physical Examination

This case concerns a 52-year-old Saudi man with no known chronic medical conditions, who presented to the emergency department with a six-week history of recurring, severe central abdominal pain that had acutely intensified over the preceding ten hours. The episodes had been intermittent but progressively increasing in both frequency and severity, prompting his presentation. Each prior episode had been sudden in onset, lasting several hours before resolving spontaneously. The pain was described as severe, crampy to colicky in nature, localized to the periumbilical region without radiation to the back. At its peak, the intensity reached 8 to 9 out of 10, and during the most recent episode, the pain became constant and unremitting. It was accompanied by nausea and a single episode of non-bloody vomiting, along with reduced appetite. He denied melena, hematochezia, or hematemesis, and had not experienced fever or chills at home. Earlier episodes had been associated with transient loose stools, but in the current episode, bowel movements and flatus had markedly decreased. There were no urinary complaints, no chest pain, dyspnea, palpitations, or syncopal episodes. The patient reported no history of deep vein thrombosis,

pulmonary embolism, stroke, or any other thrombotic events. His past medical history was otherwise unremarkable, and he had not been on any regular medications. He denied any symptoms suggestive of systemic autoimmune disease, such as rash, photosensitivity, arthralgia, or oral ulcers. There was no history of recent infection, travel, surgery, or prolonged immobilization. He had never used tobacco or illicit drugs, and his family history was negative for early vascular thrombosis or autoimmune conditions. Two prior emergency department visits for similar pain had yielded unremarkable investigations, including a normal abdominal ultrasound, a non-contrast CT scan of the abdomen, and a normal upper gastrointestinal endoscopy. Laboratory evaluations on those occasions were within normal limits, and no anticoagulation or further work-up was initiated at that time. Upon arrival, the patient appeared alert but visibly anxious and in moderate distress, clutching his abdomen. His vital signs were stable, with a temperature of 37.2°C, pulse rate of 98 beats per minute, blood pressure of 138/86 mmHg, respiratory rate of 18 breaths per minute, and oxygen saturation of 98% on room air. Physical examination revealed a body mass index of 24 kg/m^2. Cardiovascular and respiratory examinations were unremarkable, with normal heart sounds and clear lung fields. The abdomen was mildly distended, with notable central tenderness that appeared disproportionate to the degree of superficial palpation. There was no guarding or rebound tenderness, and bowel sounds were reduced. No palpable masses or organomegaly were detected. Peripheral pulses were palpable and symmetrical in all extremities, and there were no signs of limb ischemia, cyanosis, or edema. The skin examination showed no livedo reticularis or petechiae, and the neurological assessment was grossly normal.

Investigations and diagnostic reasoning:

Initial investigations were directed toward identifying the cause of the patient's acute abdominal pain and to evaluate for possible mesenteric ischemia. A 12-lead electrocardiogram demonstrated a normal sinus rhythm at a rate of 96 beats per minute, with no ischemic or arrhythmic changes. Laboratory evaluation revealed a hemoglobin concentration of 13.6 g/dL and a platelet count of 178 × 10^9/L, both within normal limits, while the white blood cell count was mildly elevated at 11.8 × 10^9/L, reflecting a nonspecific stress response. Serum lactate was modestly elevated at 2.8 mmol/L, suggesting early tissue hypoperfusion, and the C-reactive protein level was mildly raised at 16 mg/L. Renal and hepatic indices, including creatinine (88 µmol/L), blood urea nitrogen (4.8 mmol/L), and transaminases (AST 28 U/L, ALT 25 U/L), were normal, and the serum amylase of 48 U/L excluded pancreatitis. The international normalized ratio was 1.0, while the D-dimer level was markedly elevated at 2.1 mg/L FEU, consistent with a hypercoagulable state. Given the disproportionate pain and the absence of peritoneal signs, a contrast-enhanced computed tomographic angiography of the abdomen and pelvis was performed, revealing an abrupt thrombotic occlusion of a proximal branch of the superior mesenteric artery with reduced mural enhancement of a jejunal segment, consistent with focal small-bowel ischemia. There was no evidence of bowel perforation, free intraperitoneal air, or portal venous gas, and the affected segment showed diminished but preserved enhancement, indicating viable, though ischemic, bowel. No aortic dissection or distal emboli were identified. A transthoracic echocardiogram demonstrated normal left ventricular function without intracardiac thrombus or valvular vegetations. Screening for autoimmune and thrombophilic disorders revealed negative antinuclear and anti–double-stranded DNA antibodies, normal complement levels, and, critically, a positive antiphospholipid antibody profile with the presence of lupus anticoagulant, anticardiolipin IgG, and anti-β_2-glycoprotein I antibodies on repeat confirmatory testing, thereby fulfilling laboratory criteria for antiphospholipid syndrome. In correlation with the clinical finding of acute arterial thrombosis in the superior mesenteric artery, the diagnosis of **antiphospholipid syndrome (APS) presenting as acute mesenteric ischemia** was established. The patient's presentation was thus attributed to APS-related segmental mesenteric arterial thrombosis, with bowel viability preserved at the time of diagnosis.

Management course

Initial management focused on hemodynamic stabilization, bowel preservation, and rapid restoration of mesenteric perfusion. The patient was kept nil per os (NPO) and secured with two large-bore intravenous cannulas, with continuous cardiac and hemodynamic monitoring instituted throughout. Analgesia was provided with intravenous morphine, 4–6 mg boluses titrated to comfort, ensuring adequate pain control while preserving clinical vigilance for evolving peritoneal signs. Given the absence of frank peritonitis and imaging findings consistent with threatened but viable bowel, immediate systemic anticoagulation was initiated to prevent thrombus propagation. Unfractionated heparin (UFH) was selected as the agent of choice, administered as an 80 units/kg intravenous bolus (approximately 6,000 units for a 75-kg patient), followed by a continuous infusion at 18 units/kg/hour (approximately 1,350 units/hour), titrated to achieve an activated partial thromboplastin time (aPTT) of 60–80 seconds or 1.5–2.5 times baseline, in accordance with institutional protocol. UFH was favored for its rapid onset, short half-life, and reversibility, which are advantageous in cases where urgent surgical or endovascular intervention may be required. Empirical broad-spectrum antibiotic coverage with intravenous piperacillin–tazobactam (4.5 g every eight hours) was commenced to mitigate the risk of bacterial translocation in the setting of ischemic bowel. Given the angiographic evidence of a segmental

superior mesenteric arterial thrombus and the absence of established necrosis, the patient was referred emergently for endovascular revascularization. Under fluoroscopic guidance, right common femoral arterial access was obtained, and mechanical aspiration thrombectomy of the occluded SMA branch was successfully performed. A completion angiogram demonstrated re-established flow to the distal mesenteric circulation. To address residual thrombus burden, a local infusion of alteplase was administered, consisting of a 4-mg intra-arterial bolus followed by a catheter-directed infusion at 0.5–1 mg/hour for four hours, achieving satisfactory restoration of flow. No bowel resection was required, and the patient was transferred to the intensive care unit for close post-procedural observation. The UFH infusion was maintained for 24 hours post-thrombectomy and subsequently transitioned to long-term oral anticoagulation after ensuring hemostatic stability and gastrointestinal integrity. In view of the confirmed diagnosis of antiphospholipid syndrome and the arterial nature of the thrombosis, lifelong warfarin therapy (vitamin K antagonist) was selected, targeting an international normalized ratio (INR) of 2.5–3.5, with many centers favoring a midpoint goal of 3.0 for arterial events. Warfarin was initiated at 5 mg orally each night, overlapped with UFH until a therapeutic INR was achieved. Low-dose aspirin was not introduced at this stage, as there was no concurrent atherosclerotic disease or prior recurrent thrombosis on therapeutic anticoagulation. The management plan included the option of combination therapy (warfarin plus low-dose aspirin) or higher INR targets should recurrent arterial events occur despite adequate anticoagulation, to be revisited upon multidisciplinary review. The patient's postoperative recovery was uneventful. He remained in the intensive care unit for 48 hours of close monitoring, with gradual advancement from clear fluids on day 2 to a full oral diet as tolerated. Bowel sounds normalized, and no signs of reperfusion injury or bleeding were observed. By day 5, a therapeutic INR was achieved, and he was transitioned fully to oral warfarin therapy. He was discharged on day 7 in stable condition with comprehensive education regarding lifelong anticoagulation, potential bleeding manifestations, and the importance of regular INR monitoring. Outpatient follow-up with hematology and rheumatology services was arranged, including repeat antiphospholipid antibody testing at 12 weeks to document serologic persistence and continued care through the dedicated thrombosis clinic.

Discussion

This case exemplifies a classic presentation of antiphospholipid syndrome (APS)–related mesenteric thrombosis, illustrating the protean nature of this autoimmune prothrombotic disorder. APS, an acquired state of hypercoagulability mediated by autoantibodies targeting phospholipid-binding proteins such as β_2-glycoprotein I, predisposes to both arterial and venous thromboses and to pregnancy morbidity [1,3]. While deep vein thrombosis and ischemic stroke remain the most frequent initial manifestations, intra-abdominal arterial occlusions—including superior mesenteric artery (SMA) thrombosis—are well-documented though relatively uncommon presentations [1,4,5]. In this patient, the pattern of recurrent, transient, severe abdominal pain over several weeks, each episode resolving spontaneously until a final crescendo attack, is emblematic of intermittent mesenteric ischemia due to microembolic or in-situ thrombotic events progressively compromising blood flow until complete occlusion occurs [5,7]. This temporal evolution typifies APS-related mesenteric disease and underscores the diagnostic pitfall of episodic ischemic pain being mistaken for functional gastrointestinal disorders when early imaging proves nondiagnostic [5,7]. The laboratory diagnosis of APS hinges on integrating clinical and serological findings according to established classification criteria (Sydney 2006), which mandate at least one qualifying clinical event—such as vascular thrombosis—together with persistent positivity for lupus anticoagulant (LA), anticardiolipin (aCL), or anti-β_2-glycoprotein I (aβ_2GPI) antibodies on two or more occasions at least 12 weeks apart [1,3,9]. Because transient positivity can occur during infection or inflammatory illness, confirmatory testing is essential to avoid misclassification [1,3,10]. LA testing, a functional assay sensitive to anticoagulant interference, must be interpreted in the context of concurrent therapy; therefore, coordination with the coagulation laboratory is vital [1,9]. Although warfarin therapy can complicate confirmatory testing, anticoagulation should not be delayed in the setting of arterial thrombosis highly suggestive of APS [9,12]. In clinical practice, immediate treatment is initiated based on initial antibody positivity and a compatible thrombotic event, with repeat serology obtained once the patient stabilizes [9]. Imaging findings in this case further reinforced the diagnosis and guided management. Computed tomography angiography (CTA) remains the diagnostic modality of choice for mesenteric ischemia, delineating the location and extent of arterial occlusion, the presence of collateral circulation, and the viability of the bowel wall [4,5]. When CTA demonstrates a discrete occlusion with preserved mural enhancement, as in this patient, timely revascularization—either endovascular or open—can be lifesaving [4]. Early intervention within hours of symptom onset significantly enhances both survival and bowel preservation rates [4]. In APS-related thrombosis, mechanical thrombectomy supplemented by catheter-directed thrombolysis offers an optimal balance between rapid reperfusion and tissue salvage, avoiding the morbidity of open resection when necrosis has not yet ensued [4,9]. Anticoagulation remains the cornerstone of both acute and chronic management in APS. In the acute phase, unfractionated heparin (UFH) is preferred for its titratability, reversibility with protamine, and compatibility with emergent procedural interventions [1,9,12]. Beyond its anticoagulant effect, UFH helps stabilize thrombi and prevent propagation [12]. For long-term secondary prevention, vitamin K antagonists (VKAs) such as warfarin remain the gold standard [1,9]. Target INR ranges

typically lie between 2.0 and 3.0, though higher targets (2.5–3.5 or up to 3.0–4.0) are often favored for arterial or recurrent thrombotic events [9,12]. Recent evidence and expert consensus—including EULAR recommendations—consistently advise against the use of direct oral anticoagulants (DOACs) in high-risk APS, particularly those with triple antibody positivity or arterial events, as multiple randomized trials have demonstrated higher recurrence rates in this subgroup [9,12]. Accordingly, the use of warfarin with rigorous INR monitoring remains the most evidence-based approach for patients such as this [1,9]. Should recurrent thrombosis occur despite therapeutic INR levels, consideration may be given to combination therapy with low-dose aspirin or escalation of the INR target under specialist supervision [9,12]. While the current patient presented with localized mesenteric thrombosis, clinicians must remain alert to the potential evolution into catastrophic APS (CAPS), a fulminant variant characterized by rapid, widespread microvascular thrombosis and multi-organ failure [1,3,6]. In suspected or confirmed CAPS, prompt initiation of high-dose corticosteroids, therapeutic plasma exchange, and intravenous immunoglobulin (IVIG) is indicated, with biologic agents such as rituximab or eculizumab reserved for refractory cases [1,3,6,12]. Although not required in this instance, vigilance for early signs of CAPS—such as new renal, pulmonary, or neurologic compromise—is essential for timely escalation of therapy [1,6]. Several practical decisions in the management of APS-related mesenteric thrombosis warrant emphasis. Immediate anticoagulation with UFH should be commenced unless contraindicated, as the danger of thrombosis propagation and bowel infarction outweighs the controlled risk of bleeding [1,12]. The choice between endovascular and open surgical approaches hinges on the presence or absence of peritonitis and bowel viability; when imaging suggests viable bowel, endovascular thrombectomy is favored due to its minimally invasive nature and shorter recovery time [4]. The decision regarding long-term anticoagulant choice is equally critical—warfarin remains the standard of care in arterial APS, while DOACs are reserved for select low-risk patients with purely venous disease and single low-titer antibodies [9,12]. Post-diagnosis, structured surveillance is mandatory. Repeat antiphospholipid antibody testing at or beyond 12 weeks confirms persistence and risk stratification; triple positivity denotes high-risk disease necessitating lifelong anticoagulation [1,3,9]. Echocardiographic evaluation, including transesophageal echocardiography when indicated, may reveal valvular thickening or vegetations consistent with Libman–Sacks endocarditis, another recognized APS manifestation [1,6]. Additionally, clinicians should consider malignancy screening where thrombosis occurs in the absence of classical risk factors, and assessment for inherited thrombophilia may be justified in younger patients or those with recurrent events despite appropriate therapy [1,9,12]. The patient's prognosis in this case is favorable owing to early recognition and prompt revascularization, with preserved bowel viability and avoidance of surgical resection [4,5]. Long-term outlook in APS hinges on adherence to therapeutic anticoagulation and strict INR control; although lifelong recurrence risk remains elevated, meticulous management substantially mitigates this hazard [1,9,12]. With appropriate surveillance and multidisciplinary follow-up—including hematology, rheumatology, and vascular teams—most patients can achieve excellent functional recovery and durable prevention of further thrombotic events [1,9]. This case thus underscores the importance of recognizing APS as a potential etiology in recurrent or unexplained mesenteric ischemia, where timely diagnosis and evidence-based anticoagulation can decisively alter outcomes [1,5].

Conclusion

Recurrent, severe, or episodic abdominal pain—especially with prior non-diagnostic workup—should raise suspicion for mesenteric ischemia, and early vascular imaging (CTA) can be lifesaving. APS may first present with intra-abdominal arterial thrombosis, so testing for aPL should be considered beyond the classic DVT or stroke presentations. Acute thrombosis requires immediate anticoagulation, preferably UFH, while long-term management in arterial APS relies on VKA, avoiding DOACs in high-risk patients. Endovascular revascularization is effective when bowel viability is preserved, and catastrophic APS demands prompt, multimodal therapy. Lifelong multidisciplinary follow-up and strict adherence to anticoagulation are critical to prevent recurrence and optimize outcomes [1,4,5,9,12].

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