Silent Thrombosis, Loud Pain: Portal Vein Thrombosis Presenting With Flank Pain

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Abstract- This case report describes a 67-year-old male who presented with right flank pain and unintentional weight loss, subsequently diagnosed with spontaneous portal vein thrombosis (PVT). Despite the absence of cirrhosis, malignancy, or identifiable prothrombotic conditions, imaging and anticoagulation therapy played a critical role in managing his condition. This report underscores the importance of early diagnosis, individualized anticoagulation therapy, and thorough investigation of underlying causes in the management of PVT.

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Introduction

Portal vein thrombosis (PVT) is a rare but potentially life-threatening condition that can cause severe complications, including intestinal ischemia and portal hypertension. While typically associated with liver cirrhosis, malignancy, or prothrombotic disorders, PVT can also occur spontaneously without a clear etiology, posing significant diagnostic and therapeutic challenges. This report presents a case of spontaneous PVT in an elderly male and discusses the diagnostic approach, management, and follow-up, supplemented by a review of the relevant literature (1).

Case Report

A 67-year-old male presented to the emergency department with a two-week history of persistent, dull flank pain. He also reported early satiety and an unintentional weight loss of 5 kg over the past two months. His medical history included hypertension, hyperlipidemia, and a 20-pack-year smoking history, although he had quit smoking a decade earlier. He denied alcohol use, drug use, or a family history of thromboembolic or malignant conditions. On physical examination, the patient was afebrile, hemodynamically stable, and appeared non-toxic. Abdominal examination revealed mild tenderness in the right upper quadrant without rebound or guarding. Hepatosplenomegaly was noted on palpation. There were no signs of jaundice, ascites, or peripheral stigmata of chronic liver disease.

Initial laboratory investigations revealed normal liver function tests, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), and bilirubin levels. A thrombophilia panel, including screening for Factor V Leiden mutation, protein C and S deficiencies, and antithrombin III levels, was negative. D-dimer levels were elevated. Doppler ultrasound confirmed reduced flow in the portal vein. Contrast-enhanced computed tomography (CT) of the abdomen revealed a filling defect in the main portal vein consistent with thrombosis, without evidence of liver cirrhosis or intra-abdominal malignancy. Upper gastrointestinal endoscopy revealed no esophageal varices, and colonoscopy showed no abnormalities. The patient was started on therapeutic anticoagulation with low-molecular-weight heparin (enoxaparin) at a dose of 1 mg/kg twice daily. After ensuring tolerance to anticoagulation, he was transitioned to oral direct-acting anticoagulant therapy with apixaban. Supportive care included dietary counseling to address

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weight loss and optimization of antihypertensive therapy. One month after initiating treatment, repeat CT imaging demonstrated partial recanalization of the portal vein and a reduction in thrombus size. The patient reported significant improvement in abdominal pain and appetite. No adverse events, such as gastrointestinal bleeding or recurrent thrombosis, were noted. At the three-month follow-up, anticoagulation was continued due to the unresolved thrombus. Tumor marker levels and imaging remained unremarkable for malignancy. The patient was scheduled for regular follow-up every 3 months to monitor for recurrence and assess for late-onset malignancies.

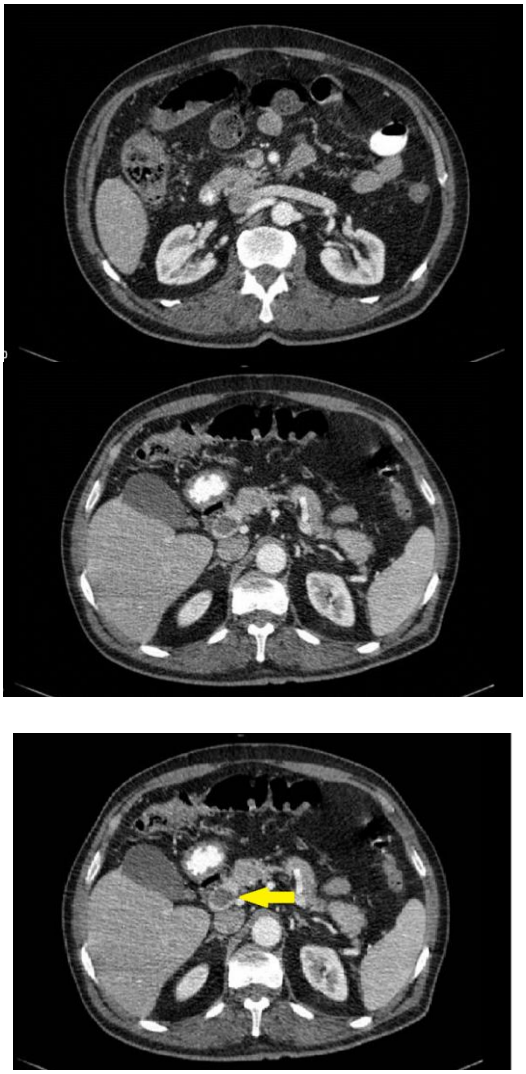

Figure 1. A hypoattenuating area within the portal vein lumen is observed (yellow arrow), representing the thrombus. This is best visualized during the portal venous phase, where the surrounding blood is hyperattenuating due to contrast enhancement. The thrombus causes non-occlusive flow, as evidenced by partial enhancement in the periphery of the vein.

Discussion

Portal vein thrombosis (PVT) has a general population prevalence of approximately 1%, but this rises significantly in patients with liver cirrhosis (10%-25%) and hepatocellular carcinoma (40%-50%). While PVT can occur at any age, it is more common in middle-aged and elderly individuals. Major risk factors encompass cirrhosis, abdominal infections (e.g., appendicitis, diverticulitis), abdominal surgery, malignancies (particularly hepatocellular and pancreatic cancer), and inherited or acquired hypercoagulable states (1,2).

The development of PVT follows Virchow's triad: endothelial injury (from infection or surgery), stasis of

blood flow (as in cirrhosis), and hypercoagulability. Acute PVT, especially with mesenteric vein involvement, can lead to bowel ischemia. In contrast, chronic PVT often results in cavernous transformation—the development of collateral veins to bypass the occlusion (2,3).

Acute PVT typically manifests with sudden, severe abdominal pain, nausea, and vomiting. Severe cases may progress to signs of intestinal ischemia, including fever, sepsis, or peritonitis. Chronic PVT is often asymptomatic and discovered incidentally, but it can present with complications of portal hypertension, such as splenomegaly, ascites, or variceal bleeding (3). Diagnosis relies on clinical suspicion confirmed by imaging. Initial laboratory workup should include liver function tests and a hypercoagulability panel. While Doppler ultrasound is a common initial imaging modality, contrast-enhanced CT or MRI offers superior sensitivity for defining the extent of the thrombus and identifying complications such as bowel ischemia or cavernous transformation (3,4). The management of acute PVT centers on anticoagulation, typically initiated with low-molecular-weight heparin (LMWH) and transitioned to oral anticoagulants (warfarin or DOACs) for 3-6 months, or indefinitely in cases of persistent hypercoagulability. If anticoagulation fails or bowel ischemia is present, thrombolysis or thrombectomy may be necessary. For chronic PVT, anticoagulation is recommended if a hypercoagulable state is identified, while management focuses on the complications of portal hypertension through beta-blockers, endoscopic variceal ligation, or, in refractory cases, transjugular intrahepatic portosystemic shunt (TIPS) (5). The prognosis of PVT depends on its etiology and the timeliness of intervention. Acute PVT with bowel infarction carries a high mortality if not treated promptly, whereas chronic PVT is often manageable but can lead to life-threatening complications like variceal bleeding. This case underscores that spontaneous PVT, while uncommon in patients without cirrhosis or malignancy, requires a high index of suspicion. Early recognition and imaging are critical to prevent devastating complications. Anticoagulation remains the cornerstone of therapy, with direct oral anticoagulants increasingly favored for long-term management. A thorough investigation for underlying causes, including occult malignancy and hypercoagulable states, is essential, even in apparently idiopathic cases. Continuous follow-up is necessary to monitor for recurrence and detect any underlying conditions that may manifest later (6).

This case emphasizes the importance of a structured approach to diagnosing and managing spontaneous PVT. Early imaging, prompt anticoagulation, and comprehensive evaluation for underlying causes are essential to optimize outcomes. Further research into the use of direct oral anticoagulants in this population is warranted.

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