Open Access Library Journal
2023, Volume 10, e10231
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Abdominal Pain: An Ominous Sign of Portal Vein Thrombosis—Case Series

Ishrat Binte Reza[1*], SM Kawser Zafor Prince[2], Imran Shikder[1], Faisal Mahmud Riyad[3], Sharmin Akhter Rupa[4], HAM Nazmul Ahasan[1], Quazi Tarikul Islam[1]

[1]Department of Medicine, Popular Medical College & Hospital, Dhaka, Bangladesh
[2]Department of Community Medicine, Popular Medical College & Hospital, Dhaka, Bangladesh
[3]Department of Surgery, Popular Medical College & Hospital, Dhaka, Bangladesh
[4]Department of Radiology, Popular Medical College & Hospital, Dhaka, Bangladesh
Email: *ishrat.reza@yahoo.com

How to cite this paper: Reza, I.B., Prince, S.K.Z., Shikder, I., Riyad, F.M., Rupa, S.A., Ahasan, H.N. and Islam, Q.T. (2023) Abdominal Pain: An Ominous Sign of Portal Vein Thrombosis-Case Series. *Open Access Library Journal*, **10**: e10231.
https://doi.org/10.4236/oalib.1110231

Received: May 9, 2023
Accepted: June 27, 2023
Published: June 30, 2023

Abstract

Portal vein thrombosis (PVT) is a disease in which thrombosis occurs from the intrahepatic branches of the portal vein and may extend to the splenic vein and/or superior mesenteric vein. It is most often associated with liver cirrhosis. PVT not associated with cirrhosis is rare. The aim of this case series of portal vein thrombosis is to give importance that even though PVT is a rare cause of abdominal pain, timely diagnosis, and appropriate management is vital due to its lethal complications such as mesenteric ischemia and mesenteric infarction and late complication like portal hypertension.

Subject Areas

Hematology

Keywords

Portal Vein Thrombosis, Abdominal Pain, An Uncommon Disease

1. Introduction

Portal venous thrombosis is an uncommon disease, difficult to diagnose, and leading to mortality. Clinical suspicion is crucial in diagnosing the disease [1] [2] [3]. Congenital or acquired prothrombotic disorders, other thrombophilia factors, or local factors play a role in etiology. Typically, a combination of multiple factors is detected [4].

Acute PVT is often asymptomatic or manifests with mild pain and is generally a

coincidental finding on abdominal imaging performed for other causes. However, SMV and mesenteric arc involvement can present with hematochezia due to congestion and ischemia in the intestine depending on the severity of involvement or can manifest as multiple organ failure ranging from shock, sepsis, and even death. Chronic PVT can be asymptomatic and can be detected incidentally on imaging methods [4]. Collateral development (portal cavernous) occurs around the thrombotic portal vein. In these patients, signs of portal hypertension such as splenomegaly, esophageal varices, anemia, and thrombocytopenia can be detected. Upper GI bleeding can be the first symptom in 20% - 40% of cases.

The diagnosis of PVT is made with imaging studies, such as CT scans, MRIs, or Doppler ultrasounds. Treatment of PVT depends on the severity of the condition and the underlying cause. In general, acute PVT is treated with anticoagulation therapy, while chronic PVT may be treated with anticoagulation or surgical intervention.

The prognosis for PVT varies depending on the severity of the condition and the underlying cause. In general, acute PVT has a good prognosis with prompt treatment. However, chronic PVT can be a progressive condition that can lead to complications such as portal hypertension, esophageal varices, and liver failure.

Abdominal Doppler ultrasonography (U.S.) for portal venous thrombosis and portal venous phase contrast-enhanced tomography (CT) for mesenteric thrombosis are valuable methods for accurate diagnosis and follow-up [5]. This case report describes two cases of portal venous thrombosis. The first case involves a 38-year-old male who presented with diffuse abdominal pain and vomiting, and a CT scan showed thrombosis within the portal vein branches, complicated by acute appendicitis. The patient underwent laparoscopic appendectomy and was treated with low molecular weight heparin and later with an oral Anti-coagulant (Rivaroxaban). The second case involves a 50-year-old female who presented with nonspecific diffuse abdominal pain and nausea. A CT scan revealed extensive venous thrombosis involving the portal and splenic veins, as well as the superior mesenteric vein, with resultant ischemia/infarction of small bowel loops. The patient was previously treated conservatively with low molecular weight heparin and oral anticoagulant for six weeks. On recent admission, the patient was found to have large esophageal and fundal varices with congestive gastropathy, and a Doppler ultrasound of portal and hepatic veins showed complete occlusion by thrombus. A CT angiogram of the abdomen with a portogram revealed a sequel of portal vein thrombosis with cavernous transformation of the portal vein with mild splenomegaly and portal hypertensive gastropathy. This report discusses the rarity of portal venous thrombosis, its causes and delays in diagnosis and treatment, and its association with acute appendicitis. We confirm that the patients had given their consent for the Case reports to be published.

2. Case 1

A 38-year-old male residing in Saudi Arabia presented to us with complaints of

diffuse abdominal pain and vomiting for 10 days. His symptoms were progressive and had progressed to the point that he was unable to eat or drink water or take medications without severe abdominal pain. On examination, the abdomen was soft and non-distended but was diffusely tender to palpation. The patient was afebrile with stable vital signs. Laboratory findings were as follows: white blood cell count 12,700/mm^3 with a normal differential, hemoglobin 10.8 g/dL, and platelet count 276,000/mm^3. Liver function tests, total protein, albumin, globulin, fasting lipid profile, and serum lipase were normal. A CT scan of the whole abdomen with I.V. contrast (Figure 1) was done and showed thrombosis within the right branch of the portal vein, main portal vein, superior mesenteric vein, and its branches complicated by acute appendicitis.

But the patient had no clinical sign of Appendicitis.

A complete hypercoagulability workup was completed. Antithrombin III level protein C and S levels and homocysteine levels were within normal limits. Pathergy test was negative. Clinically it was thought that acute appendicitis would have been the cause of this illness. He was referred to the Surgery Department and underwent laparoscopic appendectomy. The appendix was noted as being inflamed and swollen. There was no evidence of ischemic change of the intestine or active bleeding. Microorganisms were not found in the blood culture. He was treated with low molecular weight heparin and later with an oral Anti-coagulant (Rivaroxaban).

His hospital course was uncomplicated, with significant improvement in his pain and the ability to tolerate a regular diet without any gastrointestinal symptoms.

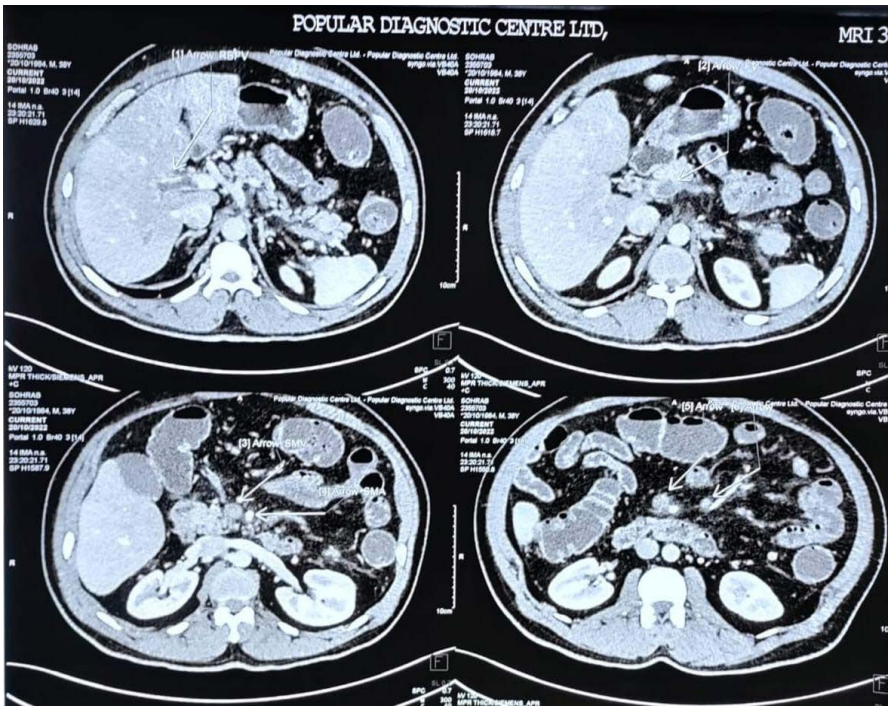

Figure 1. CT scan of abdomen with I.V contrast (case 1).

The patient's follow-up after hospital discharge revealed that he had no recurrence of abdominal pain or vomiting. His laboratory tests showed a normal white blood cell count, hemoglobin, and platelet count. His liver function tests, total protein, albumin, globulin, fasting lipid profile, and serum lipase were all within normal limits. A Doppler ultrasound of the abdomen was performed, which revealed recanalization of the portal vein and superior mesenteric vein, indicating that the anticoagulant therapy had been successful in treating thrombosis. The patient was advised to continue taking rivaroxaban as prescribed, and he was instructed to follow up with his primary care physician regularly for monitoring and management of his anticoagulation therapy. He was also advised to maintain a healthy lifestyle, including regular exercise and a balanced diet, to prevent the development of other cardiovascular diseases.

We found a similar case in the American Journal of case report [6]. This paper presents three case reports of patients with portal vein thrombosis who were presented with abdominal pain and other gastrointestinal symptoms. The authors discuss the importance of recognizing portal vein thrombosis as a potential cause of abdominal pain and the need for prompt diagnosis and treatment to prevent potentially catastrophic consequences. The paper also discusses the risk factors, diagnosis, and treatment of portal vein thrombosis. Overall, this paper is similar to this case in that it highlights the importance of recognizing portal vein thrombosis as a potential cause of abdominal pain and emphasizes the need for prompt diagnosis and treatment.

Overall, the patient's case highlights the importance of considering thrombosis in the differential diagnosis of patients with unexplained abdominal pain and vomiting, particularly in patients who have risk factors for hypercoagulability. Timely diagnosis and management with anticoagulant therapy can prevent complications and improve outcomes.

3. Case 2

A 50-year-Old female was a resident of Brunei for 10 yrs. presented with non-specific diffuse abdominal pain along with nausea for 1 month with no history of hematemesis and melena. She was normotensive, non-diabetic, and a known cause of hypothyroidism. On query, she had a history of hospital admission 2 years back for severe abdominal pain and vomiting. Before hospital admission, she suffered from postprandial pain for about 15 days.

2 years back CT scan of the abdomen (**Figure 2**) with I.V contrast was done after admission which revealed Extensive venous thrombosis involving the entire course of portal & the splenic veins as well as the superior mesenteric vein with resultant ischemia /infraction of small bowel loops in the mid jejunum.

She was then treated conservatively with low molecular weight heparin followed by an oral anti-coagulant for 6 weeks. She was asymptomatic for 2 yrs.

On recent admission, a physical examination revealed no abnormality. Her liver and renal function were normal. Endoscopy of Upper GIT revealed large

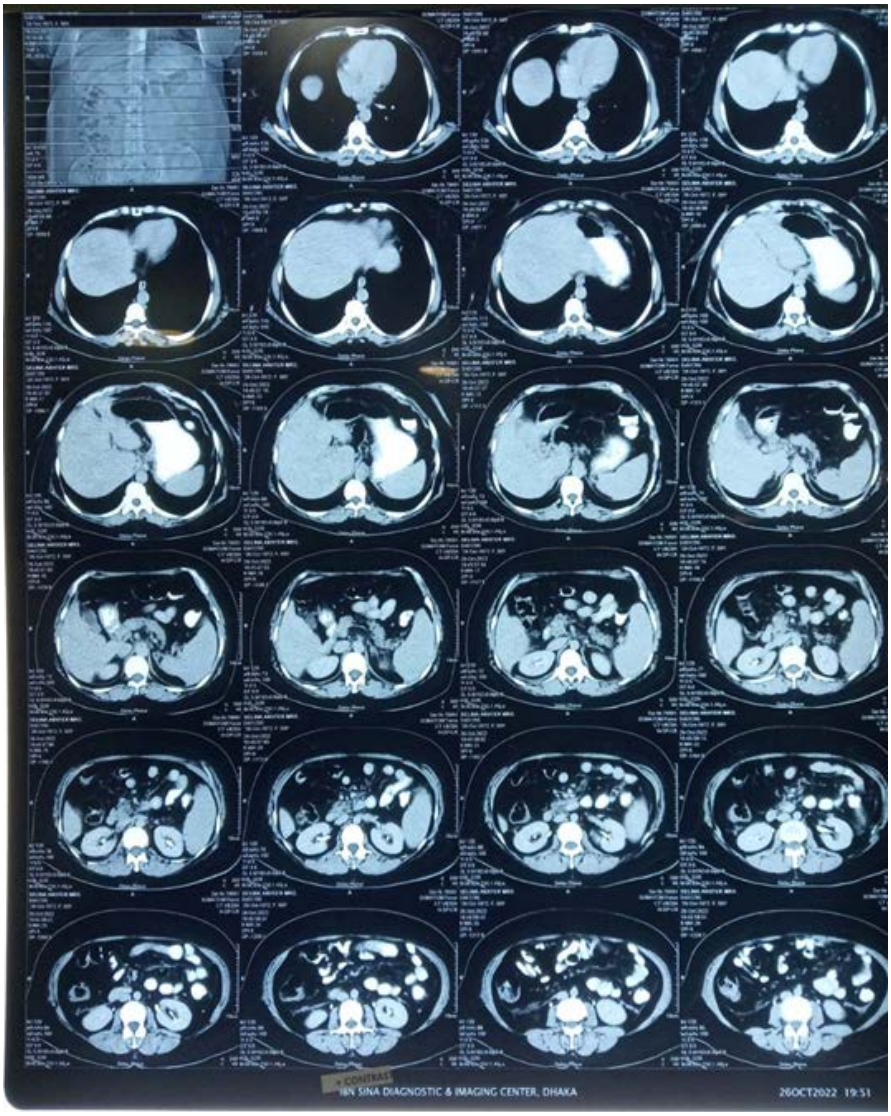

Figure 2. CT scan of abdomen with I.V contrast (Case 2).

esophageal and fundal varices with congestive gastropathy. Doppler USG of Portal and hepatic vein (**Figure 3**) showed non-visualization of the portal and splenic vein suggesting complete occlusion by thrombus.

Hypercoagulability workup was completed. Antithrombin III levels, protein C and S levels, homocysteine, and anti-phospholipid antibody levels were within normal limits. Pathergy test was negative. CT angiogram of the abdomen with a portogram (**Figure 4**) was done and the finding was the sequel of portal vein thrombosis with cavernous transformation of the portal vein with mild splenomegaly and portal hypertensive gastropathy.

Band ligation of varices was done but LMW /DOAC were not used because the patient is already on non-cirrhotic portal hypertension.

Talking about the management, given the presence of portal vein thrombosis, the patient requires anticoagulant therapy to prevent the formation of new thrombi and reduce the risk of embolic events. Low molecular weight heparin

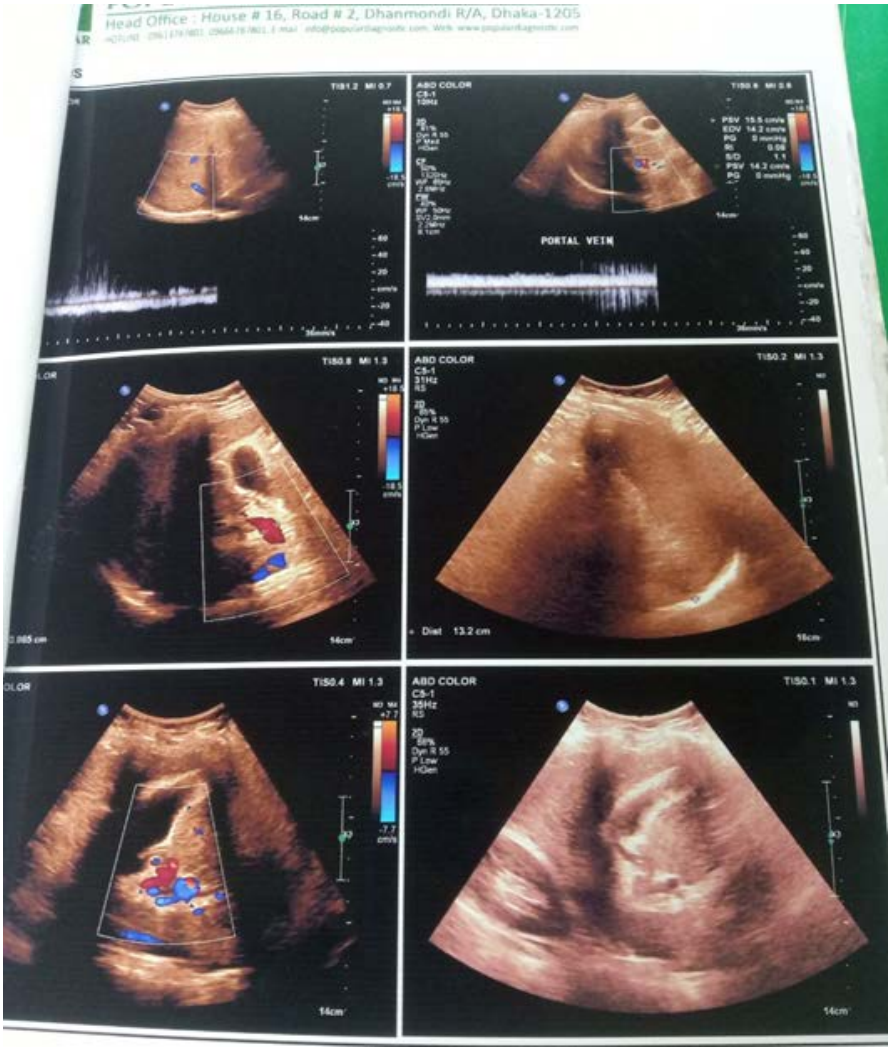

Figure 3. Doppler USG of Portal and hepatic vein.

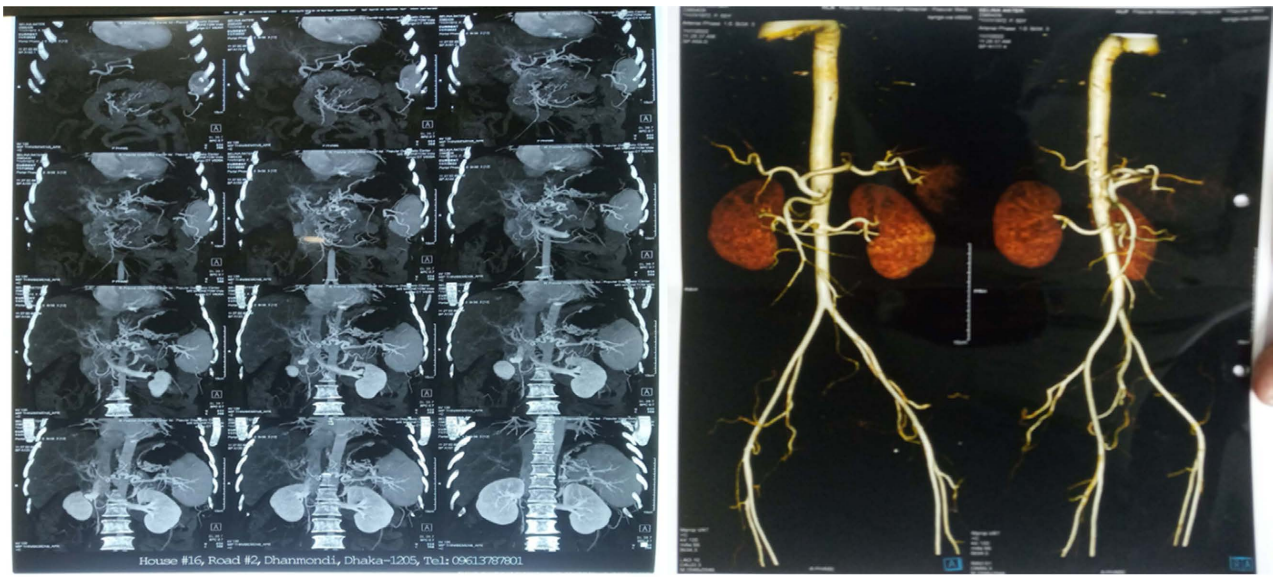

Figure 4. CT angiogram of abdomen with Portogram.

followed by oral anticoagulant therapy was previously used in this patient and can be continued. The patient has large esophageal and fundal varices with congestive gastropathy, which requires management to reduce the risk of bleeding. Band ligation of varices was done, which is a standard procedure for the management of varices. However, since the patient has non-cirrhotic portal hypertension, LMW or DOAC may not be used. Given the history of extensive venous thrombosis, the patient needs to be closely monitored for the recurrence of thrombotic events, especially in the portal vein. Periodic imaging studies such as Doppler ultrasonography or CT angiogram may be required to assess the patency of the portal vein and the progression of cavernous transformation. The patient has no identifiable hypercoagulable disorder. However, further testing may be needed to exclude any underlying thrombophilic conditions. The patient presented with diffuse abdominal pain, which needs to be managed symptomatically. Analgesics and antiemetics may be given as needed. The patient has a known history of hypothyroidism, which needs to be managed appropriately to prevent any complications. Thyroid function tests should be monitored periodically, and thyroid hormone replacement therapy may be required. Overall, the management plan for the patient should be tailored to her specific needs and medical history, with close monitoring for any complications.

4. Discussion

Portal venous thrombosis is a rare disease. It can be seen in 2.7 out of 100 thousand people, with a mortality rate of 20% - 50% [1] [2] [3]. There may often be delays in diagnosis and treatment because of the variability of signs and symptoms. In general, venous thrombi results from a combination of multiple factors [4]. Advanced age, intra-abdominal infections, cancer, cirrhosis, a history of using oral contraceptives, splenectomy, and previous upper abdominal surgery such as gastrectomy are among local or general thrombophilia factors [5]. Portal thrombosis is often neglected as part of the differential diagnosis in patients with acute abdomen who do not have the previously mentioned factors. Only 40% of patients with portal vein thrombus have local factors, therefore in half of the patients, any local factor that triggers thrombosis will not be found.

We found 39 reports of Porto-mesenteric vein thrombosis complicating acute appendicitis in children and adults from 1979 to 2016 [7]. The time from the initial symptom to diagnosis varied from a few days to 6 weeks. Delayed diagnosis occurred mostly due to the nonspecific presentation or symptoms which might be attributed to the appendicitis itself [8].

In our first case diagnosis of appendicitis was made based on abdominal pain which was not localized, raised leukocyte counts and a CT scan of the abdomen confirmed the diagnosis. An appendectomy was performed. On abdominal exploration, the appendix was edematous and inflamed. In cases where clinically acute appendicitis is suspected yet unsupported by the US, the selectivity of the US is reported as 61.5%. In such patients, preoperative abdominal CT may be

useful in diagnosis [9].

The portal venous phase of CT in conjunction with Doppler U.S. is a valuable diagnostic method in diagnosing Porto mesenteric thrombosis that provides the ability of early detection [10]. Early diagnosis can avoid bowel gangrene, perforation, and peritonitis findings, it can reduce the need for early or late laparotomy in 67% of patients [8].

In the second case, the initial presentation was similar to those of the first case. Porto-mesenteric vein thrombosis can also develop even in the absence of identifiable risk factors, as seen in our 2nd case. She was treated conservatively with Low Molecular weight followed by oral anticoagulant for 6 weeks. But her nonspecific symptoms started again within 2 years and later we found that she developed non-cirrhotic portal hypertension due to chronic portal vein thrombosis. Inadequate treatment may be the reason for this condition.

A multidisciplinary approach is essential for good management of this condition and good clinical progress.

Portal vein thrombosis is classified into 2 groups: acute and chronic. it is not always clinically possible to differentiate between the two. Acute and chronic PVT are defined as sequential stages of the same disease that occur for similar reasons, but their clinical treatments differ.

Acute PVT is often asymptomatic or manifests with mild pain, and is generally a coincidental finding on abdominal imaging performed for other causes. However, SMV and mesenteric arc involvement can present with hematochezia due to congestion and ischemia in the intestine depending on the severity of involvement or can manifest as multiple organ failure ranging from shock, sepsis, and even death.

Chronic PVT can be asymptomatic and can be detected incidentally on imaging methods. Collateral development (portal cavernous) occurs around the thrombotic portal vein. In these patients, signs of portal hypertension such as splenomegaly, esophageal varices, anemia, and thrombocytopenia can be detected. Upper GI bleeding can be the first symptom in 20% - 40% of cases.

According to the AASLD guideline for the treatment of acute portal vein thrombosis, low molecular weight heparin followed by oral anticoagulant drug. Duration depends on the underlying cause from 3 - 6 months or more. In the case of chronic Portal vein thrombosis, there is no real consensus on the use of anticoagulation. It may be an option in patients with documented persistent prothrombotic states [11]. All patients should be screened for esophageal varices and treatments like variceal ligation, prophylactic beta-blocker, and TIPS all are recommended.

5. Conclusion

We emphasize the need for timely diagnosis and appropriate treatment of PVT. Since it is a rare cause of abdominal pain it may present with lethal complications such as mesenteric ischemia and infarction.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Acosta, S., Alhadad, A., Svensson, P. and Ekberg, O. (2008) Epidemiology, Risk and Prognostic Factors in Mesenteric Venous Thrombosis. *British Journal of Surgery*, **95**, 1245-1251. https://doi.org/10.1002/bjs.6319

[2] Abdu, R.A., Zakhour, B.J. and Dallis, D.J. (1987) Mesenteric Venous Thrombosis—1911 to 1984. *Surgery*, **101**, 383-388.

[3] Rhee, R.Y., Gloviczki, P., Mendonca, C.T., Petterson, T.M., Serry, R.D., Sarr, M.G., *et al.* (1994) Mesenteric Venous Thrombosis: Still a Lethal Disease in the 1990s. *Journal of Vascular Surgery*, **20**, 688-697. https://doi.org/10.1016/S0741-5214(94)70155-5

[4] Rosendaal, F.R. (1999) Venous Thrombosis: A Multicausal Disease. *Lancet*, **353**, 1167-1173. https://doi.org/10.1016/S0140-6736(98)10266-0

[5] Sacerdoti, D., Serianni, G., Gaiani, S., Bolognesi, M., Bombonato, G. and Gatta, A. (2007) Thrombosis of the Portal Venous System. *Journal of Ultrasound*, **10**, 12-21. https://doi.org/10.1016/j.jus.2007.02.007

[6] Seedial, S.M., Mouli, S.K. and Desai, K.R. (2018) Acute Portal Vein Thrombosis: Current Trends in Medical and Endovascular Management. *Seminars in Interventional Radiology*, **35**, 198-202. https://doi.org/10.1055/s-0038-1660798

[7] Valla, D.C. and Condat, B. (2000) Portal Vein Thrombosis in Adults: Pathophysiology, Pathogenesis and Management. *Journal of Hepatology*, **32**, 865-871. https://doi.org/10.1016/S0168-8278(00)80259-7

[8] Van Gansbeke, D., Avni, E.F., Delcour, C., *et al.* (1985) Sonographic Features of Portal Vein Thrombosis. *American Journal of Roentgenology*, **144**, 661-867. https://doi.org/10.2214/ajr.144.4.749

[9] Samant, H., Asafo-Agyei, K.O. and Garfield, K. (2023) Portal Vein Thrombosis. In: *StatPearls,* StatPearls Publishing, Treasure Island. https://www.ncbi.nlm.nih.gov/books/NBK534157/

[10] Condat, B., Pessione, F., Helene Denninger, M., *et al.* (2000) Recent Portal or Mesenteric Venous Thrombosis: Increased Recognition and Frequent Recanalization on Anticoagulant Therapy. *Hepatology*, **32**, 466-470. https://doi.org/10.1053/jhep.2000.16597

[11] Plessier, A., Murad, S.D., Hernandez-Guerra, M., *et al.* (2007) A Prospective Multicentric Follow-Up Study on 105 Patients with Acute Portal Vein Thrombosis (PVT): Results from the European Network for Vascular Disorders of the Liver (EN-Vie). *Hepatology*, **46**, Article No. 310A. https://doi.org/10.1016/S0168-8278(07)61601-8

Abbreviation

PVT—Portal Vein Thrombosis

US—Ultrasonography

SMV—Superior Mesenteric Vein

CT—Computed Tomography