British Journal of Hospital Medicine

Br. J. Hosp. Med. (Lond) **2026**; 87(11): 51661
https://doi.org/10.31083/BJHM51661

Case Report

Progression From Acute Extensive Portal Vein Thrombosis to Portal Cavernoma With Varices in a Patient With Myelofibrosis: A Case Report

Chenghao Wang[1,2,†], Junxiu Chen[1,2,†], Ran Wang[1], Dongyang Wang[1], Jiahao Xu[1,3], Xingshun Qi[1,2,3,*]

[1]Department of Gastroenterology, General Hospital of Northern Theater Command (Teaching Hospital of Shenyang Pharmaceutical University), 110840 Shenyang, Liaoning, China
[2]Department of Clinical Pharmacy, Shenyang Pharmaceutical University, 110016 Shenyang, Liaoning, China
[3]Postgraduate College, China Medical University, 110001 Shenyang, Liaoning, China
*Correspondence: xingshunqi@126.com (Xingshun Qi)
[†]These authors contributed equally.
Academic Editor: Kassem Sharif
Submitted: 9 March 2026 Revised: 23 June 2026 Accepted: 7 August 2026 Published: 20 November 2026

Abstract

Aims/Background: Portal vein thrombosis refers to thrombosis in the main portal vein and/or its branches, with or without thrombosis in the mesenteric vein and splenic vein. Myeloproliferative neoplasms progress slowly and are often accompanied by hepatosplenomegaly with increased risk of bleeding and thrombosis. Myeloproliferative neoplasms, including myelofibrosis, are the primary cause of non-cirrhotic portal vein thrombosis, and in turn, portal vein thrombosis is usually the initial manifestation of myeloproliferative neoplasms. Herein, we report a case of myelofibrosis complicated by acute extensive portal vein thrombosis, which aims to highlight the importance of achieving portal vein recanalization in patients with non-cirrhotic portal vein thrombosis, as well as the prevention and management of portal hypertension-related complications in the absence of portal vein recanalization. **Case Presentation:** A 44-year-old female who presented with acute and severe abdominal pain for 7 days was admitted to the General Hospital of Northern Theater Command. She was diagnosed with thrombosis in the main portal vein and its branches and the superior mesenteric vein by contrast-enhanced computed tomography, and finally progressed to portal cavernoma with varices. **Results:** After anticoagulation combined with thrombolytic therapy, abdominal pain disappeared, but portal vein thrombosis was not significantly improved. During the follow-up period, the patient received long-term anticoagulation with oral rivaroxaban, but still progressed to portal cavernoma with varices, and subsequently underwent endoscopic and pharmacologic therapy. **Conclusion:** It is necessary to achieve portal vein recanalization in patients with non-cirrhotic portal vein thrombosis as soon as possible. If portal vein recanalization fails, more attention should be paid to the management of pre-hepatic portal hypertension-related complications.

Keywords: portal vein thrombosis; myeloproliferative neoplasm; anticoagulation; thrombolysis; case report

1. Introduction

Portal vein thrombosis (PVT) refers to the formation of thrombosis in the main portal vein and/or its branches, with or without thrombosis in the mesenteric vein and splenic vein [1]. PVT is rare in non-cirrhotic patients [2]. Its estimated incidence is only 2 to 4 cases per 100,000 people each year, but its mortality increased by 3.25% annually from 2018 to 2024 in the United States [3]. Virchow's triad, including hypercoagulability, vessel wall injury, and decreased venous flow, is the primary pathogenesis of PVT [2]. Myeloproliferative neoplasms (MPN), including myelofibrosis, are the primary cause of non-cirrhotic PVT [4,5]. Doppler ultrasound is always the initial imaging screening method for PVT, and contrast-enhanced CT and MRI scans are used to confirm the diagnosis of PVT [2]. The critical goal of the management of PVT is to achieve portal vein recanalization. Anticoagulation can significantly improve the recanalization of portal vein or mesenteric vein thrombosis. Thrombolytic therapy can be considered in patients with recent and extensive PVT when anticoagulation shows poor efficacy [6]. Failure to achieve timely recanalization of PVT predisposes to the development of portal hypertension-related complications, such as esophagogastric variceal bleeding (EGVB) and ascites, thereby significantly increasing the risk of adverse outcomes. Herein, we report a case of MPN with acute extensive portal vein thrombosis that was not recanalized after anticoagulation combined with thrombolytic therapy, but eventually progressed to cavernous transformation of the portal vein (CTPV) with varices. This case was prepared following the case report (CARE) framework (**Supplementary Material**).

2. Case Report

A 44-year-old female who presented with acute and severe abdominal pain for 7 days was admitted to the Gen-

Copyright: © 2026 The Author(s). Published by IMR Press.
This is an open access article under the CC BY 4.0 license.

Publisher's Note: IMR Press stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

eral Hospital of Northern Theater Command on June 20, 2023. On June 18, she underwent abdominal contrast-enhanced computed tomography (CT) scans at another hospital, which indicated thrombosis in the main portal vein and its branches and the superior mesenteric vein. Her family history and past medical history were unremarkable, and she had neither a history of alcohol consumption nor smoking. After admission, the physical examination showed that the abdomen was soft, with tenderness, but without rebound tenderness. Her height is 158 cm and weight is 46 kg, and her vital signs were as follows: temperature: 36.5 ℃, blood pressure: 133/74 mmHg, heart rate: 74 beats/minute, and respiratory rate: 20 breaths/minute. Laboratory results indicated that interleukin-6 (IL-6) was 15.60 pg/mL (reference range: 0–6.4 pg/mL), procalcitonin (PCT) was 0.13 ng/mL (reference range: 0–0.05 ng/mL), white blood cell count (WBC) was 10.4 × 10^9/L (reference range: 4.0–10.0 × 10^9/L), neutrophil percentage (NE%) was 86.9% (reference range: 40–75%), D-dimer was 1.11 µg/mL (reference range: 0–0.55 µg/mL), fibrinogen (FIB) was 4.06 g/L (reference range: 2–4 g/L), protein C (PC) was 52% (reference range: 70–140%), and protein S (PS) was 40.7% (reference range: 52–118%). Thromboelastogram (TEG) showed that angle (α) was 78.4 degrees (reference range: 53–72 degrees) and maximum amplitude (MA) was 73.2 mm (reference range: 50–70 mm). Based on these findings, she was diagnosed with acute extensive portal vein thrombosis. Her platelet count, INR, liver function, and renal function were normal. Thus, on June 20, low-molecular-weight heparin (LMWH) sodium injection was administered at a dosage of 5000 IU subcutaneously every 12 hours. Then, the PCR test was conducted to clarify a suspected diagnosis of MPN. On June 21, the JAK2 V617F mutation was positive.

On June 26, abdominal pain did not significantly subside, and contrast-enhanced CT scans revealed multiple thrombi in the main portal vein and its branches (Fig. 1). Simultaneously, the D-dimer level was 1.16 mg/L. Therefore, considering the poor response to anticoagulation therapy, urokinase was added for thrombolytic therapy on June 28. Then, abdominal pain alleviated. On July 5, her abdominal pain disappeared. Follow-up CT examination indicated that thrombosis in the portal vein system was not significantly improved compared to prior imaging (Fig. 2). After discharge, she took long-term rivaroxaban.

In July 2023, the patient was definitely diagnosed with myelofibrosis by bone marrow aspiration at another hospital, and then received interferon. According to the recommendation of hematologists, interferon was discontinued in March 2025.

On June 19, 2025, she was readmitted to our hospital owing to abdominal distension for a month, accompanied by lower extremity edema. Physical examination showed increased abdominal circumference. Her vital signs were as follows: temperature: 36.5 ℃, blood pressure: 113/75 mmHg, heart rate: 100 beats/minute, and respiratory rate: 18 breaths/minute. Abdominal contrast-enhanced CT scans indicated thrombosis in the main portal vein and its branches, accompanied by CTPV and ascites (Fig. 3). Then, furosemide combined with spironolactone was given. On June 22, laboratory tests showed that hemoglobin (Hb) was 37 g/L (reference range: 120–160 g/L), red blood cell (RBC) was 1.54 × 10^{12}/L (reference range: 3.5–5.5 × 10^{12}/L), WBC was 3.4 × 10^9/L, platelet count (PLT) was 92 × 10^9/L (reference range: 125–350 × 10^9/L), hematocrit was 0.121 L/L (reference range: 0.37–0.48 L/L), D-dimer was 1.76 mg/L, FIB was 6.76 g/L, and fecal occult blood was positive, but she denied any history of melena or hematemesis. Accordingly, severe anemia was diagnosed. Therefore, packed red blood cells and sucrose iron infusion were given. On June 26, endoscopy indicated multiple varices in the middle and upper esophagus, gastric fundus, and upper gastric body, with focal vascular wall thinning and red color sign (RC+). According to the updated APASL guidelines and European Society of Gastrointestinal Endoscopy (ESGE) guideline [7,8], endoscopic variceal ligation is recommended for the esophageal varices and varices at the lesser curvature of the cardia. Thus, endoscopic variceal ligation (EVL) was performed for esophageal varices and varices at the lesser curvature of the cardia (Fig. 4). The patient had no discomfort after that. On July 2, hemoglobin was 71 g/L, and other parameters and abdominal distension also improved.

On September 10, 2025, the patient was readmitted to our hospital due to fatigue for about one week. Laboratory tests indicated that WBC was 1.8 × 10^9/L, RBC was 2.74 × 10^{12}/L, Hb was 54 g/L, PLT was 72 × 10^9/L, and serum albumin level was 34.4 g/L (reference range: 40–55 g/L). Packed red blood cells were transfused on September 11. On September 12, endoscopy demonstrated severe esophagogastric varices (Fig. 5), and then, EVL was performed for esophageal varices. On September 18, laboratory tests showed that WBC was 1.9 × 10^9/L, RBC was 3.34 × 10^{12}/L, PLT was 66 × 10^9/L, and Hb was 69 g/L. Fatigue alleviated, and then the patient was discharged.

On May 12, 2026, she was followed by telephone, stating that ascites recurred.

3. Discussion

Our patient was positive for the JAK2 V617F mutation and diagnosed with MPN, which is the primary cause of non-cirrhotic PVT [5]. JAK2 V617F mutation is closely associated with MPN, which can lead to abnormal changes in various blood cells, as well as the structure and function of endothelial cells. Consequently, it results in increased cellular aggregation and adhesion and endothelial cell activation, thereby increasing the risk of PVT [9]. Additionally, antithrombin, PC, and PS deficiency is an important factor contributing to thrombosis [10]. Although the patient showed only mild decreases in PC and PS levels, they may be secondary to acute liver injury. Thus, they cannot be considered the etiology of PVT.

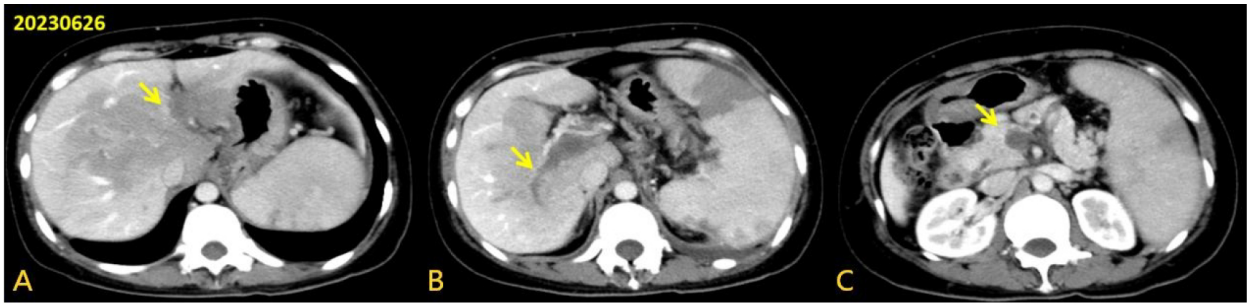


Fig. 1. Abdominal contrast-enhanced computed tomography (CT) scans on June 26, 2023. (A) Thrombosis within the left portal vein branch (yellow arrow). (B) Thrombosis within the right portal vein branch (yellow arrow). (C) Thrombosis within the superior mesenteric vein (yellow arrow).

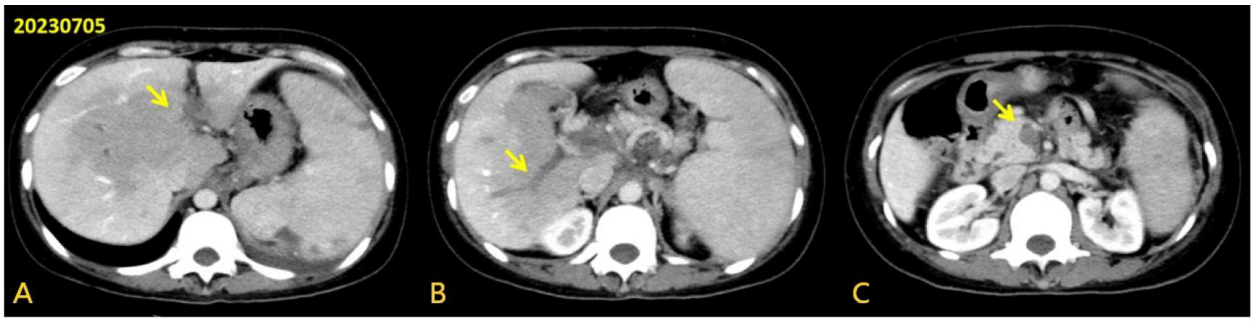


Fig. 2. Abdominal contrast-enhanced CT scans on July 5, 2023. (A) Thrombosis within the left portal vein branch (yellow arrow). (B) Thrombosis within the right portal vein branch (yellow arrow). (C) Thrombosis within the superior mesenteric vein (yellow arrow).

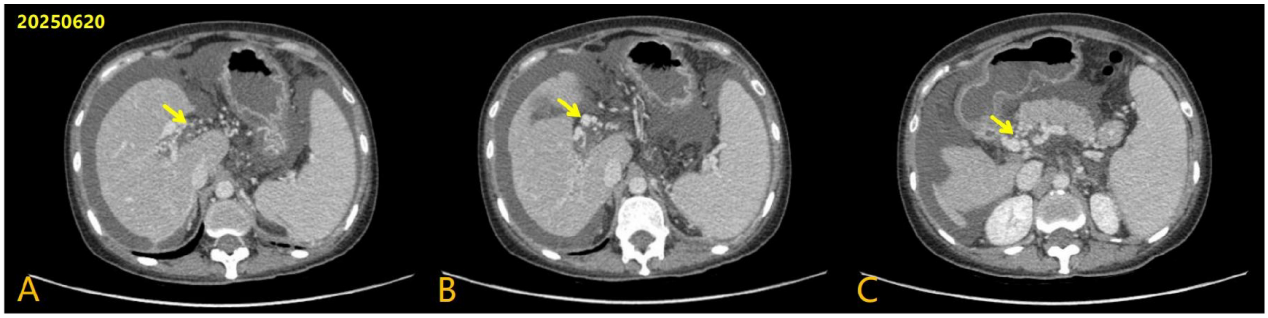


Fig. 3. Abdominal contrast-enhanced CT scans on June 20, 2025. (A–C) Cavernous collateral vessels around the main portal vein (yellow arrow).

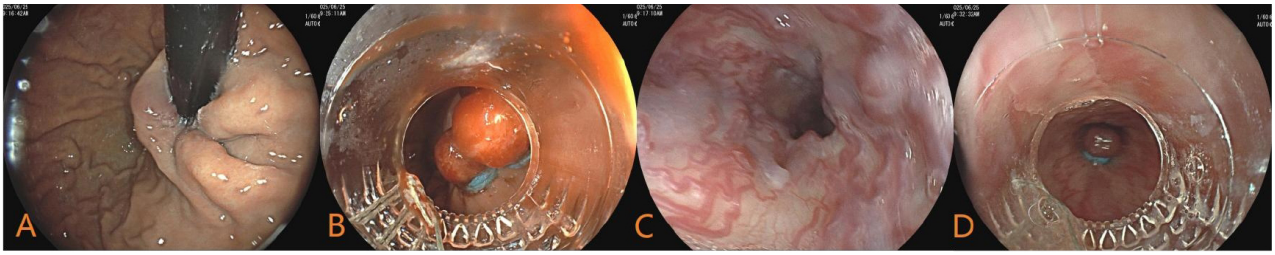


Fig. 4. Endoscopy on June 26, 2025. (A) Severe gastric varices on endoscopy. (B) Endoscopic variceal ligation was performed for gastric varices. (C) Severe esophageal varices on endoscopy. (D) Endoscopic variceal ligation was performed for esophageal varices.

Achieving portal vein recanalization is a key goal in the management of PVT. Compared with non-anticoagulation, anticoagulation can significantly improve the recanalization of portal vein or mesenteric vein throm-

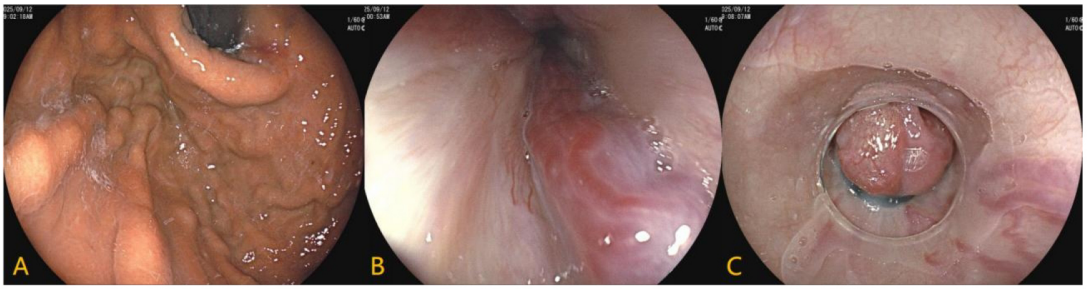


Fig. 5. Endoscopy on September 12, 2025. (A) Gastric fundal varices on endoscopy. (B) Severe esophageal varices on endoscopy. (C) Endoscopic variceal ligation.

bosis, without increasing the risk or severity of bleeding in non-cirrhotic PVT patients [11]. Therefore, anticoagulation was started promptly after admission. For patients with recent extensive PVST and intestinal ischaemia unresponsive to anticoagulation, thrombolysis can be considered [12]. Our patient's D-dimer level remained elevated, and thrombus burden and clinical symptoms did not significantly improve in spite of anticoagulation therapy. Therefore, thrombolytic therapy was initiated. Streptokinase, urokinase, and tissue plasminogen activator are common choices for thrombolysis [13]. Urokinase, which activates plasminogen to generate plasmin and degrades fibrin to dissolve thrombi, was administered in our patient, considering that anticoagulation therapy did not significantly improve PVT [14]. Unfortunately, there was no recanalization of PVT in spite of anticoagulation and thrombolysis.

CTPV refers to the formation of cavernous collateral vessels around the portal vein after the portal vein is partially or completely occluded. It can lead to an increase in resistance to blood flow within the portal venous circulation, and ultimately result in portal hypertension. Esophagogastric varices is a common complication of CTPV with a high risk of mortality [7]. Therefore, the management of variceal bleeding is very vital. Combined endoscopic and pharmacologic therapy is the first-line intervention for esophagogastric variceal bleeding [7,15]. Our patient was diagnosed with severe esophagogastric varices accompanied by a high risk of bleeding after admission, and thus, EVL was performed. During follow-up, she was readmitted with severe fatigue, and was found to have severe esophagogastric varices requiring EVL.

Ascites is an independent prognostic factor in patients with non-cirrhotic PVT. The survival rate is significantly lower in patients with ascites than in those without [15]. Specifically, the cumulative 5- and 10-year survival rates in patients with and without ascites are 83% vs 95% and 42% vs 87% [16]. In our patient, ascites was diagnosed at the second admission, suggesting poor prognosis.

Advantages and Limitations

In our case, some clinically significant issues are summarized. First, this case outlines the whole course of disease progression from acute PVT to portal cavernoma with pre-hepatic portal hypertension-related complications. Second, this case highlights the importance of timely recanalization of occlusive PVT on the outcomes. Third, this case also emphasizes the necessity of early initiation of anticoagulant and thrombolytic therapy. Fourth, the algorithm regarding management of portal hypertension secondary to extrahepatic portal vein obstruction, which is a rare vascular disorder of the liver, should be further explored. However, our case has several limitations. First, some clinical data may be incomplete or missing. Second, at the last follow-up visit, comprehensive endoscopic and imaging reassessment was not performed.

4. Conclusion

Achieving portal vein recanalization is a primary therapeutic goal. As demonstrated in the present case, PVT recanalization may not always be achieved despite anticoagulation combined with thrombolysis. In such cases, subsequent management should focus on the prevention, surveillance, and treatment of complications related to prehepatic portal hypertension, particularly esophagogastric variceal bleeding and ascites. Regular endoscopic surveillance for esophagogastric varices, management of ascites, and individualized long-term anticoagulation after careful assessment of thrombotic and bleeding risks are essential for improving long-term outcomes.

Learning Points

• MPN, including myelofibrosis, is the primary cause of non-cirrhotic PVT.

• If acute PVT is not recanalized, it would progress to portal cavernoma with a high risk of pre-hepatic portal hypertension-related complications.

• Occurrence of ascites often suggests poor outcomes in patients with non-cirrhotic PVT.

Availability of Data and Materials

All data included in this study are available upon request by contacting the corresponding author.

Author Contributions

CW: Study design, literature search and drafting of the original manuscript; JC: Critical revision of the manuscript, analysis of clinical data and interpretation of figures; RW and DW: Patient management, clinical follow-up, acquisition of clinical and imaging data, figure preparation; JX: Acquisition of imaging data, analysis of radiological findings and figure preparation; XQ: Conception and supervision of the study, critical revision of the manuscript, endoscopic therapy, patient management. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This work was approved by the medical ethics committee of the General Hospital of Northern Theater Command [Y (2026)55] and strictly adhered to the Declaration of Helsinki. Informed consent was obtained from the patient for the publication of their information and imaging.

Acknowledgment

Not applicable.

Funding

This work was supported by the Joint Program of Science and Technology Program of Liaoning Province (2024JH2/102600269).

Conflicts of Interest

Given his role as an Editorial Board member, Xingshun Qi had no involvement in the peer review or editorial decision-making process for this article and had no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to Kassem Sharif. The other authors declare no conflicts of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/BJHM51661.

References

[1] Gao F, Wang R, Han L, Zhang R, Qi X. Early thrombolysis combined with anticoagulation and antibiotics for acute portal venous system thrombosis secondary to intra-abdominal infection. Archives of Medical Science: AMS. 2023; 19: 283–287. https://doi.org/10.5114/aoms/159083

[2] Elkrief L, Hernandez-Gea V, Senzolo M, Albillos A, Baiges A, Berzigotti A, et al. Portal vein thrombosis: diagnosis, management, and endpoints for future clinical studies. The Lancet. Gastroenterology & Hepatology. 2024; 9: 859–883. https://doi.org/10.1016/S2468-1253(24)00155-9

[3] Mattiuzzi C, Lippi G. Trends and demographic patterns of portal vein thrombosis mortality in the United States, 2018-2024. Blood Coagulation & Fibrinolysis: an International Journal in Haemostasis and Thrombosis. 2026; 37: 44–45. https://doi.org/10.1097/MBC.0000000000001398

[4] Elkrief L, Payancé A, Plessier A, d'Alteroche L, Ronot M, Paradis V, et al. Management of splanchnic vein thrombosis. JHEP Reports: Innovation in Hepatology. 2023; 5: 100667. https://doi.org/10.1016/j.jhepr.2022.100667

[5] Willington AJ, Tripathi D. Current concepts in the management of non-cirrhotic non-malignant portal vein thrombosis. World Journal of Hepatology. 2024; 16: 751–765. https://doi.org/10.4254/wjh.v16.i5.751

[6] Gao F, Wang L, Pan J, Yin Y, Wang J, Xu X, et al. Efficacy and safety of thrombolytic therapy for portal venous system thrombosis: A systematic review and meta-analysis. Journal of Internal Medicine. 2023; 293: 212–227. https://doi.org/10.1111/joim.13575

[7] Lesmana CRA, Shukla A, Kumar A, Shalimar, Qi X, Gani RA, et al. Management of acute variceal bleeding: updated APASL guidelines. Hepatology International. 2025; 19: 1003–1031. https://doi.org/10.1007/s12072-025-10894-4

[8] Gralnek IM, Camus Duboc M, Garcia-Pagan JC, Fuccio L, Karstensen JG, Hucl T, et al. Endoscopic diagnosis and management of esophagogastric variceal hemorrhage: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. Endoscopy. 2022; 54: 1094–1120. https://doi.org/10.1055/a-1939-4887

[9] Bhuria V, Baldauf CK, Schraven B, Fischer T. Thromboinflammation in Myeloproliferative Neoplasms (MPN)-A Puzzle Still to Be Solved. International Journal of Molecular Sciences. 2022; 23: 3206. https://doi.org/10.3390/ijms23063206

[10] Arachchillage DJ, Mackillop L, Chandratheva A, Motawani J, MacCallum P, Laffan M. Thrombophilia testing: A British Society for Haematology guideline. British Journal of Haematology. 2022; 198: 443–458. https://doi.org/10.1111/bjh.18239

[11] Naymagon L, Tremblay D, Zubizarreta N, Moshier E, Troy K, Schiano T, et al. The efficacy and safety of direct oral anticoagulants in noncirrhotic portal vein thrombosis. Blood Advances. 2020; 4: 655–666. https://doi.org/10.1182/bloodadvances.2019001310

[12] Di Nisio M, Valeriani E, Riva N, Schulman S, Beyer-Westendorf J, Ageno W. Anticoagulant therapy for splanchnic vein thrombosis: ISTH SSC Subcommittee Control of Anticoagulation. Journal of Thrombosis and Haemostasis: JTH. 2020; 18: 1562–1568. https://doi.org/10.1111/jth.14836

[13] Rashedi S, Leyva H, Hamade N, Pfeferman MB, Ortega-Paz L, Sadeghipour P, et al. Fibrinolytic Therapy for Thromboembolic Diseases: Approved Indications and Future Directions. Journal of the American College of Cardiology. 2025; 86: 1065–1087. https://doi.org/10.1016/j.jacc.2025.07.061

[14] Ghasemi SH, Ahmadian MT, Assempour A, Ahmadi Tafti SH. Comparative study on efficacy of thrombolytic protocols: Dual therapy against standard tPA regimen. Biophysical Journal. 2025; 124: 1704–1714. https://doi.org/10.1016/j.bpj.2025.04.013

[15] Qi XS, Bai M, He CY, Yin ZX, Guo WG, Niu J, et al. Prognostic factors in non-malignant and non-cirrhotic patients with portal cavernoma: an 8-year retrospective single-center study. World Journal of Gastroenterology. 2013; 19: 7447–7454. https://doi.org/10.3748/wjg.v19.i42.7447

[16] Qi X. Portal Vein Thrombosis: Recent Advance. Advances in Experimental Medicine and Biology. 2017; 906: 229–239. https://doi.org/10.1007/5584_2016_118