EJCRIM
European Journal
of Case Reports
in Internal Medicine

A CASE OF PORTAL VEIN THROMBOSIS DUE TO CHOLECYSTITIS: A 10-YEAR FOLLOW-UP CASE REPORT

Tetsushi Azami, Yuichi Takano, Kozue Ito, Naoki Tamai, Jun Noda, Fumitaka Niiya, Masatsugu Nagahama

Division of Gastroenterology, Department of Medicine, Showa Medical University Fujigaoka Hospital, Yokohama, Japan

Corresponding author's e-mail: tetsushiazami@yahoo.co.jp

Received: 30/09/2025
Accepted: 07/10/2025
Published: 20/11/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent to publish his clinical history and any accompanying images was obtained from the patient.
This article is licensed under a Commons Attribution Non-Commercial 4.0 License

How to cite this article: Azami T, Takano Y, Ito K, Tamai N, Noda J, Niiya F, Nagahama M. A case of portal vein thrombosis due to cholecystitis: a 10-year follow-up case report. *EJCRIM* 2025;**12**:doi:10.12890/2025_005873

ABSTRACT

We present a rare case of portal vein thrombosis (PVT) secondary to acute cholecystitis with a 10-year follow-up. A 62-year-old man presented with fever and epigastric pain and was diagnosed with acute cholecystitis. Cholecystitis is a rare local intra-abdominal cause of PVT. After antibiotic treatment, jaundice developed, and imaging revealed left PVT. Anticoagulation was initiated, but thrombosis persisted. Despite the persistent thrombus, long-term follow-up revealed no portal hypertension; however, atrophy of the left lobe of the liver was observed. This case highlights the key aspects of the long-term natural history of cholecystitis-associated PVT and underscores the importance of timely diagnosis and management.

KEYWORDS

Portal vein thrombosis, cholecystitis, hepatic atrophy

LEARNING POINTS

- Cholecystitis can be a cause of acute portal vein thrombosis.
- Early anticoagulation therapy is crucial for thrombus resolution.
- Portal vein thrombosis may lead to localized hepatic atrophy over the long term.

INTRODUCTION

Portal vein thrombosis (PVT) is widely recognized to be caused by liver cirrhosis and systemic diseases such as hematologic disorders. However, local inflammatory factors such as cholecystitis can also cause PVT, and several case reports have been documented. Nevertheless, reports on the long-term prognosis of PVT associated with cholecystitis are extremely limited, and many aspects of its natural history remain unclear. Herein, we report a rare case of PVT due to cholecystitis in which the thrombus persisted despite anticoagulant therapy, and we were able to follow the patient for 10 years.

CASE DESCRIPTION

A 62-year-old man presented at a local clinic with fever and epigastric pain. His medical history included surgery (distal gastrectomy and Billroth II reconstruction) for duodenal ulcer at age 51. Blood tests and a non-contrast

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

© EFIM 2025 - European Journal of Case Reports in Internal Medicine - Doi: 10.12890/2025_005873

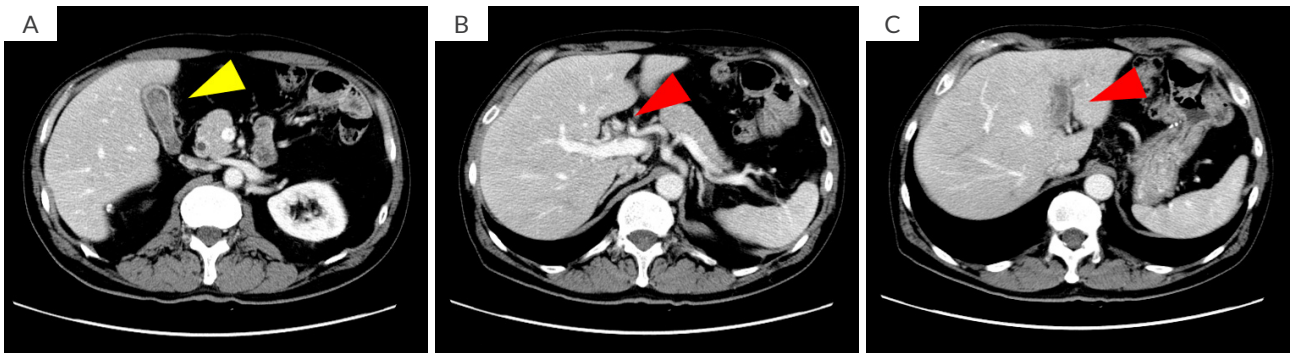


Figure 1. A) Contrast-enhanced computed tomography revealing thickening of the gallbladder wall and increased density of the surrounding fatty tissue (yellow arrowhead); B, C) A thrombus is observed in the left branch of the portal vein (red arrowhead).

computed tomography (CT) led to the diagnosis of moderate acute cholecystitis, and he was admitted for conservative management. After improvement in abdominal pain and inflammatory marker levels with fasting and antibiotic therapy (cefoperazone/sulbactam), the patient was discharged. No medications were prescribed at discharge. However, 20 days after symptom onset, he developed jaundice and elevated hepatobiliary enzyme levels, suggesting the presence of cholangitis, and was referred to our hospital, despite the absence of liver dysfunction during his prior admission.

On admission, blood tests revealed levels of total bilirubin, direct bilirubin, aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase of 6.0 mg/dl, 4.7 mg/dl, 79 U/l, 99 U/l, and 860 U/l, respectively, indicating hepatobiliary dysfunction. Coagulation parameters, including prothrombin time; activated partial thromboplastin time; and antithrombin III, protein C, and protein S levels, were within normal limits.

Contrast-enhanced CT revealed gallbladder wall thickening with collapse, but no biliary ductal abnormalities. However, a thrombus was identified in a branch of the left portal vein *(Fig. 1)*. Magnetic resonance cholangiopancreatography similarly showed no biliary obstruction *(Fig. 2)*. Liver dysfunction was diagnosed as a consequence of PVT, and anticoagulation therapy was initiated with danaparoid on day 1, followed by continuous intravenous heparin for 11 days and warfarin on day 7. Jaundice and liver enzyme elevations gradually improved, and the patient was discharged on day 12 (32 days after the initial symptom onset).

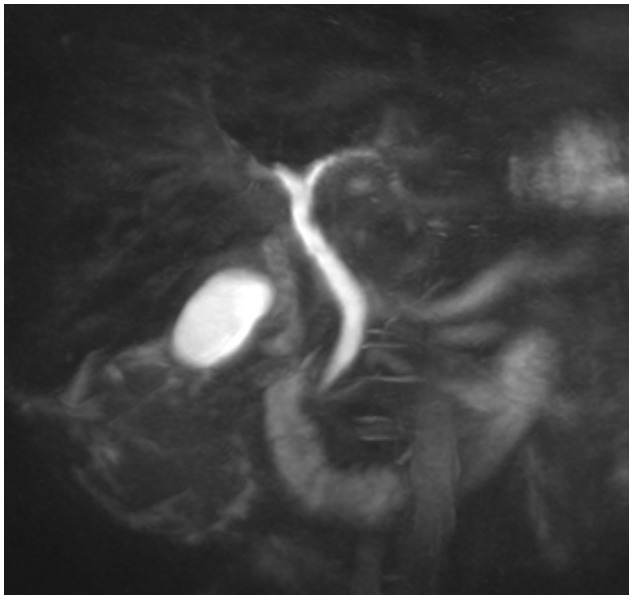


Figure 2. Magnetic resonance cholangiopancreatography reveals no abnormalities in the bile ducts.

Follow-up CT confirmed the persistence of the thrombus, although hepatic function continued to recover. Three months later, the patient underwent elective cholecystectomy, with no evidence of malignancy upon pathological examination. Postoperatively, warfarin was continued; however, the thrombus persisted, suggesting that it had become organized, and anticoagulation was discontinued after 1 year. Over the subsequent 10-year follow-up, atrophy of the left lobe of the liver was noted *(Fig. 3)*. Still, no signs of portal hypertension, such as esophagogastric varices or ascites, or abnormal liver function were observed.

DISCUSSION

PVT was classified based on the presence or absence of underlying cirrhosis. In non-cirrhotic cases, the contributing factors are broadly categorized as systemic (e.g., myeloproliferative neoplasms, antiphospholipid antibody syndrome, and prothrombin gene mutations) or local (e.g., intra-abdominal infections, trauma, malignancy, and surgery), often in combination. Non-cirrhotic PVT typically presents acutely, with anticoagulation as the mainstay of treatment, and early initiation improves recanalization rates by approximately 40–45%[1].

Cholecystitis represents a rare local intra-abdominal cause of PVT. *Table 1* summarizes previous reports of cholecystitis-associated PVT[2–9], including the present case. Among the nine detailed cases, the median age was 50 years (range 31–77 years), with seven male and two female patients. The thrombus locations included the main trunk (n = 4), left branch (n = 4), and right branch (n = 2), with the thrombus extending from the main trunk to the right branch in one patient. Liver dysfunction was observed in 89% of cases (n = 8). Anticoagulation was administered in all cases; recanalization was successful in three, unsuccessful in three, and unknown in three.

In this patient, the thrombus was confined to a branch of

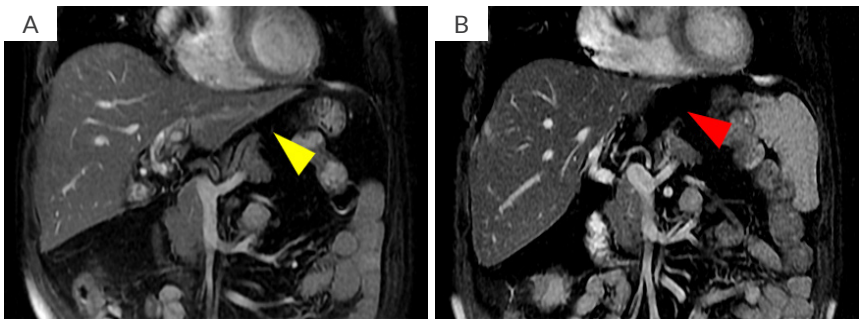


Figure 3. A) Initial magnetic resonance imaging (MRI) using fast imaging employing steady-state acquisition (FIESTA) reveals preserved volume of the left hepatic lobe (yellow arrowhead); B) MRI FIESTA performed 10 years after onset reveals atrophy of the left hepatic lobe (red arrowhead).

Reference	Age (years)	Sex	Severity of cholecystitis	PVT location	Liver dysfunction	Time from symptom onset to diagnosis (days)	Anticoagulant therapy	Dissolution of thrombus
Our case	62	M	Mild	Left branch	Yes	20	Danaparoid sodium, heparin, warfarin	Failure
Faisal et al. 2025[2]	44	M	Moderate	Left branch	Yes	2	Heparin, enoxaparin	N/A
Guevara et al. 2022[3]	45	M	Mild-moderate	Main trunk, right branch	Yes	9	Acenocoumarol, aspirin	Success
Kushiro et al. 2021[4]	77	F	Mild-moderate	Main trunk	Yes	54	Rivaroxaban	Failure
Rivera-Alonso et al. 2021[5]	51	M	Mild	Left branch	Yes	11	N/A	Success
Chaudhary et al. 2018[6]	41	M	Mild	Main trunk	No	16	Enoxaparin, warfarin	Success
Filkins et al. 2017[7]	50	M	Mild-moderate	Main trunk	Yes	N/A	N/A	N/A
Muneer et al. 2015[8]	31	M	Mild-moderate	Right branch	Yes	7	N/A	N/A
Kalangi et al. 2014[9]	66	F	Moderate	Left branch	Yes	30	Apixaban	Failure

Table 1. Summary of previous reports.

the left portal vein. The mechanisms of venous thrombosis include hypercoagulability, endothelial injury, and venous stasis. The inflammatory cytokines released during cholecystitis can induce a hypercoagulable state, whereas perivascular oedema and compression can contribute to stasis and endothelial injury. Yoshimitsu et al. reported that the cystic vein predominantly drains into the S5 (96%) and S4 (93%) branches of the portal vein[10], suggesting that the thrombus in our case may have originated in the cystic vein and propagated via the S4 branch to the left portal vein.

In this case, liver dysfunction was observed. Alternative causes, such as common bile duct stones and drug-induced liver injury, were considered but ruled out based on imaging findings and medication history. In previously reported cases of cholecystitis-associated PVT, liver dysfunction was documented in the majority of patients, even when the thrombus was confined to a portal vein branch *(Table 1)*. It remains unclear whether the thrombus itself directly caused liver dysfunction, or whether factors such as cholestasis and systemic inflammation associated with cholecystitis acted as predisposing factors for PVT. Nevertheless, when liver dysfunction is encountered during the course of cholecystitis, the possibility of PVT should be taken into consideration and appropriately investigated.

Despite anticoagulation therapy, recanalization was not achieved. Excluding patients with unknown outcomes, the recanalization success rate was 50% (3/6). In previous reports, unsuccessful cases were characterized by delayed treatment initiation (e.g., at 54 or 30 days). In our case, anticoagulation therapy began 20 days after symptom onset, and this delay may have contributed to the persistent thrombus. In cases of cholecystitis, in addition to assessing disease severity, evaluation of portal vein blood flow (Doppler ultrasound or contrast CT) should be considered whenever possible.

In this case, the thrombus was confined to a branch of the left portal vein; therefore, portal hypertension did not develop. However, localized atrophy of the left lobe of the liver was observed, likely due to chronic reduction of portal blood flow. Long-term follow-up of cholecystitis-associated PVT is rare. This study provided valuable clinical insights.

In conclusion, although rare, cholecystitis is an important cause of PVT. Even with anticoagulation therapy, delayed initiation of treatment may result in persistent thrombus formation and subsequent localized hepatic atrophy. This case highlights the key aspects of the long-term natural history of cholecystitis-associated PVT and underscores the importance of timely diagnosis and management.

REFERENCES

1. Intagliata NM, Caldwell SH, Tripodi A. Diagnosis, development, and treatment of portal vein thrombosis in patients with and without cirrhosis. *Gastroenterology* 2019;**156**:1582–1599. doi: 10.1053/j.gastro.2019.01.265
2. Almudaiheem FA, Aljabali M, Ali B, Alanazi M. Left portal vein thrombosis as an unusual complication following acute cholecystitis: case report and literature review. *J Surg Case Rep* 2025;**2025**:rjaf118. doi: 10.1093/jscr/rjaf118
3. Guevara S, Miyara SJ, Aronsohn J, Homsi JT, McCann-Molmenti A, Mumford JM, et al. COVID-19-associated portal vein thrombosis post-cholecystitis. *Int J Angiol* 2022;**32**:262–268. doi: 10.1055/s-0042-1743409
4. Kushiro S, Miyagami T, Kanai Y, Yang KS, Naito T. Delayed diagnosis of portal vein thrombosis due to premature closure of cholecystitis. *Am J Med* 2021;**134**:e543–545. doi: 10.1016/j.amjmed.2021.05.015
5. Rivera-Alonso D, Rivera-Alonso I, Burneo-Esteves M, Martinez-Ruiz C, Rojo-Abecia M, Moreno-Sanguino C. Acute portal vein thrombosis in mild cholecystitis. A consequence of coronavirus disease 2019 infection? *Cir Cir* 2021;**89**:399–402. doi: 10.24875/CIRU.20001044
6. Chaudhary HA, Yusuf Abubeker I, Mushtaq K, Obeidat K, Kartha A. Portomesenteric thrombosis secondary to acute cholecystitis: A case report. *Case Rep Gastrointest Med* 2018;**2018**:9409081. doi: 10.1155/2018/9409081
7. Filkins LM, Gaston DC, Mathison B, Smith T, Couturier MR, Murphy RD, et al. Biliary Strongyloides stercoralis with cholecystitis and extensive portal vein thrombosis. *Open Forum Infect Dis* 2017;**4**:ofx217. doi: 10.1093/ofid/ofx217
8. Muneer M, Abdelrahman H, El-Menyar A, Zarour A, Awad A, Al-Thani H. Acute cholecystitis complicated with portal vein thrombosis: A case report and literature review. *Am J Case Rep* 2015;**16**:627–630. doi: 10.12659/AJCR.894846
9. Kalangi H, Yancovitz SR, Camins B. A unique case of hypervirulent Klebsiella pneumoniae acute cholecystitis complicated by portal vein thrombophlebitis: A case report and literature review. *IDCases* 2024;**36**:e01935. doi: 10.1016/j.idcr.2024.e01935
10. Yoshimitsu K, Honda H, Kaneko K, Kuroiwa T, Irie H, Chijiiwa K, et al. Anatomy and clinical importance of cholecystic venous drainage: helical CT observations during injection of contrast medium into the cholecystic artery. *AJR Am J Roentgenol* 1997;**169**:505–510. doi: 10.2214/ajr.169.2.9242765