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of

Acute Cholecystitis	Complicated	with Port	al Vein
<b>Thrombosis: A Case</b>	<b>Report and</b>	Literature	Review

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Patient:	Male, 31				
Final Diagnosis:	Acute cholecystitis complicated with portal vein thrombosis				
Symptoms:	Abdominal discomfort • fever • vomiting				
Medication:	—				
<b>Clinical Procedure:</b>	Abdominal ultrasound and MRI				
Specialty:	Gastroenterology and Hepatology				
Objective:	Rare co-existance of disease or pathology				
Background:	Portal vein thrombosis (PVT) is an infrequent clinical condition usually associated with multiple etiological fac- tors and diseases. In some cases, PVT remains undiagnosed and is incidentally detected during routine exam- ination for a known etiology.				
Case Report:	Here we present a rare case of portal vein thrombosis associated with acute cholecystitis in a 31-year-old man				
Conclusions:	Conservative treatment may be a feasible and safe approach for the management of PVT with acute cholecys- titis, if treated at an early stage. Moreover, initial diagnosis based on radiological evaluation is possible only if the surgeons are familiar with this unusual condition. Therefore, a high index of suspicion is required for ear- ly diagnosis and management of patients with acute cholecystitis-associated PVT.				
MeSH Keywords:	Cholecystitis, Acute • Diagnosis • Portal Vein				
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# Background

Acute portal vein thrombosis (PVT) is an unusual clinical condition resulting from a combination of acquired, inherited, and local precipitating factors [1]. Although the diagnosis of inherited coagulation disorder does not influence the management of VTE, there is increased risk of recurrence in such patients with homozygotes gain-of-function mutations [2]. The most frequent local thrombotic risk factors are cholecystitis, cholangitis, appendicitis, splenectomy, pancreatitis, and liver cirrhosis or tumors [3]. The signs and symptoms of PVT are often nonspecific, and these patients mainly present with acute febrile illness and abdominal pain [4]. Unfortunately, abdominal pain is not considered as the primary indication of PVT; therefore, it might be neglected in some cases [5]. The use of Doppler ultrasonography and CT scan has improved the rate of radiological diagnosis of PVT [6], but PVT remains undiagnosed in some cases and is incidentally detected during routine examination for other reasons [7]. In such cases, the specific underlying pathological condition related with PVT manifested the initial clinical presentation, time course, and prognosis [8]. Particularly, the presentation of PVT with acute cholecystitis is an unusual event and only a few cases have been reported in the literature. Here, we present a rare case of portal vein thrombosis associated with acute cholecystitis.

jaundice. The laboratory findings revealed impaired liver function profile with high alanine aminotransferase (65 U/l), aspartate aminotransferase (65 U/l), total bilirubin (90 umol/l) and alkaline phosphatase (312 U/l). Anti-thrombin III was slightly raised at 122% (reference range: 80-120%). The coagulation profile was unremarkable, with no deficiency of protein C and S, negative finding for factor V Leiden, and complement component 3 (C3 and 4) was 156 and 51 mg/dl, respectively. Antismooth muscle antibody, anti-mitochondrial antibody, anti-nuclear antibody, anti-LKM antibody, and viral hepatitis screens were negative. The blood culture report was also negative. Our case had no history of splenectomy. Abdominal ultrasonography showed features of acute cholecystitis and right portal vein thrombosis. Moreover, the magnetic resonance imaging (MRI) study confirmed the diagnosis of PVT (Figures 1, 2). The patient was managed conservatively with intravenous antibiotics and anticoagulation. Within few days, the patient started recovering with normalization of the liver function profile and subsequently, he was discharged home on the 8th day post hospitalization.

## Discussion

The present case report describes a rare case of portal vein thrombosis (PVT) secondary to acute cholecystitis. The association of acute cholecystitis with PVT is considered relatively rare and is usually presented as case series and reports [9]. The pathogenesis of PVT is multifactorial and is primarily associated with liver or pancreatic malignancies, with an overall frequency of 21–24% [10]. In contrast, local thrombotic risk factors such as cholecystitis, cholangitis, appendicitis,

Figure 1. Axial T2 (A) and coronal T2 (B) images of the upper abdomen showing a thrombus involving the right portal vein (red arrows).

# **Case Report**

A 31-year-old man presented with right upper-quadrant abdominal pain, fever, and vomiting for 7 days but had no history of





Figure 2. Axial (A) and coronal (B) post-contrast T1 fat saturation images showing a filling defect involving the right portal vein and extending to its segmental branches with associated minor perfusion geographical changes.

Table 1. Summary of the literature review.

Authors	Year	Cases	Clinical presentation	Management
Harch et al. [16]	1987	1	Abdominal pain	Anticoagulation
Kidney et al. [17]	1998	1	Severe right upper quadrant and epigastric pain	Conservative management
Inoguchi et al. [18]	2004	1	Abdominal pain, nausea	Percutaneous transhepatic gallbladder drainage
Choi et al. [20]	2004	6	-	-
El-Wahsh [8]	2006	1	Abdominal pain, nausea	Conservative management followed by bowel rest and anticoagulation
Menéndez-Sánchez et al. [19]	2010	1	Abdominal pain	Surgery and antibiotic treatment
Hsu et al. [3]	2012	1	Abdominal pain, fever	Anti-coagulation and laparoscopic cholecystectomy
Present case	2014	1	Abdominal pain, fever, vomiting	Conservative management

splenectomy, and pancreatitis are commonly observed [11–13]. However, we did not observe any association of local risk factors in our case with no history of splenectomy or other complications. The most frequent symptoms include abdominal pain, nausea, vomiting, diarrhea, or constipation [12]. The clinical signs are usually non-specific and patients often present with abdominal tenderness and distension, diminished bowel sounds, fever, and shock.

Moreover, patients with PVT have differential presentation depending upon the site of the thrombosis (portal vein or portal vein territory), acute or chronic, and collateral veins development. These collateral veins develop in few days after portal vein obstruction, and get completed within 3 to 5 weeks [14,15]. Several causes of PVT have been reported in the literature, however, PVT post acute cholecystits is rare event and only few cases have been reported so far (Table 1) [3,8,16–20]. This could be attributed to the subclinical course of the disease, which is only detected incidentally during radiological examination. Orgen et al. [11] reported the rate of incidental detection of PVT is around 1% of the general population based on 23 796 consecutive autopsies from Sweden. Choi et al. [20] reported 6 cases of PVT associated with acute cholecystitis in a retrospective radiological evaluation of patients with transiently increased hepatic attenuation on CT scan. These authors observed no etiological factors associated with PVT and speculated that the occurrence of PVT post-acute cholecystitis could be due to inflammation or an infectious process that involves the cystic vein. Similarly, we did not observe any coexistent clinical conditions responsible for PVT and there was no abscess collection to indicate any involvement of inflammation or infectious process in our case.

The treatment goal in PVT is to prevent extension of the thrombus and to attain portal vein patency. The various treatment approaches for PVT include the management of the underlying disease, use of antibiotics, hydration, anticoagulation therapy, and (infrequently) thrombolytic therapy or surgical embolectomy [21]. However, some studies suggested spontaneous resolution of the PVT [22,23]. The time interval between thrombosis formation and start of the anticoagulation treatment dictates the outcome of recanalization [21]. It has been reported that in case of acute PVT, early anticoagulation treatment results in favorable outcome. For instance, Turnes and colleagues [24] showed that the rate of recanalization was greater in patients who received anticoagulation early in the first week post diagnosis in comparison to the second week anticoagulation therapy (69% vs. 25%).

In our case, the patient was treated conservatively with subcutaneous heparin injection. We treated the primary condition "acute cholecystitis" that improved patient condition, in terms of liver derangement normalization. However, the therapeutic management should also aim to resolve extensive portal vein obstruction and to avoid serious complications [10].

Our report is limited in that the patient did not undergo followup abdominal CT imaging after conservative management to look for any change in imaging results. We only followed clinical and laboratory findings for resolving the disease manifestations of the patient until complete recovery.

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## Conclusions

Conservative treatment could be a feasible and safe approach for the management of PVT with acute cholecystitis, if treated at an early stage. The occurrence of PVT secondary to acute cholecystitis could possibly be explained by the pericholecystic inflammation that affects the portal vein, which resulted in PVT. Moreover, initial diagnosis based on Doppler ultrasonography and CT scan is possible only if the surgeons are familiar with this uncommon condition. Therefore, the diagnosis of such patients requires a high index of suspicion and early management of associated etiology to achieve better outcomes.

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#### **Conflicts of interests**

The authors have no conflicts of interests and no financial issues to disclose.

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