Clinical Case Report

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Acute portal vein thrombosis after hepatectomy in a patient with hepatolithiasis

A case report and review of the literature

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Abstract

Rationale: Portal vein thrombosis is defined as any thrombosis that develops in the portal vein system. It is considered a very rare and extremely lethal complication of hepatopancreatobiliary surgery.

Patient concerns: Acute portal vein thrombosis after hepatectomy in patients with hepatolithiasisis very rare. Acute portal vein thrombosis is considered as a dangerous complication after hepatectomy. It is easy to ignore the symptom of acute portal vein thrombosis. Once the appropriate time of treatment is past, it would lead to patients' death.

Diagnose: Acute portal vein thrombosis after hepatectomy in a patient with hepatolithiasis

Interventions: We consider anticoagulation therapy and percutaneous transhepatic portal vein puncture and thrombectomy once the diagnosis of acute portal vein thrombosis is confirmed.

Outcomes: The patient's liver function continued to deteriorate, eventually resulting in death.

Lessons: Acute portal vein thrombosis after hepatectomy is difficult to diagnose. The management of acute portal vein thrombosis remains controversial according to its severity, location or time of discovering.

Abbreviations: POD = postoperative day, PVT = portal vein thrombosis.

Keywords: acute portal vein thrombosis, hepatectomy, hepatolithiasis

1. Introduction

Portal vein thrombosis (PVT) is defined as any thrombosis that develops in the portal vein system.[1] It is usually recognized in patients with cavernomatous transformation of the portal vein with portal hypertension.[2] Postoperative PVT is considered a very rare and extremely lethal complication of hepatopancreatobiliary surgery.[3,4] Most reported cases have occurred in patients undergoing liver transplantation, splenectomy, or pancreaticoduodenectomy. Risk factors include liver cirrhosis, chronic hepatitis, splenectomy, the Pringle maneuver, and portal hypertension.[5–7] We herein report a case of acute postoperative PVT in a patient with hepatolithiasis. Compared with the published literature, our case is unique because no similar reports can be found, previously established risk factors could not explain the reason for the occurrence of PVT, and the cause of the patient's death despite the performance of all necessary treatments remains unknown.

2. Case report

In November 2015, a 44-year-old man was admitted to the outpatient clinic of our hospital with a 1-month history of fever, abdominal pain, and jaundice. He reported having undergone cholecystectomy in 1993 and exploratory surgery of the common bile duct in 1995 and 2010. The patient denied any history of chronic liver disease. He did not take any special dietary or herbal agents and did not drink alcohol. Measurement of serologic markers and an ultrasonographic examination were negative for viral hepatitis, Budd–Chiari syndrome, and autoimmune hepatitis. A preoperative liver test, routine blood test, coagulation function test, and alpha-fetoprotein measurement showed no abnormalities. Abdominal ultrasonography and magnetic resonance cholangiopancreatography showed calculi in the main and left hepatic bile ducts, and the left lateral lobe was atrophic. No sign of hepatic PVT was detected by either ultrasound or magnetic resonance imaging (Fig. 1). Common bile duct exploration and left lateral lobe hepatectomy was then performed. The Pringle maneuver was not used during the surgery, and no obvious blood loss occurred during the hepatectomy.

The postoperative course was uneventful with the exception of mild abdominal pain in the first 5 days after the surgery. On the sixth day, however, the patient showed obvious jaundice. Ultrasonography showed PVT of the main portal vein. Anticoagulation therapy using intravenous heparin at 10,000 U/day was administered. Twenty-four hours later, a coagulation disorder

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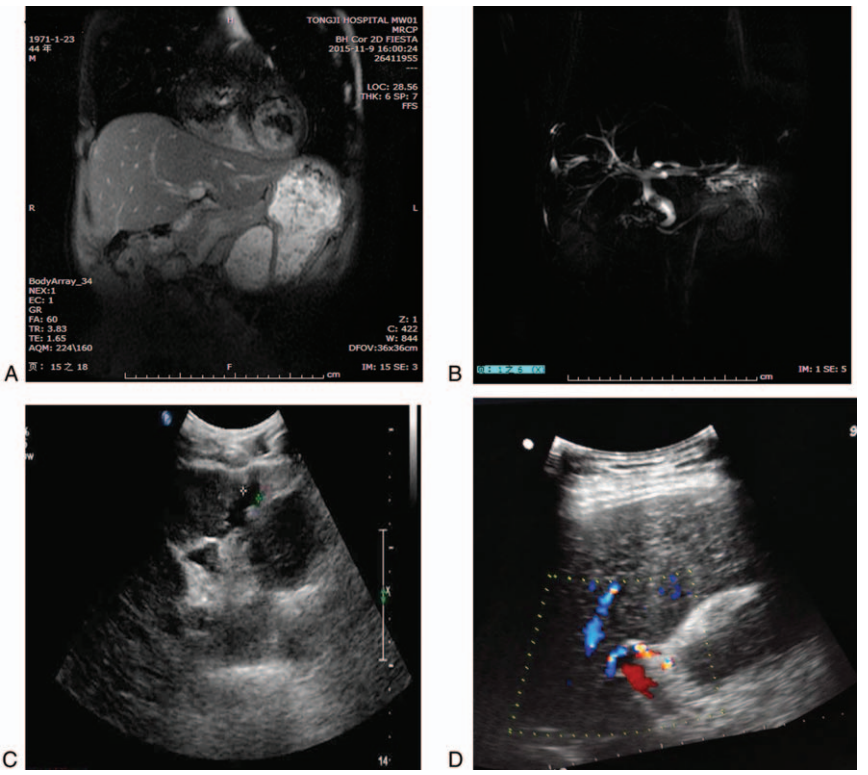

Figure 1. Imaging examination. (A, B) Preoperative magnetic resonance imaging showed no sign of portal vein thrombosis. Calculi were present in the main and left hepatic bile ducts. (C) Preoperative ultrasonography showed no signs of portal vein thrombosis. (D) Ultrasonography showed thrombosis in the main portal vein and left portal vein. The direction of flow in the portal vein was countercurrent (shown by blue color in Doppler ultrasonography).

and obvious signs of bleeding were detected. After a discussion with clinicians from multiple disciplines, plasmapheresis with subsequent percutaneous transhepatic portal vein puncture and thrombectomy was performed. After this treatment, the portal vein showed a small blood flow signal. However, the liver function continued to deteriorate. One day later, the patient died. All blood test results are shown in Figure 2. After communication with the patient's daughter, we received consent for publication of this case.

3. Discussion

Postoperative PVT is a very rare complication. Most reports regarding PVT have focused on liver transplantation or pancreaticoduodenectomy.[3,4] In the past 2 years, PVT has been considered to be a complication after hepatectomy, and several large-scale retrospective studies have confirmed this.[8,9] The reported prevalence of PVT after hepatectomy ranges from 2.1% to 9.0%, and the risk factors for portal thrombosis include the duration of the Pringle maneuver during the operation,[9]

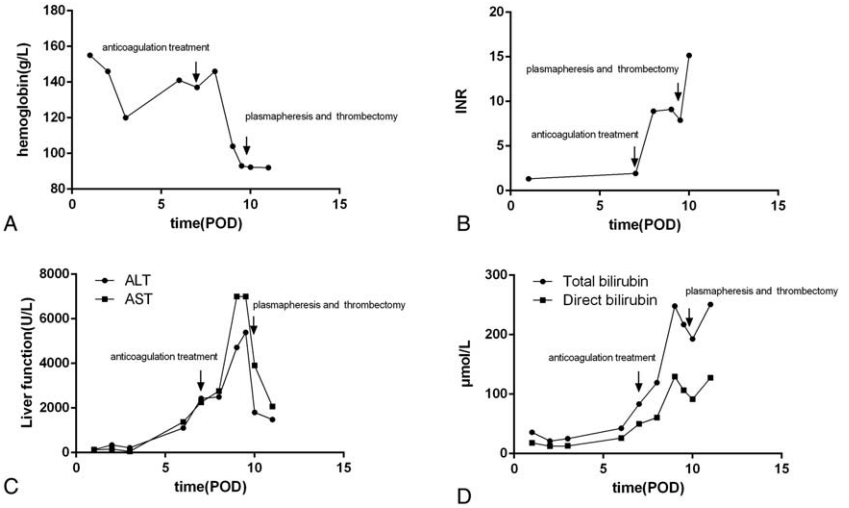

Figure 2. Postoperative blood test results. (A) Hemoglobin level. (B) International normalized ratio. (C, D) Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and bilirubin levels. ALT = alanine aminotransferase, AST = aspartate aminotransferase.

intra-abdominal infection/inflammation,[10,11] postoperative bile leakage,[8] splenectomy,[8] and cirrhosis.[12] The patient in the present case had none of these risk factors, but PVT was discovered 5 days postoperatively. We immediately administered anticoagulation therapy; 1 day later, however, we discovered obvious signs of bleeding. Direct thrombectomy was performed after plasmapheresis, but this treatment had little effect despite the fact that we had eliminated the thrombosis of the main portal vein.

We reviewed the published English-language literature describing patients with acute PVT after hepatectomy to investigate the cause and treatment of this complication, and the results are shown in Table 1. Only 2 studies focused on acute PVT after hepatectomy without concomitant splenectomy. Among the cases of acute PVT, right-sided hepatectomy was performed more frequently. Kuboki et al[8] proposed that the portal vein often became kinked after right-sided hepatectomy, which could be the cause of acute PVT. In their study, they developed a new procedure by which to straighten the portal vein. By anchoring sutures between the anterior wall of the inferior vena cava and the posterior wall of the portal vein has the ability to stretch the kinked portal vein and reduce the incidence of acute PVT after hepatectomy.[8] Anticoagulation therapy[13] or thrombolytic therapy[14] is the most common treatment for postoperative PVT. However, the effects of anticoagulation treatment remain controversial because the rate of effective recanalization of the portal vein ranges from 24% to 81%.[15,16] Additionally, liver function might be damaged in patients with acute main PVT. Anticoagulation or thrombolytic therapy is a high-risk treatment for patients who have undergone major hepatectomy because such therapy may contribute to massive bleeding of the cut surface of the liver. Surgical thrombectomy has been introduced to increase the rate of effective recanalization.[17] Kuboki et al[8] strongly recommended that if PVT detected on postoperative day 5 or earlier after initial hepatectomy, urgent surgery such as thrombectomy would be the first choice; if PVT is detected on postoperative day 6 or later, immediate anticoagulation therapy is the first choice of the treatment of PVT.[8]

We have not found an adequate explanation for the acute PVT after hepatectomy in the present case. It seems that none of the above-mentioned explanations can be applied to our case. In our experience, the second or third surgery for recurrent hepatolithiasis usually contributes to a higher risk of diffuse abdominal inflammation and bile leakage. A higher dosage of antibiotics was used immediately after the surgery; thus, a sustained increase in the white blood cell count was not observed. However, after the antibiotic dosage was lowered, the PVT and increased white blood cell count were detected. Therefore, we hypothesized that diffuse abdominal inflammation after the hepatectomy was the cause of the PVT in this patient. However, more data would be needed to confirm our hypothesis.

To further improve the diagnosis and treatment of PVT, all patients who have undergone hepatectomy should be monitored for the development of postoperative PVT. Although the prevalence of post-hepatectomy PVT is relatively low, it is associated with a high risk of hepatic failure and death. Early diagnosis of acute postoperative PVT is difficult because of the lack of specific symptoms.[17] In our opinion, the above-mentioned improvements in the surgical technique of major hepatectomy might decrease this prevalence of acute PVT. Such surgical skills should be tested in a large group of patients. However, it seems to us that early prevention should be considered most important after hepatectomy. Once the patient

Table 1

Published English-language literature of patients with acute portal vein thrombosis.

No.	Literature	No. of patients	Age	Usage of prophylactic anticoagulation	Operational procedure	Time of discovering PVT		Associated liver disease		Location of PVT		Treatment technique and response rate			Mortality
						Early	Late	Cirrhosis	Malignance	MPV	PPV	Surg	Lytic	Anti-coag	
1	Yoshiya et al[9]	19/208	66.7 ± 0.8	No	Major hepatectomy (8/40) Minor hepatetomy (11/168)	0	19	3/19	16/19	7/19	11/19	0	0	9/19	0
2	Kuboki et al[8]	25/1193	64.0 ± 1.5	No	Major hepatectomy (18/446) Minor hepatetomy (7/747)	12/25	13/25	4/25	24/25	–		9/11	–	12/14	2/25
4	Stambo and Grauer[18]	1	–	No	Pancrease biopsy	1	0	0	1	1	0	1/1	1/1	0	0
5	Janssen et al[19]	172	–	No	Operative (23/172)*	–	–	124/172	148/172	89%	11%	1/NR	0	46/NR	0
6	Amitrano	121	–	No	Operative(23/121)*	–	–	0/121	0/121	–	–	–	4/4	29/41	0
7	Hongwei[20]	10/90	–	(90/90)	Spleenectomy(90/90)	10/10	–	90/90	0/90	10/10	–	–	–	90/90	0
8	Kawanka et al[21]	3/37	–	(37/37)	Spleenectomy(37/37)	37/37	–	37/37	0/90	37/37	–	–	–	37/37	0
9	Lai et al[22]	31/148	–	(31/31)	Spleenectomy(31/31)	31/31	–	31/31	–	31/31	–	–	–	31/31	0

Anti-coag = anti-coagulation therapy, lytic = thrombolytic therapy, MPV = main portal vein, PPV = peripheral portal vein, Surg = surgical thrombectomy.
*the specific surgical procedure was not mentioned in the study.

has shown an abnormal change in liver function or has developed abdominal pain after hepatectomy, PVT should be considered. At this point, aggressive Doppler ultrasonography and D-dimer or fibrin degradation product measurement should be performed to rule out PVT. If PVT is detected in the early postoperative period and the patient's coagulation function has not yet deteriorated, surgical thrombectomy or endovascular thrombectomy should be performed. Once the liver function has deteriorated, treatment of acute PVT becomes limited and the risk of mortality greatly increases.

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