A Case of Portal Vein Thrombosis after Cesarean Section

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Portal vein thrombosis (PVT) commonly occurs in patients with liver cirrhosis and may be caused by hypercoagulable states, such as malignancy, infection, or thrombophilia. Without appropriate treatment, PVT may develop into life-threatening conditions, such as liver failure, disseminated intravascular coagulation, mesenteric vein thrombosis, and intestinal necrosis. We herein report a case of PVT after cesarean section in a woman with no underlying medical conditions.

The patient was a 35-year-old multiparous woman with no previous medical complications. She underwent elective cesarean section at 38 weeks' gestation for indications of pregnancy after a previous cesarean section. When she walked on the first postoperative day, her oxygen saturation decreased to 89 % ; therefore, pulmonary thromboembolism was suspected. Contrast-enhanced computed tomography was performed and showed no pulmonary embolism, but revealed a thrombus of the portal vein in hepatic segment 8. PVT was diagnosed and anticoagulant therapy was immediately initiated. Liver enzymes remained normal at diagnosis, increased transiently on postoperative day 6, and subsequently normalized. She underwent routine abdominal ultrasound, and the portal vein thrombus disappeared 9 weeks after cesarean section. A blood examination was conducted to clarify why the patient developed PVT ; however, she had no predisposition to thrombosis.

Since the thrombus was detected early and treatment was promptly initiated, the patient did not develop severe complications. Pregnancy and cesarean section were considered potential triggers for PVT. This case highlights the need to recognize PVT as a possible manifestation of venous thromboembolism in peripartum management. *Shinshu Med J 74 : 51—57, 2026*

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Ⅰ Introduction

Portal vein thrombosis (PVT) generally develops in association with a number of conditions, such as liver cirrhosis, malignancy, infection, and thrombophilia, and is rare in patients without underlying diseases[1]. PVT presents with non-specific symptoms, including fever, abdominal pain, and nausea, making an early diagnosis difficult, and often progresses by the time of diagnosis[2]. Without appropriate treatment, PVT may develop into life-threatening conditions, including liver failure, disseminated intravascular coagulation (DIC), mesenteric vein thrombosis, and intestinal necrosis[3]. In contrast, there have been few case reports of PVT occurring during pregnancy and the postpartum period[4]. We herein report a postpartum case of incidentally detected PVT after cesarean section.

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Ⅱ Case Presentation

The patient was a 35-year-old multiparous woman (gravida 3, para 1) with no remarkable medical history or comorbidities. Her family history was notable only

for prostate cancer in the maternal grandfather, with no other relevant familial diseases. Her previous pregnancy resulted in an emergency cesarean section at 39 weeks' gestation due to inefficient uterine contractions and prolonged labor. Her current pregnancy was achieved through artificial insemination by her husband (AIH) at a local reproductive medicine clinic. She was referred to our hospital at 11 weeks' gestation for her pregnancy and peripartum management. The subsequent pregnancy course was uneventful.

She was admitted at our hospital at 38 weeks 3 days' gestation for peripartum management after previous cesarean section. On admission, a physical examination revealed a height of 150 cm, weight of 53.9 kg, body mass index (BMI) 24.0, body temperature of 36.4 ℃, blood pressure of 101/61 mmHg, and pulse rate of 61 beats/min, with no remarkable findings. On 38 weeks 4 days' gestation, she underwent elective cesarean section under combined spinal-epidural anesthesia due to a previous history of cesarean section. The surgery was completed without any intraoperative complications, and intraoperative blood loss was 700 g. The neonate was a female, with a birth weight of 3,158 g, Apgar scores of 9 and 9 at 1 and 5 minutes, respectively, and umbilical arterial blood pH of 7.28. The baby was in a stable condition.

The patient was subcutaneously administered heparin calcium (10,000 units/day) for venous thromboembolism (VTE) prophylaxis from postoperative day 0. On postoperative day 1, during her first ambulation, a pulse oximeter revealed that her blood oxygen saturation decreased from 96 % to 89 % in room air, despite no subjective symptoms. Blood laboratory tests revealed that her fibrin/fibrinogen degradation products (FDP) D-dimer level was elevated at 3.7 μg/mL (normal range 0.0-1.0 μg/mL) **(Table 1)**. Contrast-enhanced computed tomography (CT) excluded thrombi in the pulmonary arteries, inferior vena cava, and lower extremity veins, but revealed bilateral pleural effusion and atelectasis of the lower lobes of both lungs, suggesting the causes of low SpO_2. In addition, contrast-enhanced CT showed a lesion of hyperattenuation in hepatic segment 8 on plain images, which demonstrated poor enhancement in the venous phase

Table 1 Laboratory findings at the diagnosis of portal vein thrombosis

【Complete Blood Count】	
WBC (/μL)	13,060
Hb (g/dL)	11.7
PLT ($\times 10^4$/μL)	23.9
【Coagulation Studies】	
PT (sec)	11.7
APTT (sec)	25.2
FIBG (mg/dL)	480.0
AT-III (%)	84.7
FDP D-dimer (μg/mL)	3.7 (normal range 0.0-1.0)
【Serum Chemistry】	
BUN (mg/dL)	5.0
Cre (mg/dL)	0.60
UA (mg/dL)	5.3
Na (mmol/L)	140
K (mmol/L)	3.8
Cl (mmol/L)	107
AST (U/L)	22
ALT (U/L)	8
LDH (U/L)	227
T-Bil (mg/dL)	0.59

(Fig. 1). Based on these findings, the patient was diagnosed with PVT. After a consultation with a gastroenterologist, anticoagulant therapy was initiated using the continuous intravenous infusion of heparin sodium, followed by a switch to oral medication. Since the patient wanted to continue breastfeeding, oral warfarin potassium, which may be used during breastfeeding, was initiated on postoperative day six instead of direct oral anticoagulants (DOACs).

Her postoperative course is shown in **Fig. 2**. Liver enzymes were initially normal, increased on day 6 (AST 75 U/L, ALT 61 U/L), and then decreased. Trans-abdominal ultrasonography on day 6 showed no progression of PVT. The patient was discharged on postoperative day 9 with continuing warfarin (4 mg/day). She was followed up with monthly ultrasonography and blood testing, which confirmed the disappearance of the thrombus and the normalization of her FDP-D dimer level (0.4 μg/mL) by 9 weeks after surgery **(Fig. 2, 3)**. We did not detect any abnormalities related to thrombophilia, such as antithrombin III (AT-III), antinuclear antibody, anti-

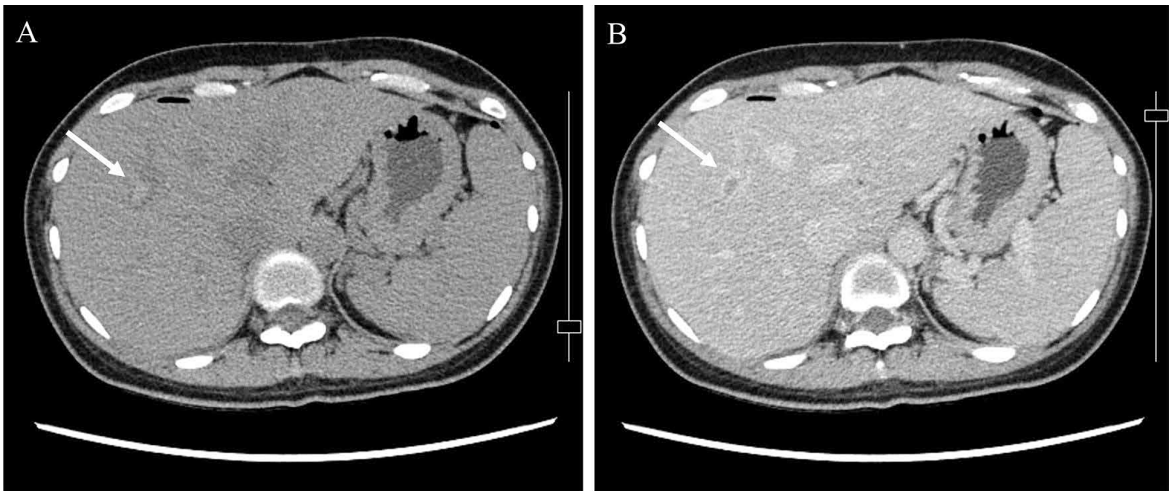

Fig. 1　Contrast-enhanced CT (at the diagnosis of portal vein thrombosis)
A : Unenhanced phase　B : Venous phase (Arrow : portal vein thrombus)
A hyperdense lesion in segment 8 on unenhanced CT (A) corresponds to a hypodense, non-enhancing area in the venous phase (B).

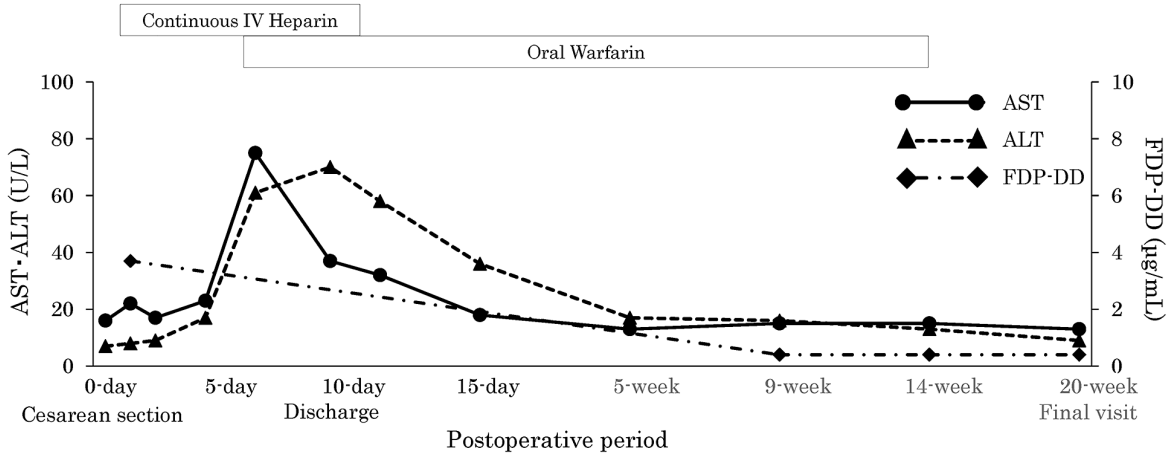

Fig. 2　Postoperative clinical course

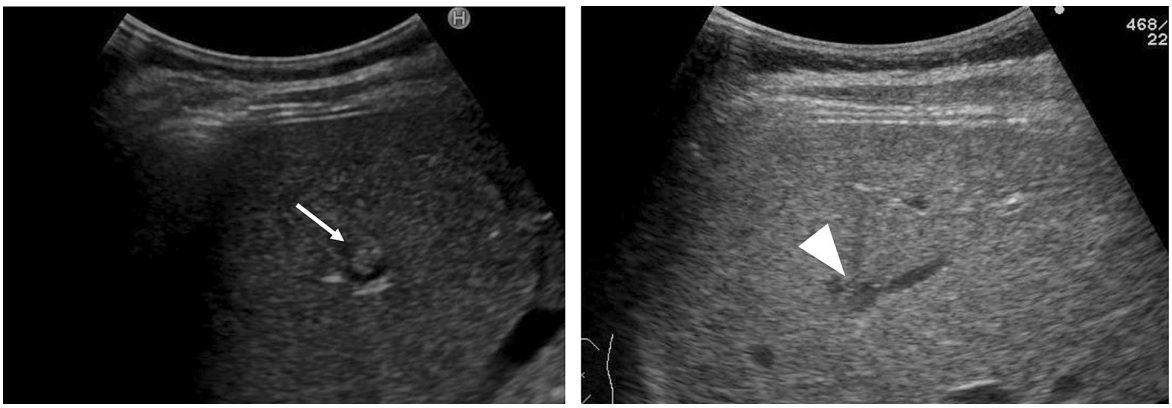

Fig. 3　Transabdominal ultrasound findings
A : At the diagnosis of portal vein thrombosis (Arrow : thrombus)
B : At the disappearance of portal vein thrombosis (Arrowhead : disappearance of the thrombus)

dsDNA antibody, anti-beta2 glycoprotein I antibody, anticardiolipin antibody IgG, anticardiolipin antibody IgM, lupus anticoagulant, protein C, and protein S. Warfarin was discontinued 14 weeks postoperatively. The patient has remained free of recurrence, and follow-up was concluded at 20 weeks.

Ⅲ Discussion

PVT was previously considered a rare disease, occurring in 0.05-0.5 % of autopsy cases[5]. However, with advances in imaging modalities, such as CT and magnetic resonance imaging (MRI), PVT is more commonly detected, and has been reported to occur in 0.6-16 % of patients with compensated cirrhosis[5)6)] and up to 35 % of those with decompensated cirrhosis or hepatocellular carcinoma[1]. The main causes of PVT are liver cirrhosis, malignant tumors, infections, and prothrombotic states[7].

Factors associated with the development of venous thrombosis generally include vascular endothelial damage, abnormal blood flow, hypercoagulability, reduced fibrinolytic activity, and abnormalities in the platelet count and function. When these factors affect the portal vein due to various underlying conditions, thrombus formation may occur within the portal vein. Liver cirrhosis is the most common condition associated with PVT in adults. In cirrhotic patients, congestion of the portal venous system and reduced portal blood flow due to portal hypertension are considered to be the primary mechanisms of PVT, and inflammation of the portal vein as well as coagulation abnormalities are also contributing factors. In cases of PVT secondary to malignant tumors, such as hepatocellular carcinoma or pancreatic cancer, direct invasion or compression of the portal vein by the tumor, as well as surgical manipulation, play major roles in thrombus formation. In intra-abdominal infections, such as acute appendicitis or biliary tract infections, increased coagulation activity associated with portal venous infection and dehydration is considered to lead to PVT[8].

During the pregnant and postpartum periods, women are susceptible to VTE due to high estrogen levels and the presence of Virchow's triad : venous stasis, endothelial injury, and hypercoagulability. The incidence of peripartum VTE is reportedly 1-12 per 10,000 pregnancies, with most of these cases being deep vein thrombosis (DVT) in the lower limbs or pelvis, or pulmonary thromboembolism (PTE)[9]. Therefore, we initially suspected VTE/PTE in the present case and performed a contrast-enhanced CT scan.

In contrast, PVT associated with pregnancy is rare and has only been reported in approximately 30 cases worldwide. We searched Japanese cases using the Japanese Medical Abstracts Society database (Ichushi®, Japan, https://login.jamas.or.jp/) and MEDLINE of National Library of Medicine (PubMed®, USA, https://pubmed.ncbi.nlm.nih.gov/) from 1993 to 2024, and identified only seven cases, including ours **(Table 2)**[10)-15)]. The onset of PVT occurred in two cases during the first trimester of pregnancy and in five cases during the postpartum period, which is consistent with the high-risk period for VTE associated with pregnancy[16]. In five postpartum cases, three were complicated by hypertensive disorders of pregnancy and all underwent cesarean section. The risk of VTE was previously shown to be approximately two-fold higher in women with hypertensive disorders of pregnancy than in controls[17] and four-fold higher after cesarean section than after vaginal delivery[18]. In two cases, AT-III levels were low. One case had no remarkable family history. Blood laboratory tests during the acute phase of thrombosis showed mildly low AT-III levels ; however, the causal relationship with PVT was unclear[10]. In the other case, extrahepatic venous shunt surgery was performed for extrahepatic portal vein obstruction in childhood. Although there were no serious complications in adulthood, the patient had AT-III deficiency due to chronic liver dysfunction, which was suspected to be one of the factors that led to the development of PVT[15]. Although our patient had no comorbidities, pregnancy and cesarean delivery likely contributed to thrombus formation.

Since PVT presents clinically with non-specific symptoms, including fever, abdominal pain, nausea, vomiting, and abnormal liver function tests, its diagnosis is often delayed compared to DVT and PTE.

Table 2　Case reports from Japan (1993–2024)

Case (Year)	At diagnosis	Complication	Pregnancy outcome	Coagulation disorder	Symptom	Time to diagnosis	Laboratory findings at diagnosis	Location of the thrombus	Diagnostic method	Treatment	Prognosis
Kamamura (1993)[10]	Preg.7W	Obesity	AA	AT-III	Upper abdominal pain Hematochezia	4 weeks	AST 15 ALT 8	Main portal vein SMV	Ultrasound CT Angiography	Aspirin Warfarin	No sequelae
Shimada (2011)[11]	POD2	PE Obesity	Cesarean	None	None		AST 731 ALT 532	Intrahepatic portal vein	Ultrasound	Heparin	No sequelae
Teramoto (2013)[12]	POD9	None	Cesarean	None	Nausea Vomiting Upper abdominal pain	3 days	AST 12 ALT 10 FDP-DD 15.85	Main portal vein SMV	Contrast-enhanced CT Angiography	Urokinase Danaparoid Warfarin	No sequelae
Tomeno (2016)[13]	POD1	PE (HELLP syndrome)	Cesarean	NA	Upper abdomen pain	1 day	AST 6007 ALT 6565 FDP-DD 72.49	Intrahepatic portal vein	Ultrasound Contrast-enhanced CT	Plasmapheresis Danaparoid Heparin Warfarin	No sequelae
Endoh (2017)[14]	POD2	PE	Cesarean	None	None		AST 496 ALT 506 FDP-DD＞30	Intrahepatic portal vein Pulmonary artery	Contrast-enhanced CT	Heparin Enoxaparin Warfarin	No sequelae
Hosomi (2024)[15]	Preg.8W	EHPVO post-surgery	Cesarean	AT-III	Leg edema	9 weeks	ALT 23 FDP-DD 2.4	Extrahepatic portal vein	Contrast-enhanced CT	Heparin Warfarin	No sequelae
Our case	POD1	None	Cesarean	None	None		AST 22 ALT 8 FDP-DD 3.7	Intrahepatic portal vein	Contrast-enhanced CT	Heparin Warfarin	No sequelae

Preg : pregnancy　POD : postoperative day　PE : preeclampsia　HELLP : Hemolysis, Elevated Liver enzymes, Low Platelets
EHPVO : extrahepatic portal vein obstruction　AA : artificial abortion　AT-III : antithrombin III　NA : not available
FDP-DD : fibrin/fibrinogen degradation products and D-dimer　SMV : superior mesenteric vein
Units : AST (U/L) ALT (U/L) FDP-DD (μg/mL)

Therefore, PVT may develop severe complications, such as liver failure, DIC, and thrombosis obstructing the superior mesenteric vein and causing intestinal necrosis, which may be fatal[13]. Among the seven Japanese cases identified, one developed liver failure and DIC[13], and two had thrombus extension into the superior mesenteric vein[6)9)]. The diagnostic interval ranged from 1 day to 9 weeks. All cases recovered without sequelae ; however, the mortality rate among non-cirrhotic, non-malignant PVT cases was previously shown to be approximately 8 %[19]. As such, a prompt diagnosis by imaging is crucial. In the present case, PVT was incidentally detected during an evaluation for suspected PTE, allowing for early anticoagulation and a favorable outcome. Clinicians need to consider PVT as part of the VTE spectrum in peripartum management, and when VTE is suspected or accompanied by upper abdominal pain, an examination of not only the lower extremities and lungs, but also the abdominal vessels is recommended.

Although contrast-enhanced CT is the most useful diagnostic tool[20], exposure to radiation and contrast agents must be considered. Ultrasonography is non-invasive and repeatable, and needs to be employed early in patients with abdominal pain or prolonged liver function abnormalities. In our patient, ultrasonography was valuable for monitoring thrombus regression and treatment efficacy.

Anticoagulation remains the cornerstone of therapy, similar to DVT and PTE ; however, optimal agents and durations have yet to be established. While DOACs show efficacy for non-cirrhotic PVT[21], safety data in pregnancy and lactation are insufficient, and warfarin is the treatment of choice if breastfeeding is desired. Warfarin is generally avoided in lactation due to package insert cautions in Japan ; however, its high protein binding results in negligible secretion into breast milk. Previous studies have shown undetectable levels of warfarin in breast milk and infant plasma[22], suggesting a minimal risk. In the present case, the transition from intravenous heparin to oral warfarin achieved stable anticoagulation, leading to complete resolution by 9 weeks and the discontinuation of therapy at 14 weeks.

Ⅳ Conclusion

We encountered a case of PVT incidentally detected by contrast-enhanced CT after cesarean section. Although clinical symptoms and laboratory findings are non-specific, a delay in treatment may result in a poor prognosis. The recognition of PVT as part of the VTE spectrum in peripartum care is essential for a timely diagnosis and management.

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