梨花醫大誌 : 第 31 卷 第 1 號 2008
Ewha Med J Vol. 31, No. 1, 2008

A Case of Superior Mesenteric Vein and Portal Vein Thrombosis Associated with Normal Delivery Presented by Acute Pancreatitis

Youn Ju Na · Min Jung Kang · Ji Min Jung · Chang Yoon Ha
Hae Sun Jung · Su Jung Baik · Sun Young Yi

Department of Internal Medicine, School of Medicine, Ewha Womans University

= 국문초록 =

정상 분만 후에 상부 장간막 정맥과 간정맥 혈전을 동반한 급성 췌장염의 1예

이화여자대학교 의학전문대학원 내과학교실
나윤주 · 강민정 · 정지민 · 하창윤 · 정해선 · 백수정 · 이선영

급성 췌장염의 가장 흔한 원인은 술과 담석으로 알려져 있다. 간정맥과 장간막 정맥의 혈전을 동반한 급성 췌장염은 임신이나 분만 뒤에 드물게 발견될 수 있다. 임신 전에 복부 손상, 자가면역성질환, 혈전의 과거력이 없었던 28세 여자 환자가 정상 분만 후에 계속되는 우상복부 통증을 주소로 내원하였다. 복부 전산화 단층촬영술에서 췌장의 종대와 간정맥과 상 장간막 정맥의 혈전이 발견되어 항 응고치료를 시작하였다. 이는 두 가지 기전으로 추정할 수 있는데, 임신으로 인한 담낭 기능 저하로 생긴 담즙 찌꺼기는 급성 췌장염을 일으킬 수 있으며, 동반된 과다응고와 염증이 복부정맥의 혈전을 일으킬 수 있다. 또 다른 기전은 통증이 심한 정상 분만 후에 복부정맥 혈전으로 인한 혈류 감소로 급성 췌장염이 유발될 수 있다. 환자는 항 응고치료 후에 환자의 상복부 통증이 호전되었으며 이후 시행한 복부 전산화 단층촬영술에서 혈전은 호전되었으나 해면상 정맥 형성은 진행하였다. 이에 정상 분만 뒤에 급성 췌장염을 동반한 복부정맥혈전의 1예를 보고하는 바이다.

중심 단어 : 급성 췌장염 · 복부정맥 · 혈전.

Introduction

During pregnancy, biliary sludge and gallstone formation could occur, and that is the cause of acute pancreatitis in pregnant women[1]. Portal vein thrombosis (PVT) or mesenteric vein thrombosis (MVT) is an rare occurrence in pregnancy[2]. That is usually associated with genetic coagulation abnormality such as hereditary deficiency of natural anticoagulants (protein C, S and anti-thrombin III), taking oral contraceptives, abdominal trauma, myeloproliferative disorders or autoimmune disease. In the literature review, there is no report that PVT and MVT simultaneously occurred after normal delivery without any coagulation abnormality and associated underlying diseases, and presented acute pancreatitis. Here, we report a rare case of 28 years old female with subacute PVT and MVT with acute pancreatitis after normal delivery.

Case

A 28-year-old woman was admitted to hospital due to a right upper quadrant pain. She had given birth to a baby one month ago and developed an intermittent right upper quadrant pain. She had never used oral contraceptives, and had no smoking and alcohol history. Moreover, there was no history of abortion, abdominal trauma, pancreatitis, autoimmune disease, myelodysplastic disease, or thromboembolism. A physical examination showed direct and moderate rebound tenderness on the right upper abdominal quadrant. Her blood pressure was 110/60mm Hg, heart rate 96/min, and respiratory rate 28/min. Laboratory tests showed a white blood cell count of 7,900/mm^3 (neutrophil count 50%), hematocrit 35.9%, and platelet 149,000/mm^3. Amylase of 180U/L and lipase of 100U/L were slightly elevated. The levels of alkaline phosphatase, total bilirubin, serum aminotransferase, triglyceride levels and prothrombin time were within normal values. Results of tests for antiphospholipid syndrome markers, such as IgG and IgM anti-cardiolipid antibody were negative. Protein C was 0.27mg/dl (reference value, 0.17−0.32) and protein S 3.01mg/dL (reference value, 0.95−2.3). The lupus anticoagulant test and FANA were negative, and IgG, IgM, IgA, C3, and C4 were within normal values. Pancreas dynamic computed tomography with angiography showed a diffuse swollen pancreas and thrombosis of the superior mesenteric vein (SMV) and PV. In addition, it showed infiltration at the head of the pancreas and around celiac axis and superior mesenteric artery (SMA), extending to small bowel mesenteric root (Fig. 1). Abdominal ultrasonography showed no abnormality of the common bile duct and gallbladder. Doppler showed no portal vein flow. Gastroduodenofibroscopy showed chronic superficial gastritis without gastric or esophageal varix .

She was started on intravenous heparin by infusion at 1,000units/hr to maintain an activated partial thromboplastin time of 60 to 80%. On the 5th hospital day, oral warfarin was substituted for heparin. Second follow up pancreas dynamic CT after 7days showed that fluid collection was decreased around the celiac axis, SMA and mesenteric vein. Swelling of the pancreatic head was also decreased and SMV and PV thrombosis were slightly decreased (Fig. 2). The abdominal pain complete disap-

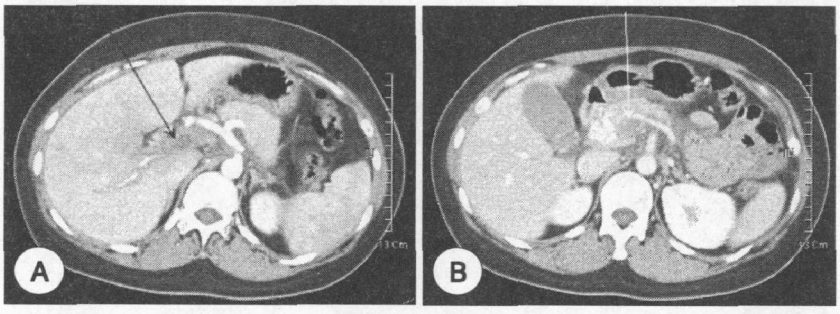

Fig. 1. Initial pancreatic dynamic CT. CT showed thrombosis of portal vein (black arrow, 1A), thrombosis of the superior mesenteric vein, and infiltration at the pancreatic head and around celiac axis and SMA, extending to the small bowel mesenteric root (white arrow, 1B).

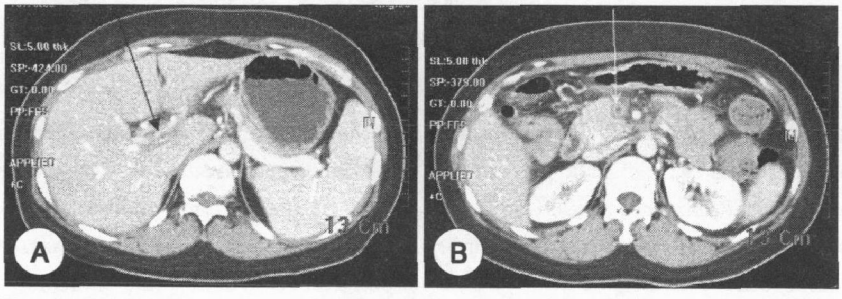

Fig. 2. Follow-up CT was performed on 7th hospital day. Thromboses of the PV (black arrow, 2A) and SMA (white arrow, 2B) were decreased. Also fluid collection was almost disappeared and decreased pancreatic swelling.

peared on hospital day seventh and she was discharged with oral warfarin after 2 weeks in hospital. One month after discharge, follow-up dynamic CT showed a normal sized pancreas and the absence of peripancreatic fluid. The size of SMVT and PVT were also further decreased. However, progressive obliteration of SMV and PV and more cavernous formation in the porta hepatis with pericholecystic collateral vessels were observed (Fig. 3). She was discontinued oral warfarin after 3 month. After 9 month of oral warfarin's discontinuation, she was re-admitted due to hematemesis of esophageal varix. Esophageal band ligation was successfully done.

Discussion

Delivery may have many complications, but abdominal vein thrombosis is unusual[3], and SMVT and PVT with acute pancreatitis is very rare[2]. After childbirth, the described patient here had epigastric pain and a radiating back pain and dynamic CT showed SMVT and PVT with pancreatic swelling and peripancreatic infiltration. It is difficult to determine the order of disease, we could propose two processes. The first possible process is that a painful normal delivery could have resulted in abdominal venous thromboses and cavernous transformation, and that subsequent to these events a blood supply insufficiency caused acute pancreatitis. Another one is that pregnancy-induced biliary sludge could have resulted in acute pancreatitis, and that subsequent to hypercoagulability and inflammatory change caused abdominal vein thrombosis and cavernous transformation.

During pregnancy, biliary sludge and gallstone formation may occur in 31% and 3% of pregnant women, respectively[1]. However, acute pancreatitis occurs at a rate of 1 per 3,300 pregnancies[4]. Moreover, symptoms of cholecystis and biliary dyskinesia due to sludge formation usually disappear at postpartum[1]. Initial dynamic CT showed that the head of the pancreas was swollen and that diffuse infiltration was present in the peripancreatic area. Microscopic biliary sludge could be considered as a precursor of gallstones and the cause of acute pancreatitis in many cases[5]. However, there was no evidence of biliary sludge or ductal dilatation by abdominal ultrasonography at one month after pain-onset. Also, if the thrombosis was induced by acute biliary pancreatitis, most of thrombosis should be disappeared after pancreatitis improved. But there is still option that microbiliary sludges after delivery could have disappeared.

Abdominal vein thrombosis can be inherited or acquired. The causes of inherited thrombophilia are ; factor V leiden mutation[6], prothrombin mutation[6], or deficiency of protein C, protein S or anti-thrombin[7], whereas the cause of acquired thrombophilia are ; age[8], malignant disease[9], surgery, immobilization, drugs, antiphopholipid syndrome, pregnancy, or Behcet's disease[10]. However we found no definite cause of pancreatitis or abdominal vein thrombosis except a delivery in this case. Many causes in portal vein thrombosis are known such as trauma, intra-abdominal sepsis, pancreatitis and prothrombotic disorders, but over 50% of the etiologies in individual patients remained unidentified[11]. Portal vein thrombosis can obstruct blood flow to the liver and may extend towards intrahepatic portal veins. Moreover, these thromboses may extend to involve splenic or mesenteric veins. Collateral vessels which bypass an obstructed segment begin to form in a few days after obstruction and within 3−5 weeks organize into a cavernous transformation[12][13] just like presented case here. MVT is observed in 10% of all patients

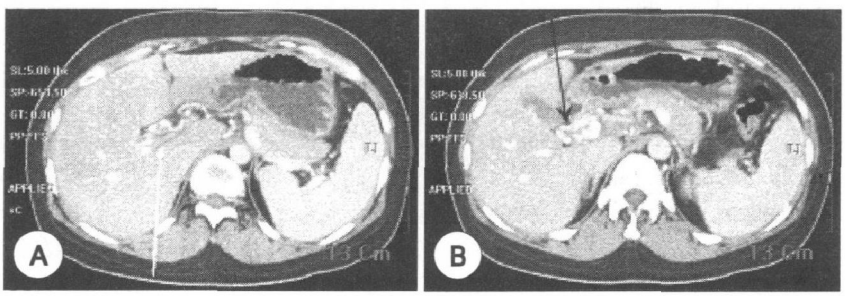

Fig. 3. 2nd follow-up CT. Dynamic CT of the pancreas was performed after 4 weeks. Progressive obliteration of PV (white arrow, 3A) was associated with cavernous formation in the porta hepatic (black arrow, 3B).

with mesenteric ischemia and in 18% of those with acute mesenteric ischemia[14]. The important risk factors are considered to be the presence of a large spleen, cirrhosis, surgery, abdominal inflammation, intra-abdominal cancer, and a thrombophilic condition[7]. Moreover, a genetic factor predisposing thrombosis is present in 78% of patients[15]. Except fulminant cases, patients with mesenteric venous thrombosis typically present later with more diffuse, nonspecific abdominal pain associated with anorexia and diarrhea[7].

After diagnosis, treatment must be initiated by anticoagulation, unless the patient has findings of peritoneal irritation, in which case laparotomy is indicated. Intravenous fluid resuscitation with crystalloids and blood products should be started promptly to correct for volume deficits and metabolic derangements. In addition, broad spectrum antibiotics should be given as early as possible[14].

In the present case, after the initiation of anticoagulation treatment, pancreatic swelling and abdominal pain reduced. In the dynamic CT after 6 week, swelling of the pancreatic head and pericholecystic fluid accumulation had completely disappeared. But, thrombosis and vessel obliterations were unchanged and the cavernous transformation at the porta hepatis had progressed. She experienced esophageal varix bleeding which was complication of portal hypertension after one year follow-up.

We conclude from this case report that abdominal venous thrombosis with acute pancreatitis, which developed after normal delivery. After anticoagulation, acute pancreatitis with abdominal pain was improved. But esophageal varix bleeding occurred after one year.

References

1) Maringhini A, Ciambra M, Baccelliere P, et al : *Biliary sludge and gallstones in pregnancy : incidence, risk factors, and natural history. Ann Intern Med 1999 ; 119 : 116-120*

2) Cornu-Labat G, Kasirajan K, Simon R, Smith DJ, Herman ML, Rubin JR : *Acute mesenteric vein thrombosis and pancreatitis. A rare association. Int J Pancreatol 1997 ; 21 : 249-251*

3) James AH, Jamison MG, Brancazio LR, Myers ER : *Venous thromboembolism during pregnancy and the postpartum period : incidence, risk factors, and mortality. Am J Obstet Gynecol 2006 ; 194 : 1311-1115*

4) Ramin KD, Ramin SM, Richey SD, Cunningham FG : *Acute pancreatitis in pregnancy. Am J Obstet Gynecol 1995 ; 173 : 187-191*

5) Ko C : *Biliary sludge and acute pancreatitis during pregnancy. Nat Clin Pract Gastroenterol Hepatol 2006 ; 3 : 53-57*

6) Samama MM, Rached RA, Horellou MH, et al : *Pregnancy-associated venous thromboembolism (VTE) in combined heterozygous factor V Leiden (FVL) and prothrombin (FII) 20210 A mutation and in heterozygous FII single gene mutation alone. Br J Haematol 2003 ; 123 : 327-334*

7) Bayraktar Y, Harmanci O : *Etiology and consequences of thrombosis in abdominal vessels. World J Gastroenterol 2006 ; 12 : 1165-1174*

8) Oger E : *Incidence of venous thromboembolism : a community-based study in Western France. Thromb Haemost 2000 ; 83 : 657-660*

9) Green KB, Silverstein RL : *Hypercoagulability in cancer. Hematol Oncol Clin North Am 1996 ; 10 : 499-530*

10) Duzgun N, Ates A, Aydintug OT, et al : *Characteristics of vascular involvement in Behcet's disease. Scand J Rheumatol 2006 ; 35 : 65-68*

11) Stringer MD, Heaton ND, Karani J, Olliff S, Howard ER : *Patterns of portal vein occlusion and their aetiologiccal significance. Br J Surg 1994 ; 81 : 1328-1331*

12) Ohnishi K, Okuda K, Ohtsuki T, et al : *Formation of hilar collaterals or cavernous transformation after portal vein obstruction by hepatocellular carcinoma. Observations in ten patients. Gastroenterology 1984 ; 87 : 1150-1153*

13) De Gaetano AM, Lafortune M, Patriquin H, De Franco A, Aubin B, Paradis K : *Cavernous transformation of the portal vein: patterns of intrahepatic and splanchnic collateral circulation detected with Doppler sonography. Am J Roentgenol 1995 ; 165 : 1151-1155*

14) Oldenburg WA, Lau LL, Rodenberg TJ, Burger CD : *Acute mesenteric ischemia : a clinical review. Arch Intern Med 2004 ; 164 : 1054-1062*

15) Imperato M, Moujahid M, Mennecier D, et al : *Acute mesenteric venous thrombosis. A retrospective study of 9 patients. Ann Chir 2006 ; 131 : 595-600*