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Surgical Thrombectomy for Complete Portal Vein Thrombosis Induced by Blunt Abdominal Trauma

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Abstract

Background: Blunt abdominal injury is a known, but very rare cause of portal vein thrombosis. In most cases, full anticoagulant therapy is sufficient. Surgical thrombectomy is an extremely rare procedure in the treatment of acute portal thrombosis.

Materials and methods: We present a case of a young patient with portal vein thrombosis and its branches after a blunt abdominal injury resulting from the sport. Due to severe infarction of the intestinal loops, we chose surgical thrombectomy as the only causal treatment option. This procedure is generally rarely reported in the scientific literature. However, it has not yet been described with a blunt abdominal injury. The patient was subsequently shown to have a Leiden mutation in a heterozygous form. The interplay of all factors including blunt abdominal injury, Leiden mutation, and intestinal malrotation could lead to acute thrombosis of the portal vein and its branches.

Conclusions: We successfully used direct surgical thrombectomy from portal vein and its branches for the patient with portal vein thrombosis and severe small intestine infarction after a blunt abdominal trauma as the only available treatment option, because other treatment options were not suitable in this patient. In our case, early diagnosis, right decision to choose surgical treatment, and direct surgical trombectomy resulted in success and the patient has fully returned to normal life.

Keywords: Blunt injury • Portal vein thrombosis • Leiden mutation • Surgical thrombectomy

Introduction

Blunt abdominal trauma is a known but very rare cause of portal vein thrombosis. Most of the documented post-traumatic cases occur as a result of high-energy trauma, while only a few cases arise as a result of low-energy trauma. Approximately 40–60 % of patients with portal vein thrombosis are associated with thrombophilia [1]. Long-term anticoagulant therapy or local thrombolysis are generally used to treat this condition. If conservative treatment fails, surgical thrombectomy may be considered with an efficiency of about 30 % [2]. However, surgical thrombectomy may be the only chance for patients with impending or developing intestinal ischemia, such as in our patient's case.

Materials and methods

We present a case of a 36-year-old male patient, completely healthy until this incident, with developed acute thrombosis of the portal vein and its branches (superior mesenteric vein and lienal vein) as a result of a low-energy blunt abdominal trauma occurred during ice-hockey match. The patient was pushed into the boards during an amateur hockey game. Subsequently, in the evening of that day, severe abdominal pain and enterorrhagia developed, for which the patient was admitted to the intensive care unit of a regional hospital.

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CT scan of the abdomen showed congenital bowel malrotation, suspected acute pancreatitis, and hypoplasia of the inferior vena cava and hepatic veins. Due to the suspicion of acute post-traumatic pancreatitis, the patient was transferred to a specialized University hospital's intensive care unit the next morning. Updated CT of the abdomen has shown hemoperitoneum, portal vein thrombosis, and intestinal infarsation. A large calcified spleen cyst of 120 mm in diameter was an incidental finding (see Figure 1). Therefore, fluid resuscitation was initiated via administration of two units of red blood cells and fresh frozen plasma, one unit of platelets, and four grams of fibrinogen.

The patient was hemodynamically unstable, and he was given a small dose of catecholamines, e.g., noradrenaline 5 mg in 50 ml of saline solution, the flow rate at 5.0 ml /min (NORADRENALIN, Zentiva a.s. Prague, Czech Republic). Due to circulatory instability and suspicion of hemoperitoneum, the patient was indicated for emergency surgery. Intravenous antibiotic therapy was started preoperatively: cefuroxime 1.5 g (CEFUROXIM KABI, Fresenius Kabi, s.r.o., Prague, Czech Republic), metronidazole 500 mg (METRONIDAZOL B. BRAUN, B. Braun, Melsungen AG, Melsungen, Germany), and gentamicin 480 mg (GENTAMICIN B. BRAUN, B. Braun, Melsungen AG, Melsungen, Germany). Intraoperatively, a crash laparotomy was used to enter the abdominal cavity, where about 1.5 litres of hemorrhagic effusion was found besides severe infarction of the entire digestive tract from the stomach to the sigmoid colon with a dominant finding on the small intestine.

The small intestine was livid, with extensive intramural hematoma, and no palpable pulsations in the mesentery. The expected intestinal malrotation was confirmed. Small intensive loops were located in the right half of the abdomen, while the entire colon was in the left half. A lesser sac was opened, the pancreas was slightly bruised after contusion but intact. We could identify a large calcified spleen cyst about 12 cm in diameter in the left subphrenic space. Due to massive infarction and portal thrombosis, a vasographic intervention was considered but contraindicated due to urgency of the situation and low predictive effect of the procedure. Therefore, we decided for a direct thrombectomy of the portal vein and its branches as the only possible solution (Figure 1).

Portal vein in the hepatoduodenal ligament was identified and dissected.

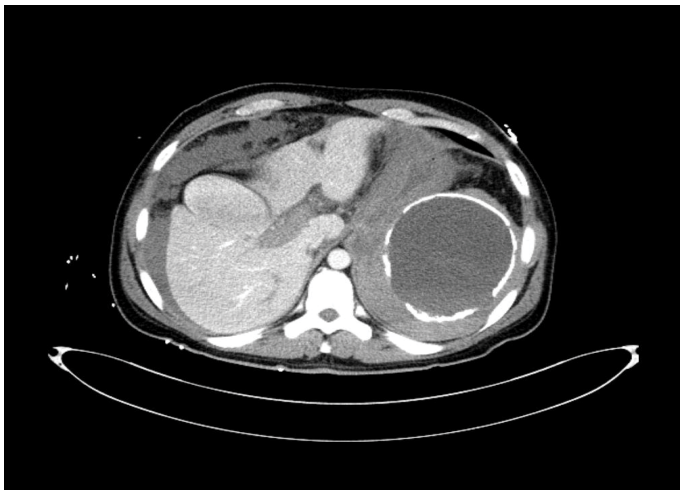

Figure 1. CT of the abdomen (venous phase) showing portal vein thrombosis and a large calcified spleen cyst.

Afterwards, upper mesenteric vein and splenic vein were identified on the lower edge of the pancreas in the lesser sac. Those branches were secured on tapes and clamped. An upper mesenteric vein venotomy and direct thrombectomy were performed using a Fogarty embolectomy catheter, including removal of thrombi from the portal vein and all branches. Blood flow through all visceral veins was restored. A calcified thrombosed splenic cyst after the injury was suspected to be a probable source of emboli. Therefore, the lienal vein was ligated to prevent recurrent thrombosis. Splenectomy was postponed after the stabilization of the patient. Venotomy was closed with PDS 6/0. Blood flow in the bowel improved and the ischemia of the bowel relieved. Estimated blood loss (EBL) during the surgery was about 1,000 ml. After the operation, the patient was transferred to the intensive care unit, his condition was stabilized, and anticoagulant treatment was started. Due to high blood losses into the drain, the patient was indicated for an early second-look six hours after emergency surgery. A surgical source of bleeding was not found in the abdominal cavity. Thus, a tamponade of the subhepatic space was performed.

The vitality of small intestine was significantly improved, the lividness of the loops disappeared, and the pulsation in the mesentery was well palpable. The patient was kept with open-abdomen and was returned to the ICU. The next day, a splenectomy was completed, including the removal of an intact cyst. Intestinal loops were without noticeable ischemia, and the portal vein was soft. Therefore, the abdominal cavity was closed. The patient was kept in ICU, in good condition. The endotracheal tube was removed the next day and early oral intake was started. After 10 days from admission, the patient was transferred from the ICU to the standard in-patient ward and was released home after another 6 days. Follow-up was conducted in the out-patient department 1, 3, 5, and 12 months after the incident. The patient is in good condition, with full peroral intake and good quality of life. Standard vaccination after splenectomy was performed. The patient underwent a comprehensive hematological examination for the presence of a congenital or acquired thrombophilic state. Factor V Leiden thrombophilia in heterozygous form has been confirmed. Anticoagulation treatment was started for six months. At present, the patient is without any problems for 12 months after the surgery. The patient has fully returned to normal life.

Results

Surgical thrombectomy is described in the literature as an extremely rare procedure in the treatment of acute portal thrombosis. Indeed, in practice, this intervention method is rarely used. We did not come across a similar documented case related to a blunt abdominal trauma. In most cases, full anticoagulant therapy is sufficient. We successfully used direct surgical thrombectomy from portal vein and its branches for the patient with portal vein thrombosis and severe small intestine infarction after a blunt abdominal trauma as the only available treatment option, because other treatment options were not suitable in this patient. This procedure is very rare, and there is not much experience with this type of intervention in abdominal surgery. For the general surgeon, it is “once in a life time” procedure. In our case, early diagnosis, right decision to choose surgical treatment, and direct surgical trombectomy resulted in success and the patient has fully returned to normal life.

Discussion

Portal vein thrombosis occurs in the presence of risk factors, most often local or congenital and acquired coagulation disorders. Local factors include inflammatory diseases, injuries, tumours in the abdominal cavity, and liver cirrhosis. Approximately 40–60 % of patients with portal vein thrombosis have been associated with a thrombophilia [3-5]. In our case, portal vein thrombosis occurred after a low-energy blunt abdominal trauma. Subsequently, a thrombophilia in this patient was confirmed. Additionally, intestinal malrotation with coiled upper mesenteric vein may also have had a partial effect. It can be assumed that the coincidence of all the following factors, including blunt abdominal trauma, Leiden mutation, and congenital intestinal malrotation, could have led to acute thrombosis of the portal vein and its branches.

Reported cases of blunt abdominal injuries being the cause of portal vein thrombosis are very rare. We found only 21 published cases in the PubMed database. However, most cases are caused by a high-energy trauma. Only about four cases, including this case, were caused by low-energy trauma (Tables 1,2).

Table 1. Etiology of portal vein thrombosis.

Localized causes	Systemic causes
Cirrhosis (28 %)	Inherited thrombophilia: Factor V Leiden mutation Prothrombin gene mutation 20210a Protein C, S, and AT III deficiency Hyperhomocysteinemia
Malignancy (27-44 %): Hepatocellular carcinoma, gastric and pancreatic adenocarcinomas, cholangiocarcinoma, lymphoma	Acquired thrombophilia: Myeloproliferative disorders, JAK2 mutation, essential thrombocythemia, polycythemia vera, myelofibrosis Antiphospholipid syndrome Pregnancy Hormonal therapy
Abdominal infections Inflammatory conditions: Pancreatitis, cholecystitis, diverticulitis, cholangitis, appendicitis, peptic ulcer perforation, inflammatory bowel diseases	
Portal vein injuries: Abdominal surgery (cholecystectomy, splenectomy, colectomy, etc.), trauma, liver transplant	Non-abdominal malignancy

Table 2. Literature review of portal vein thrombosis after blunt abdominal trauma.

Author	Type of injury	Treatment	Result
Webb (1965)	N/A	N/A	N/A
Maddrey (1968)	Traffic accident	N/A	N/A
Taylor (1978)	Traffic accident	Splenectomy	Survived
Voorhees (1979)	N/A	N/A	N/A
Gipoulou (1981)	N/A	Splenectomy	N/A
Duvoux [5] (1994)	Traffic accident	Anticoagulation	Survived
Beaufort [6] (1996)	Sport injury	Anticoagulation	Survived
Fried [7] (2002)	N/A	Small bowel resection	Survived
Gonzalez [8] (2006)	Traffic accident	Anticoagulation	Survived
Rastogi [9] (2008)	Traffic accident	Distal pancreatic resection, anticoagulation	Survived
Gopal [10] (2009)	Traffic accident	Anticoagulation	Survived
Rajkomar [11] (2010)	Fall	Anticoagulation	Survived
Collaço [12] (2010)	Traffic accident	ICU stay	Died
Sood [13] (2010)	Traffic accident	Small bowel resection, anticoagulation	Survived
Noguchi (2010)	Traffic accident	Anticoagulation	Survived

Long-term anticoagulant therapy and invasive methods such as surgical thrombectomy or local thrombolysis are generally used to treat portal vein thrombosis. However, thrombolysis is contraindicated in the case of blunt abdominal injuries. According to the European Association for the Study of the Liver [6,7], anticoagulant therapy is currently the preferred treatment method. Since 2015, this association recommends to start treatment by low-molecular-weight heparin (LMWH) with a therapeutic anti-Xa level in the range of 0.5-0.8 IU/ml. This recommendation has an A1 level in EBM (evidence-based medicine). Oral anticoagulation with Warfarin in the therapeutic INR range of 2–3 has a B1 level of evidence [8-12]. The use of new (direct) oral anticoagulants is not recommended due to the lack of extensive clinical studies and should only be given under the supervision of a hematologist [12].

Anticoagulant therapy is unsuccessful in about 10 % of cases of portal vein thrombosis [13]. If anticoagulant therapy fails, surgical thrombectomy may be considered. The efficacy of thrombectomy is best documented in liver transplant patients, where this complication is relatively common. The current trend in surgical thrombectomy is the simultaneous local thrombolysis administration, as complete thrombectomy is extremely difficult due to smaller thrombi in the periphery attached to the venous walls. Successful thrombectomy is achieved in about 30 % of cases with portal vein thrombosis. Therefore, this method is generally not recommended. However, thrombectomy may be the only chance for patients with impending or developing intestinal ischemia in acute cases. In these cases, thrombosis of other visceral veins is typically present [14,15]. The mortality rate in patients with intestinal ischemia and multiple organ dysfunction or failure is approximately 20–50 % [16,17]. Therefore, early diagnosis of portal vein thrombosis on sonography, CT scan, or MRI followed by early anticoagulant therapy or emergency surgery in extreme cases is necessary.

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