Case Report

Successful treatment of acute portal vein thrombosis extending to superior mesenteric vein with low-molecular-weight heparins: a case report

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Abstract: Portal vein thrombosis (PVT) is a rarely but potentially life-threatening disease that usually occurs in the extrahepatic portal vein (PV). Further extension to the splenic and superior mesenteric vein (SMV) causes intestinal infarction with a high mortality exceeding 50%. PVT needs individual and timely treatment, including anticoagulation, thrombolysis, surgical thrombectomy, insertion of shunts, bypass surgery, or liver transplantation in different case. Here, we present a case of acute PVT after the rectal surgery who was early diagnosed and successfully cured by low-molecular-weight heparins (LMWH) with completely asymptomatic and recanalization of the PV and SMV.

Keywords: PVT, SMVT, LMWH, anticoagulation

Introduction

As an uncommon disease with high morbidity and mortality, PVT is an abnormal thrombosis occurs in the extrahepatic portal vein and/or its branches. It also can extend concomitantly with mesenteric and/or splenic vein thrombosis (MVT/SVT) [1, 2]. Treatment of PVT currently ranges from observation with no active therapy to anticoagulation [3] or invasive approach like thrombolysis, thrombectomy [4], and transjugular intrahepatic portosystemic shunt (TIPS) placement [5]. Here we present a case of patient suffered with acute PVT and SMVT after rectal surgery who was successfully treated by low-molecular-weight heparins (LMWH).

Case presentation

An 85-year-old female patient was admitted for bleeding in the stool and diagnosed of rectal carcinoma by colonoscopy. She had a history of distal gastrectomy due to gastric ulcer bleeding 10 years ago, she takes amlodipine per day to control the blood pressure in a normal area. Both physical and laboratory examinations pre-operation were normal (Tables 1, 2). She underwent radical laparoscopic surgery (Miles') and received anti-infection, acid suppression and fluid infusion post-operation (PO). The patient began to exhaust gas from the colostomy and was given with liquid diet 2 days PO. She complained of abdominal distension and decrease of gas and stool form the colostomy 5 days PO. The colonic wall from the colostomy was mild edema accompanied with increased ascites that exceeded 2000 ml/24 hours. Symptoms were aggravated after conservative treatment (albumin supplementation, diuresis), and the patient appeared to nausea and vomiting 48 hours later; however, the signs of acute abdomen were not found. Blood tests revealed high white cell counts (14.38×10^9/L) with neutrophil counts (89.3%) and plasma D-dimer (7.41 mg/L) which were tested 9 days PO. Abdominal X-ray inspection showed some intestinal pneumatosis. She had increased and intolerable abdominal pain with tenderness and rebound tenderness in the next 24 hours and the follow-up lab findings were worse for high white blood (WBC) counts (18.54×10^9/L) and neutrophil counts (85.2%) (Table 2). The contrast enhanced computed tomography (CECT) revealed complete PVT extending to the SMVs.
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Table 1. General information

<table>
<thead>
<tr>
<th>Age</th>
<th>85</th>
</tr>
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<tbody>
<tr>
<td>Gender</td>
<td>Female</td>
</tr>
<tr>
<td>History of Basic disease</td>
<td>hypertension</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Rectal cancer</td>
</tr>
<tr>
<td>TNM stage</td>
<td>T_N_M_0</td>
</tr>
<tr>
<td>History of surgery</td>
<td>Surgical approach</td>
</tr>
<tr>
<td>Surgical approach</td>
<td>distal gastrectomy</td>
</tr>
</tbody>
</table>

Table 2. Laboratory examination

<table>
<thead>
<tr>
<th>Routine blood</th>
<th>Pre-operation</th>
<th>4 days PO</th>
<th>10 days PO</th>
<th>Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (10^9/L)</td>
<td>5.45</td>
<td>6.27</td>
<td>18.54</td>
<td>4.19</td>
</tr>
<tr>
<td>Neutrophils (10^9/L)</td>
<td>3.67</td>
<td>4.17</td>
<td>15.8</td>
<td>2.35</td>
</tr>
<tr>
<td>RBC (10^12/L)</td>
<td>4.15</td>
<td>4.1</td>
<td>4.96</td>
<td>3.62</td>
</tr>
<tr>
<td>Liver function</td>
<td>ALT (IU/L)</td>
<td>36</td>
<td>35</td>
<td>26</td>
</tr>
<tr>
<td>TB (μmol/L)</td>
<td>7.6</td>
<td>14.1</td>
<td>13.3</td>
<td>12.7</td>
</tr>
<tr>
<td>DB (μmol/L)</td>
<td>3.9</td>
<td>5.3</td>
<td>6.4</td>
<td>2.5</td>
</tr>
<tr>
<td>Coagulation function</td>
<td>PT(s)</td>
<td>11.9</td>
<td>/</td>
<td>12</td>
</tr>
<tr>
<td>APTT(s)</td>
<td>26.2</td>
<td>/</td>
<td>28.2</td>
<td>/</td>
</tr>
<tr>
<td>INR</td>
<td>1.03</td>
<td>/</td>
<td>1.04</td>
<td>/</td>
</tr>
<tr>
<td>D-dimner (mg/L)</td>
<td>0.61</td>
<td>/</td>
<td>7.41</td>
<td>/</td>
</tr>
</tbody>
</table>


The patient refused to undergo thrombectomy by surgery nor thrombolysis via TIPS because of the multiple factors as advanced age, tremendous risks, high-expense et al. We performed subcutaneous injection of low-molecular-weight heparins calcium 4100 UI q12h daily after a multidisciplinary discussion. Fortunately, the patient did not suffer any complications and completely asymptomatic after four weeks of treatment. A follow up CT scan showed partly-recanalization of the PV and SMV (Figure 2) and the patient was discharged with oral anticoagulant (warfarin) therapy for 3 months.

Discussion

Portal vein thrombosis (PVT) is an uncommon hepatic vascular disorder which has attracted more and more attention in clinic attribute to its high mortality. The chronic PVT usually occurs in the patients with liver cirrhosis, hepatocellular carcinoma (HCC) or other genetic factors like protein C deficiency, protein S deficiency et al. However, the acute PVT may be related to PV injury during surgery, stasis or hypercoagulability of blood flow [1, 6, 7]. Inexplicably, our patient accepts the rectal surgery, which would cause hemodynamic changes of the inferior mesenteric vein (IMV) but not for the PV system. We hypothesized that the risk factor in this patient was the hypercoagulability post-operation.

Acute PVT presents symptoms contains abdominal pain, abdominal distention, nausea, emesis, and fever [8-10]. These conditions may be more severe when the branches of superior mesenteric vein (SMV) were blocked which would cause intestinal necrosis. The diagnosis of PVT mainly relies on imaging examination when patients have symptoms mentioned above. Color Doppler ultrasound and CECT scan were widely used for the diagnosis of PVT, and the angiography based on contrast enhanced CT scan can precisely reveal the thrombosis and collateral vessels of PV [2, 11]. Our case suffered with severe abdominal pain and distension, nausea, vomit, tremendous ascites and was easy to diagnose.

Spontaneous recanalization of acute PVT is rare and it usually needs individual treatment due to its variability. It is necessary to hold multidisciplinary discussions among thrombosis specialists, gastroenterologists, interventional radiologists and colorectal surgeons, thus providing optimal ideas for further consideration, such as the general condition of the patient, the involvement of other branches in PV system, cirrhosis, hepatic malignancy, infec-
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Figure 1. The CECT scans 9 days after rectal surgery. Intrahepatic and extrahepatic thrombosis (red arrow) of the portal vein (A-C) extend to SMV and its branches (D) combined with severe edema (arrow head) of jejunum (E).
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Figure 2. Review of the portal vein system by CECT before discharge. The PV and SMV showed partly-recanalization when the patient was completely asymptomatic.
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tion, expense, the tolerance for complications of treatment et al. The patient in this case has severe symptoms and dramatic disease progression. Angiography by enhanced CT further confirmed the thrombus extended to SMV and its branches, and thus we advised the patient and her family to perform aggressively thrombolytic therapy by TIPS or anticoagulation with intensive observation after a multidisciplinary discussion. The patient and her family finally decided to only accept conservative treatment by LMWH. We intensively observe the changes of the disease during treatment and would perform laparotomy once the bowel necrosis occurs. Fortunately, the patient had a favorable response to the LMWH and completely relieve symptoms eventually. Surgeons might perform laparotomy at an inappropriate time by the misjudgment of intestinal infarction attribute to the severe abdominal pain and hypertension which may cause severe complications even death. Another trap of intestinal infarction was the sharp increase of WBCs. The bowel was in a state of paralytic ileus, meanwhile, amount of bacterium broke through the intestinal mucosal barrier to the blood which would result in sepsis subsequently. The operation was helpless, but might bring more complications and worse outcomes on the contrary.

Studies showed that LMWH is a safe and effective treatment. Guidelines of the American Association for the Study of Liver Diseases (AASLD) recommend an initial therapy with low-molecular-weight heparin (LMWH) which is subsequently switched to an oral anticoagulant such as warfarin [3, 7, 12]. DOACs such as rivaroxaban, apixaban, or dabigatran have been successfully adopted in PVT but only for patients who were unable to use LMWH nor warfarin [13-15]. Finally, a surgical approach needs to be considered in the treatment of PVT in patients with intestinal infarction [16].

In conclusion, management of acute PVT is complex and needs close cooperation among surgery, internal medicine, and radiology.

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Disclosure of conflict of interest

None.

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