Case Report

Pancreatic Fistula after Living Donor Liver Transplantation: A Case Report

Kenei Furukawa, Hiroaki Shiba, Shinji Onda, Taro Sakamoto, Takeshi Gocho, and Katsuhiko Yanaga

Department of Surgery, The Jikei University School of Medicine

ABSTRACT

A 49-year-old woman simultaneously underwent splenectomy and living donor transplantation owing to primary biliary cirrhosis. On postoperative day 8, a pancreatic fistula with pyrexia developed and was treated with computed tomography-guided percutaneous drainage. The dosage of a corticosteroid, which had been the initial immunosuppressive agent, was tapered, and mycophenolate mofetil was administered to encourage formation of a fistula tract. The patient made a satisfactory recovery and was discharged with a drainage tube and no graft rejection on postoperative day 43. The drainage tube was removed on postoperative day 88. Pancreatic fistula is a common complication of simultaneous splenectomy and liver transplantation; to prevent this complication, discontinuing corticosteroid administration is important.

Key words: pancreatic fistula, splenectomy, living donor liver transplantation

INTRODUCTION

Postoperative pancreatic fistula is a complication of simultaneous splenectomy and living donor liver transplantation (LDLT) which reportedly occurs in 6.5% of patients. Although corticosteroids may interfere with the healing of the fistula, immunosuppressive treatment for patients with postoperative pancreatic fistula has not, to our knowledge, previously been reported. We herein report a case of pancreatic fistula after LDLT treated through changes in the administration of immunosuppressive agents.

CASE REPORT

A 49-year-old woman had had liver dysfunction for 10 years and was found in 2010 to have primary biliary cirrhosis. The patient’s Model for End-Stage Liver Disease score became 21 (Table 1). The Model of End-Stage Liver Disease scoring system is the most widely accepted method for predicting mortality in patients with cirrhosis awaiting liver transplantation. In October 2014 the patient underwent simultaneous LDLT and splenectomy through the use of an extended left lobe graft with a caudate lobe obtained from her son. The liver and the spleen resected from the patient weighed 1,402 g and 538 g, respectively. The actual weight of the graft was 360 g, which accounted for 35.8% of the standard liver volume of the recipient. The liver graft was implanted in a conventional fashion. Splenectomy was performed before reperfusion to reduce portal hypertension. The perisplenic ligaments, including the gastrocolic, gastrosplenic, splenocolic, splenophrenic and splenorenal ligaments, were all divided with a vessel-sealing system. Usually, the splenic hilum, including the splenic artery and vein, is divided with a stapling device. However, in the present case, the splenic artery and vein were divided close to the splenic hilum because the tail of the pancreas contacted widely with the spleen (Fig. 1). The initial immunosuppressive agents were tacrolimus and a corticosteroid.

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Mailing address: Kenei Furukawa, The Jikei University School of Medicine, 3-25-8 Nishi-Shimbashi, Minato-ku, Tokyo 105-8461, Japan. E-mail: k-furukawa@jikei.ac.jp
Oral intake of food was started on postoperative day (POD) 3, and the left subphrenic drain was removed on POD 4, when the drainage fluid volume was 36 ml and the amylase concentration was 431 IU/l. On POD 8, pyrexia developed. Computed tomography (CT) revealed a fluid collection under the left hemidiaphragm (Fig. 2), which was then drained percutaneously under CT guidance. The amylase level of the aspirate fluid was 156,719 IU/l, and postoperative pancreatic fistula was diagnosed. Oral intake was discontinued, and administration of broad-spectrum antibiotics, a protease inhibitor, and somatostatin was started. To encourage the fistula to heal, the corticosteroid dosage was immediately tapered, and administration of mycophenolate mofetil was started. Follow-up CT revealed decreased fluid collection under the left hemidiaphragm; therefore, oral intake was restarted.

The patient was discharged on POD 43 with a drainage tube and no graft rejection (Fig. 3). The patient was readmitted on POD 88, and the drainage tube was removed. The pancreatic fistula did not recur. To prevent primary biliary cirrhosis from recurring after LDLT the corticosteroid was re-administered, tacrolimus was discontinued, and cyclo-

Table 1. Laboratory data

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<thead>
<tr>
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<tbody>
<tr>
<td>WBC</td>
<td>6,100/μl</td>
<td>AST</td>
<td>106 IU/l</td>
</tr>
<tr>
<td>RBC</td>
<td>281×10^6/μl</td>
<td>ALT</td>
<td>57 IU/l</td>
</tr>
<tr>
<td>Hb</td>
<td>10.2 g/dl</td>
<td>LDH</td>
<td>254 IU/l</td>
</tr>
<tr>
<td>Ht</td>
<td>30.8%</td>
<td>ChE</td>
<td>82 mU/ml</td>
</tr>
<tr>
<td>Pt</td>
<td>86×10^6/μl</td>
<td>T-Bil</td>
<td>15.7 mg/dl</td>
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<tr>
<td>PT</td>
<td>52%</td>
<td>D-Bil</td>
<td>9.0 mg/dl</td>
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<tr>
<td>PT-INR</td>
<td>1.5</td>
<td>AlP</td>
<td>1,150 IU/l</td>
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<tr>
<td>APTT</td>
<td>38.7 sec</td>
<td>γGT</td>
<td>127 IU/l</td>
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<tr>
<td>Fbg</td>
<td>357 mg/dl</td>
<td>TP</td>
<td>6.3 g/dl</td>
</tr>
<tr>
<td>AT III</td>
<td>66%</td>
<td>Alb</td>
<td>2.3 g/dl</td>
</tr>
<tr>
<td>Cr</td>
<td>0.66 mg/dl</td>
<td>UN</td>
<td>12 mg/dl</td>
</tr>
<tr>
<td>NH₄</td>
<td>86 N-μg/dl</td>
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</tbody>
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WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; Ht, hematocrit; Pt, platelet; PT, prothrombin time; INR, international normalized ratio; APTT, activated partial thromboplastin time; Fbg, fibrinogen; AT, antithrombin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; ChE, cholinesterase; T-Bil, total bilirubin; D-Bil, direct bilirubin; ALP, alkaline phosphatase; γGT, γ-glutamyltranspeptidase; TP, total protein; Alb, albumin; UN, urea nitrogen; Cr, creatinine; CRP, C-reactive protein; AMA, anti-mitochondrial antibody
Pancreatic Fistula after LDLT

As of 2 years after undergoing LDLT, the patient remains well.

**DISCUSSION**

Excessive portal hyperperfusion adversely affects liver graft function, resulting in small-for-size graft syndrome or delayed graft function after LDLT. Portosystemic shunts have been reported to decrease the volume of portal hyperperfusion in LDLT, but they have also been reported to have a negative effect represented by the portal steal phenomenon. In this regard, splenectomy has been reported to be favorable for overcoming portal decompression and small-for-size graft syndrome in LDLT. However, several complications have been reported when splenectomy is performed simultaneously with LDLT. Complications of simultaneous splenectomy and LDLT reported by Ikegami et al include pancreatic fistula (6.5% of recipients), splenic vein thrombosis (4.6% of recipients), and overwhelming postsplenectomy sepsis (1.9% of recipients). Moreover, the incidence of pancreatic fistula is greater when splenectomy has been performed during LDLT than without LDLT.
Splenectomy under laparotomy: 2.4%, laparoscopic splenectomy: 0.7%). Splenectomy performed during LDLT is complex because of splenomegaly, portal hypertension, and perisplenic collateral vessels. In addition, posttransplant immunosuppression including a corticosteroid might interfere with wound healing. Therefore, the use of immunosuppressive agents is important for treating pancreatic fistula after LDLT. To the best of our knowledge, the treatment of pancreatic fistula after LDLT has not previously been reported in the English-language literature. We successfully treated a case of pancreatic fistula after LDLT through dietary control, immediate tapering of the corticosteroid dosage, and the addition of mycophenolate mofetil to facilitate healing and to avoid acute graft rejection.

Authors have no conflict of interest.

REFERENCES