Subcapsular hematoma of the liver graft after liver transplantation: A case report

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Suphapol J, Sirichindakul B, Nonthasoot B, Nivatvongs S, Komolmit P, Poonyathawon S. Subcapsular hematoma of the liver graft after liver transplantation: A case report. Chula Med J 2009 Jul - Aug; 53(4): 309 - 14

This report describes subcapsular hematoma of the liver graft after liver transplantation due to iatrogenic parenchymal injury. A 58-year-old woman underwent cadaveric liver transplantation for HCV cirrhosis with hepatocellular carcinoma. On the second postoperative day, the patient developed acute renal failure and hypovolemic shock. Ultrasonographic finding showed huge subcapsular hematoma in the right lobe of the liver. Relaparotomy finding revealed a huge subcapsular hematoma covering the entire anterior surface of the right lobe extended to the falciform ligament. Parenchymal injury due to liver compression by self-retaining retractor might be the cause of this subcapsular hematoma which is potentiated by associated coagulopathy from cirrhosis and massive blood transfusion. This experience suggests that the liver graft must be handled with special care to prevent potential mechanical injury.

Keywords: Subcapsular hematoma, Liver transplantation.

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Received for publication. May 28, 2009.

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รายงานนี้กล่าวถึงภาวะเลือดออกใต้เยื่อหุ้มตับหลังจากการปลูกถ่ายตับในผู้ป่วยหญิงอายุ 58 ปี ซึ่งมีภาวะตับแข็งและมะเร็งตับ โดยในวันที่สองหลังผ่าตัดผู้ป่วยมีภาวะความดันโลหิตต่ำ และมี ไตวายเฉียบพลัน อัลตราซาวด์พบเลือดออกใต้เยื่อบุผิวของตับข้างขวาขนาดใหญ่ จึงได้ทำการผ่าตัด ซ้ำ โดยพบเลือดออกใต้เยื่อบุของตับ ปกคลุมด้านหน้าของตับข้างขวาทั้งหมดจนถึง falcibun ligament ซึ่งสันนิฐานว่าน่าจะเกิดจากการบาดเจ็บของเนื้อตับจากการกดทับก้าน retractor ซึ่งควรจะต้องระวัง ไม่ให้เกิดอันตรายกับเนื้อตับระหว่างการผ่าตัดเพื่อป้องกันภาวะนี้

คำสำคัญ: ภาวะเลือดออกใต้เยื่อหุ้มตับ, การปลูกถ่ายตับ.

Cadaveric liver transplantation was first introduced in Thailand at King Chulalongkorn Memorial Hospital by Dr. Sriwattanawongsa and colleagues in 1986. However, liver transplantation program was established in our unit in 1996. Nowadays, patient and graft survival have improved greatly. The operative outcomes depend on patient selection, good surgical techniques and advance immunosuppressive regimens. The main technical problems of liver transplantation are vascular and biliary complications. Parenchymal liver injury caused by technical problem has also been reported, although the incidence is low. Parenchymal liver injury may occur during organ procurement, graft implantation and postoperative graft biopsy.

Herein, the first case of subcapsular hematoma after liver transplantation in our institute is reported.

Case Report

A 58-year-old woman with underlying of HCV cirrhosis developed hepatocellular carcinoma during the follow-up period. The tumor was 5 cm in size and located at posterior-superior segment of the right lobe of the liver. However, due to decompensating liver functions, hepatic resection was contraindicated. After thorough investigation by CT chest and bone scan, no metastasis could be demonstrated. So, liver transplantation was planned as her definitive treatment. While waiting for liver transplantation, following our treatment protocol, transarterial chemoembolization was done to control the tumor growth. After the first episode of chemo-embolization, the tumor size decreased to 3 cm.

Preoperative laboratory data showed serum

glutamic oxaloacetic transaminase (SGOT) 64 u/l, serum glutamic pyruvic transaminase (SGPT) 19 u/l, alkaline phosphatase (ALP) 130 u/l, total bilirubin 4.39 mg/dl, direct bilirubin 2.13 mg/dl, albumin 2.5g/dl, globulin 4.9g/dl, prothrombin time 16.7/11.8 seconds, INR 1.4, white blood cell count 3200 cell/cc, hematocrit 31%, hemoglobin 10 g/dl, platelet 70,000, BUN 13 mg/dl and Cr 0.8 mg/dl.

Cadaveric donor was a 43-year-old male with severe head injury from car accident. Organ harvesting was done by rapid flush en bloc technique. There was no anomaly of hepatic artery. Neither hepatic parenchymal injury nor subcapsular hematoma was noted during the organ retrieval.

The recipient operation was performed via inverted Tincision. The xiphoid process was removed routinely in our operation. At laparotomy, macronodular cirrhosis and 2 hepatocellular carcinoma, one in each lobe, were identified. The one in the right lobe showed shrinkage due to effect of chemo-embolization. There was 1 liter of ascites and splenomegaly. In the total hepatectomy phase, the native liver including retrohepatic IVC was removed by using the conventional technique. After completion of vascular anastomosis, the graft was well perfused. Total ischemic time was 7 hours and 5 minutes. However, there was a small subcapsular hematoma at segment 7 with torn liver capsule. Bleeding at hematoma site was successfully controlled by argon beam coagulator and fibrin-coated collagen (TachoComb^R). So the liver capsule was not fully opened to assess the degree of parenchymal injury. Intraoperative doppler ultrasound demonstrated good hepatic artery, portal vein and hepatic vein flows. Operative blood loss was 1,200 ml.

On the first postoperative day, her condition appeared uneventful. The vital signs were stable. The patient was fully alert. Urine output was good. However, she complained of severe pain at the right hypochondrium. Her liver function tests showed markedly elevated transaminase enzymes (SGOT 5,962 u/l, SGPT 2,962 u/l). Doppler ultrasound examination revealed good flows in hepatic artery, portal vein and hepatic veins. There was a 5 cm subcapsular hematoma at the right lobe of liver. The patient also had severe thrombocytopenia (platelet count < 20,000) which was later diagnosed as immunologically induced thrombocytopenia. So, single donor, HLA matched platelet was transfused to keep platelet count > 20,000.

On the second postoperative day, the patient developed hypovolemic shock and acute renal failure. The hematocrit level fell from 30 to 25%. There was no evidence of active bleeding from abdominal drainages. The liver enzymes, particularly SGOT and SGPT, showed significant reduction (SGOT 2,211 u/ I, SGPT 2,224 u/I). Doppler ultrasound revealed large

subcapsular hematoma which compressed the parenchyma of right lobe. After fluid resuscitation, relaparotomy was performed and revealed a huge subcapsular hematoma extending from the anterior surface of the right lobe to the medial segment of the left lobe which was limited by falciform ligament (Figure 1). The capsule was opened and the blood clot was evacuated. A linear laceration of liver parenchyma on the right lobe corresponded to the position of the self-retaining retractor was identified. The bleeding raw surface was controlled by fibrincoated collagen (TachoComb^R) and argon beam coagulator (Figure 2).

The postoperative recovery was gradually improved. The patient required renal supportive dialysis until the 26th postoperative day. Her liver function tests were normalized within 4 weeks post second operation. Subsequent ultrasound examination did not show any subcapsular hematoma. The patient was discharged on the 51st postoperative day without any disability.



Figure 1. Subcapsular hematoma cover entire right lobe of liver.



Figure 2. After hematoma removal.

Discussion

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Liver transplantation is now accepted as the standard treatment of decompensating liver cirrhosis associated with hepatocellular carcinoma. Most common technical problems are vascular and biliary complications. Parenchymal liver injury occurred rarely so there were few literatures which reported the complication. We reported the first case of parenchymal injury associated with significant subcapsular hematoma after liver transplantation at our institute. This complication can occur in either donor operation or recipient operation. In donor operation, liver graft can be injured during organ retrieval. In recipient operation, liver graft can be compressed and damaged by xiphoid process or retractor. After graft perfusion, bleeding can occur and subcapsular hematoma can develop. Due to frequently associated coagulopathy and thrombocytopenia from cirrhosis and massive blood transfusion, expansion of hematoma can be potentiated. In our transplant techniques, we routinely removed xiphoid process. So, this parenchymal injury was probably due to the compression of graft by selfretaining retractor, especially at the right lobe of liver during graft manipulation for vascular reconstruction. During the first operation, we underestimated parenchymal liver injury because bleeding was successfully controlled by argon beam coagulator and fibrin-coated collagen. However, with associated coagulopathy, bleeding reappeared and stretched the Glisson capsule which might explain right hypochodriac pain of the patient. Bleeding was so extensive such that hypovolemic shock and renal failure developed.

From this experience, exploration of the

hematoma and proper assessment of the extent of liver injury might be better than observation. This strategy might prevent the hematoma to expand and reduce any complication associated with the hematoma. Some authors recommend opening subcapsular hematoma to manage liver parenchymal injury properly once it occurs. (1, 2) However, there is an interesting report by D.B. Moon and colleagues from Korea. (3) They performed transarterial embolization for pseudoaneurysm which developed after percutaneous dilatation of portal vein stenosis in LDLT and percutaneous drainage for subcapsular hematoma without reoperation successfully. So transarterial embolization and percutaneous drainage may be another alternative modality. However, patient selection for any treatment modes depends on clinical status, type of liver injury and availability of interventionist. It is advisable to follow up the hematoma during the postoperative period by imaging study. If there is any sign of expanding hematoma, proper intervention should be conducted.

In conclusion, gentle manipulation of liver graft in any phase of the operation is the most important step to prevent parenchymal injury. If subcapsular hematoma occurs, it should be opened. The entire Glisson capsule covered hematoma should be removed and all bleeding points must be controlled. This method is recommended to prevent the hematoma expansion and further associated complications.

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