Transjugular liver biopsy (TJLB) is an accepted alternative method of obtaining hepatic tissue for pathologic diagnosis in patients with parenchymal liver disease, but in whom conventional percutaneous transhepatic liver biopsy is contraindicated due to coagulopathy, a large amount of ascites, or massive obesity (1, 2). Transjugular liver biopsy is also indicated in patients with liver function deterioration following liver transplant and in patients with congenital clotting disorders (1, 2).

There are generally few complications following TJLB; the liver-puncture-related major complication rate was reported to be 0.2% in a review of 64 published reports involving 7,493 adults who had undergone TJLBs (2). We have treated 3 patients with intrahepatic pseudoaneurysms following 503 TJLBs in 320 liver transplant recipients since 2005, and report our experience for managing intrahepatic pseudoaneurysms following TJLB.

Case Reports

Case 1
A 34-year-old man who had undergone right lobe living donor liver transplantation (LDLT) 24 days previously was referred to our department for TJLB caused by deteriorating liver function. The laboratory data were as follows: serum aspartate aminotransferase, 297 IU/L; alanine transaminase, 497 IU/L; total bilirubin, 8.7 mg/dL; hemoglobin, 10.4 g/dL; platelet count, $243 \times 10^3$ /mm$^3$; and international normalized ratio (INR), 1.02. Although coagulation function was normal, the clinician in charge preferred TJLB to percutaneous transhepatic biopsy in order to avoid the risk of post-biopsy bleeding. A pre-TJLB CT revealed no abnormal findings in the liver graft. A TJLB (5 passes) using an 18-gauge Quick-Core biopsy needle (Cook, Bjaeverskov, Denmark) was performed uneventfully in the right hepatic vein. A pathology examination revealed severe canalicular and ductular cholestasis without evidence of rejection. The liver function gradually improved; however, the hemoglobin level fluctuated between 7.9 and 8.9 g/dL from 4 days post-
TJLB without evidence of internal bleeding. The usual post-LDLT follow-up CT was performed 7 days after TJLB, and a 12-mm-dimension pseudoaneurysm with a focal arteriportal shunt was noted in the inferior portion of the right hepatic vein (Figs. 1A, B). Transarterial embolization of the pseudoaneurysm was planned because of the high probability of rupture. Arteriography showed a pseudoaneurysm with an arteriportal shunt in one peripheral branch of the posterosuperior intrahepatic artery (Figs. 1C, D). The branch was then embolized through a microcatheter using a mixture (1:1; < 1 ml) of N-butyl cyanoacrylate (B. Braun, Melsungen, Germany) and lipiodol.

A post-embolization arteriogram revealed that the pseudoaneurysm and arteriportal shunt were occluded (Fig. 1E). The hemoglobin level was restored following embolization and without liver function deterioration, and the patient remains healthy.

**Case 2**

A 69-year-old woman who had undergone right-lobe LDLT 113 days previously was admitted because of an abrupt increase in liver enzyme activity. The laboratory evaluation was as follows: serum aspartate aminotransferase, 487 IU/L; alanine transaminase, 395 IU/L; total bilirubin, 3.9 mg/dL; hemoglobin, 7.1 g/dL; platelet count, $44 \times 10^3 /mm^3$; and INR, 1.13.

A pre-TJLB CT revealed no abnormal findings in the liver graft. A TJLB (six passes) in the right hepatic vein was successfully performed in an effort to determine the presence of acute rejection or hepatitis C reactivation. A pathology examination demonstrated favorable hepatitis C reactivation, and ribavirin (Robavin®; Shinpoong, Ansan, Korea) was started. A follow-up CT was performed 20 days after the TJLB due to an elevated serum bilirubin level, and a 12-mm-dimension pseudoaneurysm was found in the inferior portion of the right hepatic vein. As the patient did not have any clinical symptoms, it was considered benign. However, the patient was managed conservatively with close monitoring and further evaluation.

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Fig. 1. A, B. Arterial phase axial (A) and delayed phase coronal (B) CT images show a pseudoaneurysm (arrow) in the graft liver.
C, D. Arteriogram reveals the pseudoaneurysm (arrow) with an arteriportal shunt (arrowhead) from one peripheral branch of the posterosuperior intrahepatic artery.
E. Postembolization arteriogram shows that the pseudoaneurysm has disappeared.
F. Enhanced CT obtained 1 week following embolization shows that the pseudoaneurysm has disappeared (arrow), and lipiodol is taken up in the pseudoaneurysm.
signs or symptoms related to the presence of the pseudoaneurysm, we continued close observation as we expected spontaneous thrombosis of the pseudoaneurysm. However, the post-TJLB, 27-day follow-up CT revealed the continued presence of the pseudoaneurysm, which had slightly increased in size (Fig. 2A). Owing to the risk of worsening of the pseudoaneurysm with rupture, a hepatic arteriography was immediately performed to embolize the pseudoaneurysm. The arteriogram revealed that the pseudoaneurysm arose from a branch of the posterosuperior intrahepatic artery (Figs. 2B, C). However, super-selection of the branch using a microcatheter failed because of the small arterial diameter and acute angulation. As proximal embolization of the posterosuperior intrahepatic artery might have induced further deterioration of liver function if there were a large area of liver ischemia, percutaneous tran-shepatic puncture of the pseudoaneurysm using a 22-G Chiba needle was performed under fluoroscopy and ultrasonography guidance (Fig. 2D). One milliliter of thrombin (500 IU/mL; Reyon Pharmaceutical Co., LTD., Seoul, Korea) was then injected into the pseudoaneurysm. Completion hepatic arteriography (Fig. 1E) and CT obtained 1 day after thrombin injection showed disappearance of the pseudoaneurysm. The patient was discharged 3 days following the procedure with stable liver function; however, she died of chronic rejection 7 months later.

Case 3
A 32-year-old woman was referred to our department for TJLB and evaluation of hepatic venous outflow due to an unknown origin hyperbilirubinemia and ascites. She had undergone right-lobe LDLT 46 days previously. The laboratory data were as follows: serum aspartate aminotransferase, 48 IU/L; alanine transaminase, 62 IU/L; total bilirubin, 8.3 mg/dL; hemoglobin, 10.4 g/dL; platelet count, $33 \times 10^3$ /mm$^3$; and INR, 1.20. A pre-

![Image A](A) ![Image B](B) ![Image C](C) ![Image D](D) ![Image E](E) ![Image F](F)

Fig. 2. A. Arterial phase, coronal reformating CT shows a well-enhancing intrahepatic pseudoaneurysm [arrow]. B, C. Hepatic arteriograms show that the pseudoaneurysm [arrow] arises from a small branch [arrowhead] of the posterosuperior intrahepatic artery. D. Percutaneous puncture of the pseudoaneurysm [arrow] was performed. E. The final angiogram shows disappearance of the pseudoaneurysm. Note the relatively well-preserved intrahepatic arterial branches. F. Coronal reformating CT obtained 1 week after embolization shows that the pseudoaneurysm has disappeared.

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TJLB CT revealed no abnormal findings in the liver graft. A TJLB (three passes) and stent placement in the right hepatic vein were performed uneventfully. A pathology examination demonstrated severe cholestasis with mild centrilobular hepatocellular degeneration without evidence of rejection. A CT obtained 4 days after the TJLB to evaluate hepatic vein status revealed an 11-mm pseudoaneurysm in the anteroinferior portion of the right hepatic vein (Fig. 3A). As the patient did not have any clinical signs related to the presence of the pseudoaneurysm, close observation was performed. The 4-day follow-up CT (Fig. 3B) revealed partial thrombosis of the pseudoaneurysm, and the 18-day follow-up CT (Fig. 3C) revealed complete thrombosis of the pseudoaneurysm. The patient had an uneventful recovery and the patient remains healthy.

Discussion

A TJLB reduces the risk of hemorrhage as hepatic tissue is acquired through the hepatic vein and therefore avoids any potential liver capsule damage and results in draining of associated bleeding into the hepatic vein [3]. However, various major complications, including extra-capsular hemorrhage, hemobilia, and intrahepatic pseudoaneurysms, may occur following TJLB. The first two complications are usually detected within the first several hours following TJLB with serious bleeding. However, a pseudoaneurysm may not be detected until it ruptures or until follow-up CT or US demonstrates a pseudoaneurysm [4, 5]. A hemoperitoneum and liver hematoma associated with a pseudoaneurysm 13 days following TJLB have been reported [5]. Therefore, aggressive treatment is usually considered for treating intrahepatic pseudoaneurysms because of the risk of rupture, even though patients are hemodynamically stable (4, 6). Transarterial embolization of the bleeding or pseudoaneurysm has been shown to be a safe and effective treatment for these arterial complications (4–8). However, transarterial embolization may induce liver function deterioration with liver ischemia occurring during the early post-LDLT period as hepatic arterial flow is important for regeneration of the engrafted liver [9]. In addition, it may be technically impossible to selectively embolize only the end feeder artery if there is marked redundancy and tortuosity of the hepatic artery [9].

Therefore, we performed transarterial embolization immediately after the diagnosis of a pseudoaneurysm in only one patient with a clinical suspicion of internal bleeding. However, in the remaining two patients, our first choice for managing their pseudoaneurysms was close observation as they did not have any clinical signs related to the pseudoaneurysm and there was a risk of liver ischemia after transarterial embolization. As the pseudoaneurysm in one patient disappeared with spontaneous thrombosis, we assume that close observation may be an effective alternative for managing a pseudoaneurysm if it is relatively small in size and without rupture.

However, in the other patient, we treated the pseudoaneurysm with percutaneous thrombin injection as it is a well-documented procedure for treating femoral artery pseudoaneurysms [10]. However, percutaneous thrombin injection is not well-established for

Fig. 3. A. Contrast-enhanced CT shows a pseudoaneurysm [arrow] in the graft liver. B, C. Four- (B) and 18-day (C) follow-up CT images show gradual thrombosis of the pseudoaneurysm [arrow].
treating intrahepatic pseudoaneurysms, probably due to the risk of formation of another pseudoaneurysm or bleeding. We identified only one case report in which an intrahepatic pseudoaneurysm was treated using percutaneous transhepatic embolization with thrombin and coils in a liver transplant patient following failed, selective intraarterial embolization. However, in our patient (case 2), we assumed that percutaneous thrombin injection might be preferable to transarterial embolization in order to avoid creation of a large area of liver ischemia. As the patient’s pseudoaneurysmus was successfully treated without sequelae following the procedure, we assume that percutaneous thrombin injection is another safe alternative for treating an intrahepatic pseudoaneurysm if transarterial embolization is difficult or contraindicated.

In summary, pseudoaneurysms may occur following TJLB and may be asymptomatic. Therefore, there should be a high index of suspicion regarding the area around the biopsy site following a TJLB. Although transarterial embolization is an established and relatively safe and effective method for treating pseudoaneurysms, close observation or percutaneous transhepatic thrombin injection may be other successful therapeutic methods for treating pseudoaneurysms.

References