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The computed tomographic angiography finding of hepatic artery dissection after living donor liver transplantation; what is the clinical significance?☆

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ABSTRACT

Objective: To correlate computed tomography (CT) findings of hepatic artery dissection (HAD) to clinical manifestations of arterial insufficiency and biliary stricture after living donor liver transplantation (LDLT).
Methods: Among 737 consecutive patients, we retrospectively reviewed incidence, CT findings, and complications of the HAD.
Results: HADs occurred in 43 patients, exclusively in recipient arteries. Most were resolved with recovery of true lumen without specific treatment. Five patients had insignificant hepatic arterial infarctions, and incidence of biliary stricture did not significantly differ with control group.
Conclusions: HADs frequently occurred shortly after LDLT. Most HADs spontaneously improved and did not affect incidence of late biliary stricture.

1. Introduction

Patients receiving living donor liver transplantation (LDLT) have a high risk of vascular complications owing to the small size and complex reconstruction of vascular structures [1–4]. Vascular complications can be a major cause of mortality and morbidity after LDLT. Among many types of complications, problems related to the hepatic artery (HA) are particularly important since they can cause serious outcomes, including ischemia or necrosis of the transplanted grafts and various biliary complications [5].

Documented HA complications include HA thrombosis, anastomotic stenosis, pseudoaneurysm, arterial steal syndrome, and HA dissection (HAD) [6]. Because of the rarity of HAD, few reports exist regarding this complication [6–9]. Moreover, to the best of our knowledge, no previous report has investigated a large number of patients with HAD after LDLT. Therefore, predicting disease course or generating a treatment plan can be difficult for patients with this complication.

Our study was designed to correlate the computed tomography (CT) findings of HAD to clinical manifestations of arterial insufficiency and biliary stricture after LDLT.

2. Materials and methods

This retrospective study was approved by the institutional review board of our institution, and the requirement for informed consent was waived.

2.1. Study population

We obtained a list of 737 consecutive patients who underwent LDLT at our hospital between January 2005 and December 2007. After reviewing all radiologic reports and medical records, we selected 43 patients who were diagnosed with HAD based on postoperative CT findings. Postoperative CT scans were performed whenever there was clinical need (i.e., evaluation for bleeding focus) and otherwise obtained 1 week after the surgery as a routine work-up to screen for possible vascular complications. Follow-up CT scans were then performed according to the clinical need or at set intervals as part of a scheduled protocol. On CT scans, HAD was diagnosed when the following were detected: (a) double lumen of HA with intimal flap and opacification of both true and false lumen; (b) double lumen appearance of HA with an

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Table 1
Underlying disease and types of graft in the patient and control groups

	Patient group	Control group
Underlying disease ($n=43$)		
Liver cirrhosis associated with hepatitis B or C virus	25	25
Hepatocellular carcinoma	14	14
Toxic hepatitis	2	2
Alcoholic liver cirrhosis	1	1
Budd-Chiari syndrome	1	0
Autoimmune hepatitis	0	1
Type of graft ($n=43$)		
Right lobe	31	30
Left lobe	5	8
Dual left lobe	7	5

eccentric round-shaped hyperdensity of true lumen and a surrounding crescentic hypodensity corresponding to the false lumen; or (c) an HA of increased diameter with hypodensity (presumably, thrombosed false lumen) and faulty visualization of the lumen [6,10].

The HAD study group was composed of 26 men and 17 women (mean age±standard deviation, 49.8±8.7 years; range, 24–65 years). To compare the laboratory findings and incidence of clinically significant biliary stricture, the control group ($n=43$) was composed of patients who underwent LDLT during the same period with grafts having no vascular complication matched by sex, age, underlying disease, and year of surgery. The control group consisted of 26 men and 17 women (mean age±standard deviation, 49.2±8.9 years; range, 25–65 years). Underlying disease and type of the liver graft are summarized in Table 1.

2.2. CT protocol

CT scans were obtained on a 4-channel multidetector row CT (MDCT) scanner [LightSpeed 16, GE Healthcare, Milwaukee, WI, USA ($n=14$)] or a 16 channel-MDCT scanner [Somatom Sensation 16, Siemens Medical Solutions, Erlangen, Germany ($n=29$)]. Dynamic scanning of the liver was performed including precontrast, hepatic arterial phase, and porto-venous phase. Reconstructed slice thickness of the axial images were 2.5 mm for LightSpeed 16 scanner and 3.0 mm for Somatom Sensation 16 scanner. Three-dimensional reconstruction for HAs, volume rendering, maximum intensity projection, and multiplanar reformation techniques were performed.

2.3. CT analysis

CT data of all patients in the study population were retrospectively reviewed by two radiologists (J.Y.H and K.W.K) who were blind to the study, with conduction of a consensus reading. Each radiologist was unaware of the previous radiologic report and clinical findings of the patients.

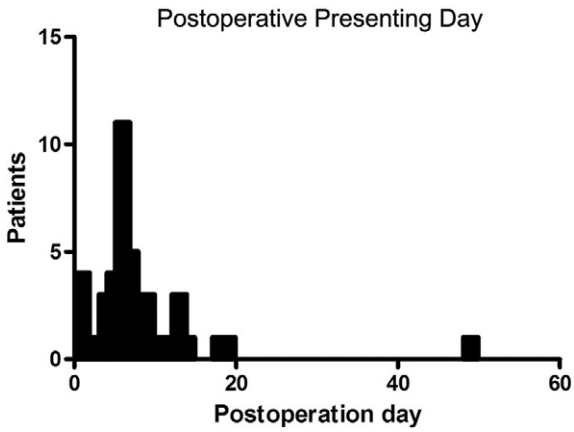

Fig. 1. Time to detection of HAD after LDLT. Bars represent the number of patients each day after LDLT.

First, postoperative presenting day of HAD was defined as the point at which HAD was detected for the first time after surgery, although it may not represent the true or exact point when HAD occurred because CT scans were not performed daily. We determined whether the involved segment was a graft HA or recipient HA, and in the latter case, we identified the extent of the lesion where HAD propagated to. We introduced a true lumen index for radiological follow-up of HAD as follows: true lumen index=diameter of true lumen/total diameter of the vessel (diameter of true lumen+false lumen). The true lumen index represents the proportion of the true lumen relative to the total cross-sectional area on CT image [11,12]. Vessel wall and thrombosed false lumen as well as true lumen were included when the total diameter of the vessel was measured. Diameters of the true and false lumens were measured at the most severely involved segment where the true lumen was most narrowed.

Second, we identified the occurrence of complications, including pseudoaneurysmal dilatation and hepatic arterial infarction. Pseudoaneurysmal dilatation was regarded as a contrast-filled protruded saccular space at the presumed recanalized false lumen of the dissected artery. Hepatic arterial infarction was considered when the liver showed segmental or subsegmental wedge-shaped, rounded, or irregularly shaped tubular areas of low attenuation lesion(s) on both hepatic arterial and portal venous phases with no abnormalities in the hepatic or portal veins [13]. Three-dimensional maximal diameters (width×height×length) of the pseudoaneurysm and the volume percentage of the hepatic arterial infarction with respect to the total volume of the liver graft were measured.

2.4. Laboratory finding analysis

To investigate the presence of acute hepatic dysfunction, peak levels of liver enzymes including aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), direct bilirubin, and total bilirubin within 7 days before and after detection of HAD were surveyed. To compare the incidence of acute hepatic dysfunction in HAD group with that of control group, time-matched data of liver enzymes were also obtained in control group.

2.5. Follow-up

We reviewed follow-up CT scans of the patients with HAD to obtain information on the radiological changes of HAD and associated findings with time course. Based on the postoperative presenting day of HAD, an organizer, who was blind to the study data, selected three follow-up CT scans for each patient for short-term at 1 week±3 days, midterm at 1 month±10 days, and long-term at 1 year±3 months. Due to the lack of follow-up CT scans in certain patients, we selected 39 CT scans for short-term follow-up, 37 for midterm follow-up, and 33 for long-term follow-up. The follow-up CT scans were performed after a mean and standard deviation, after the detection of HAD, of 7.3±2.7 days (range, 4–10) in the short-term period, 31.2±4.2 days (range, 22–40) in the midterm period, and 347.9±55.3 (range, 208–451) days in the long-term period. On each CT scan, the diameters of the true lumen and the maximal diameter of the dissected vessel were measured at the same point as that on the CT scan on postoperative presenting day of HAD. The true lumen index was calculated for each follow-up CT scan. Associated findings evaluated were pseudoaneurysmal dilatation and hepatic arterial infarction.

To evaluate the incidence of clinically significant biliary stricture during the follow-up, we reviewed radiologic reports and electric medical records. Clinically significant biliary stricture was defined as stricture detected on CT scan, ultrasonography, endoscopic retrograde cholangiography, direct cholangiography, or delayed excretion on hepatobiliary scan (DISIDA scan) which resulted abnormal status that required biliary intervention including percutaneous transhepatic biliary drainage, balloon dilatation of the bile duct, or biliary stent placement. To compare the incidence of clinically significant biliary stricture