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Case Report

RIGHT-LOBE LIVING DONOR LIVER TRANSPLANTATION FOR HEPATOCELLULAR CARCINOMA WITH DECOMPENSATED CIRRHOSIS: THE FIRST CASE AT THONG NHAT HOSPITAL

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ABSTRACT: Living donor liver transplantation (LDLT) is a potentially curative treatment for hepatocellular carcinoma (HCC) in patients with decompensated cirrhosis, particularly in countries with limited availability of deceased-donor organs. We report the first LDLT performed at Thong Nhat Hospital, Ho Chi Minh City, Vietnam. A 50-year-old man had a solitary 4.8-cm HCC within the Milan criteria on a background of Child–Pugh class C cirrhosis (MELD score, 18) associated with hepatitis B and C virus coinfection. He also had type 2 diabetes mellitus. His healthy 29-year-old brother, who was ABO-compatible, served as the living donor, and a right-lobe graft was used. Clinical, biochemical, and imaging findings were followed from the preoperative period through postoperative day (POD) 16. The operation was completed in 8 hours. Serum lactate decreased substantially within the first 12 hours and normalized by POD 2. Aspartate aminotransferase peaked at 474 U/L on POD 2 and alanine aminotransferase at 626 U/L on POD 3, followed by a progressive decline. Between POD 7 and POD 10, a secondary increase in aminotransferases, gamma-glutamyl transferase, and direct bilirubin occurred. Doppler ultrasonography and computed tomography (CT) showed no vascular thrombosis or biliary dilatation. CT demonstrated mild hepatic artery anastomotic narrowing, while the available Doppler measurements did not suggest a clear hemodynamic impact; drain-fluid bilirubin did not suggest a bile leak. Biochemical indices improved after optimization of immunosuppressive therapy; however, liver biopsy was not performed to confirm rejection. By POD 16, prothrombin activity was 82% and the platelet count was 122 × 10^9/L; the most recent serum albumin level was 42.7 g/L on POD 15. The donor recovered uneventfully and was discharged on POD 12. No vascular thrombosis, bile leak, severe infection, or biopsy-proven rejection was documented during the observation period. This case demonstrates the initial feasibility of establishing an LDLT program at Thong Nhat Hospital. Multidisciplinary preparation and technical collaboration with 108 Military Central Hospital were important contributors to the favorable early outcome. Longer follow-up and additional cases are required to establish the safety and effectiveness of the program.

Keywords: living donor liver transplantation; hepatocellular carcinoma; decompensated cirrhosis; case report; technology transfer

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1. INTRODUCTION

Hepatocellular carcinoma (HCC) arising in end-stage cirrhosis is one of the most common indications for liver transplantation worldwide. Living donor liver transplantation (LDLT) is particularly important in Asia, where organs from brain-dead donors remain scarce [1,2]. In Vietnam, the first liver transplantation was performed in 2004 at Military Hospital 103, Hanoi [3]. By early 2026, more than 750 liver transplantations had been performed in Vietnam [4]. However, the procedure is still performed mainly at a few large tertiary hospitals. Before the transplantation at Thong Nhat Hospital, six institutions in southern Vietnam performed liver transplantation, all of them in Ho Chi Minh City [5]. Thong Nhat Hospital is a Grade I general hospital under the Ministry of Health, with particular strength in the care of older patients and patients with multiple comorbidities. In 2024, Thong Nhat Hospital and 108 Military Central Hospital signed an agreement on the transfer of liver transplantation techniques. After preparation of personnel, facilities, and procedures, the first LDLT was performed on 22/08/2026. The case had several notable features: the recipient had a complex clinical picture, with HCC on a background of decompensated cirrhosis due to hepatitis B and C virus coinfection, together with type 2 diabetes mellitus, and the entire procedure was carried out under a technology transfer model from a highly experienced center. Reports of the first liver transplantation at new centers in Vietnam are scarce in the literature, whereas experience from the initial phase of a program is a useful reference for institutions building similar capacity. This article describes in detail the preoperative, operative, and postoperative course of the first liver transplantation at Thong Nhat Hospital.

2. CASE PRESENTATION

2.1. Recipient

The patient was a 50-year-old man (born in 1976) with a height of 155 cm, a weight of 53 kg, and a BMI of 22.06 kg/m^2. He had a history of hepatitis B and C virus coinfection that had progressed to Child–Pugh class C cirrhosis, with multiple hospitalizations for ascites and gastrointestinal bleeding from ruptured esophageal varices. He had type 2 diabetes mellitus on treatment. One month before admission, a liver mass was detected at a routine checkup, and he was advised to be admitted to prepare for liver transplantation.

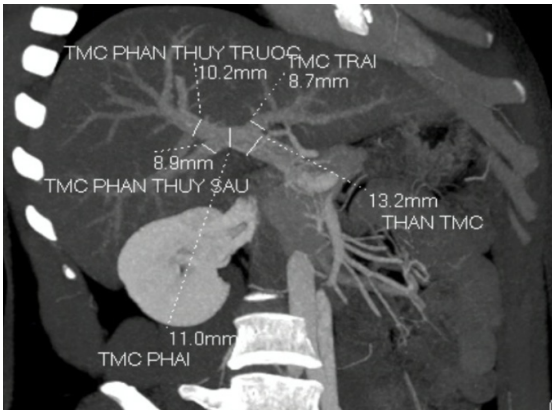
On admission, the patient was alert and hemodynamically stable, with grade 0 hepatic encephalopathy, jaundice, and ascites. Preoperative tests showed a white blood cell count of 1.9 × 10^9/L, platelet count of 38 × 10^9/L, hemoglobin 10.8 g/dL, prothrombin 60%, INR 1.47, albumin 33.9 g/L, total bilirubin 29.9 µmol/L, direct bilirubin 14.4 µmol/L, AST 45 U/L, ALT 27 U/L, AFP 4.4 ng/mL, PIVKA-II 111 mAU/mL (elevated), positive HBsAg, HBV-DNA below 10 IU/mL (on antiviral therapy), positive anti-HCV, undetectable HCV-RNA, and blood group B Rh(+). The preoperative MELD score was 18. Contrast-enhanced abdominal CT showed a strongly enhancing 4.8-cm nodule in the right lobe, consistent with the diagnostic criteria for HCC, together with cirrhosis, splenomegaly, large-volume ascites, and no portal vein thrombosis. Esophagoscopy showed previously ligated esophageal varices (banding scars) and portal hypertensive gastropathy.

2.2. Donor

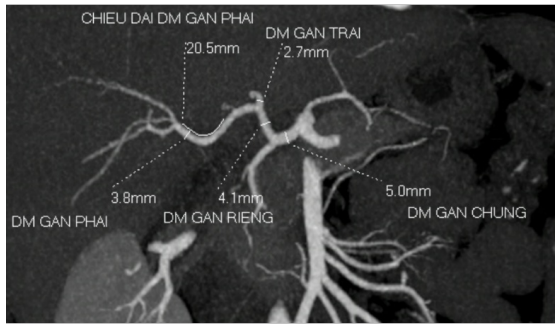
The donor was the patient's younger brother, aged 29 years (born in 1997), with a height of 166 cm, a weight of 72 kg, and a BMI of 26.1 kg/m^2. He was previously healthy and had never been hospitalized. His blood group was O Rh(+) (recipient B Rh(+); ABO-compatible). The cross-match was negative, and anti-HLA class I and II antibodies were both negative. Liver function tests, hematology, and serological screening (CMV, EBV, HIV, tuberculosis) were all within normal limits.

Preoperative imaging: multiphase contrast-enhanced abdominal CT and contrast-enhanced MRI were performed to assess liver volume and the donor's vascular and biliary anatomy. CT volumetry showed a total liver volume of 1,514.1 mL; with the transection plane along the left border of the middle hepatic vein, the right lobe measured 1,061.6 mL (70%) and the left lobe 452.5 mL (30%). With the transection plane along the right border of the middle hepatic vein, the right lobe measured 971.4 mL (64.2%) and the left lobe 542.7 mL (35.8%). The estimated pre-

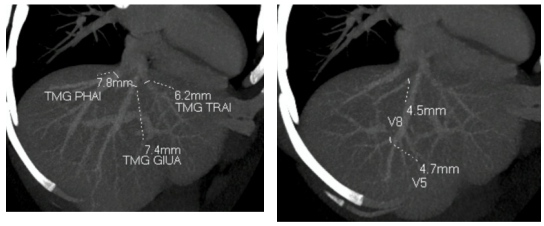
operative graft-to-recipient weight ratio (GRWR) was 2.0%, above the threshold commonly used to limit the risk of small-for-size syndrome. The donor's expected remnant liver volume was 30.0–35.8%, within the limits generally accepted in the evaluation of right-lobe donors [1]. Portal venous anatomy (Figure 1A) was standard (type I). The main portal vein measured 13.2 mm in diameter and bifurcated into the right (11.0 mm) and left (8.7 mm) branches. The right portal vein further divided into the anterior sectoral branch (10.2 mm) and the posterior sectoral branch (8.9 mm). Hepatic arterial anatomy (Figure 1B) was standard (Michel type I). The common hepatic artery measured 5.0 mm in diameter and continued as the proper hepatic artery (4.1 mm), which divided into the right hepatic artery (3.8 mm; extraparenchymal length, 20.5 mm) and the left hepatic artery (2.7 mm). Hepatic venous anatomy (Figure 1C) showed three separate hepatic vein trunks draining into the inferior vena cava: right hepatic vein, 7.8 mm; middle hepatic vein, 7.4 mm; and left hepatic vein, 6.2 mm. The veins draining segment V (V5, 4.7 mm in diameter) and segment VIII (V8, 4.5 mm in diameter) emptied into the middle hepatic vein. Biliary anatomy (Figure 1D): MRCP showed a right hepatic duct 0.25 cm in diameter with an extraparenchymal length of 0.20 cm, and a left hepatic duct 0.32 cm in diameter. Hepatic steatosis (Figure 1E): MRI-PDFF quantification in regions of interest placed in the liver parenchyma, avoiding large vessels, bile ducts, and artifacts, showed a mean fat fraction of 7.3%.



A: Portal vein.



B: Hepatic artery.

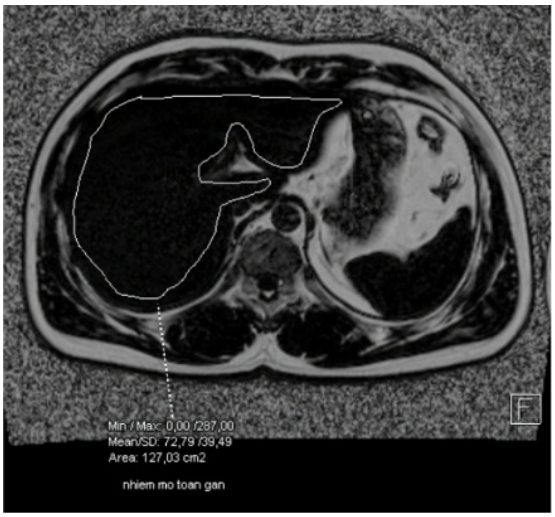


C: Hepatic veins

(three main trunks and the V5 and V8 branches).



D: Bile ducts on MRCP.



E: MRI measurement of hepatic steatosis.

Figure 1. Reconstructed CT/MRI images of the donor's vascular and biliary anatomy.

2.3. Surgery

On 22/08/2026, two surgical teams operated simultaneously: team 1 procured the right-lobe graft, and team 2 performed the recipient hepatectomy and then the implantation. The operation was completed in 8 hours as a joint effort of Thong Nhat Hospital and 108 Military Central Hospital. The actual graft weight was 850 g, corresponding to a GRWR of 1.60%. The cold ischemia time and the recipient warm ischemia time were 75 minutes and 50 minutes, respectively. During surgery, the recipient received a total of 5,150 mL of blood products, including 6 units of packed red blood cells, 13 units of fresh frozen plasma, 4 units of cryoprecipitate, and 1 unit of apheresis platelets. After surgery, both patients were transferred to the intensive care unit. Postoperative pathology confirmed poorly differentiated hepatocellular carcinoma on a background of METAVIR stage F4 cirrhosis. The tumor showed capsular invasion, intrahepatic vascular invasion, and perineural invasion. Table 1 summarizes the key events from the preoperative period to postoperative day (POD) 16, following the CARE guideline [6].

Table 1. Timeline of key events in the case.

Time	Event
1 month before admission	Liver mass detected at a routine checkup; liver transplantation recommended
17/08/2026	Hospital case conference; legal documents completed; pretransplant evaluation
22/08/2026	Right-lobe LDLT, operative time 8 h; graft 850 g, GRWR 1.60%; cold ischemia 75 min, warm ischemia 50 min
POD 1	AST and ALT began to rise (ischemia–reperfusion injury); lactate fell from 9.6 to 2.49 mmol/L after 12 h
POD 2	AST peaked at 474 U/L; lactate below 2 mmol/L
POD 3	ALT peaked at 626 U/L
POD 4	Walker-assisted mobilization started; first tacrolimus trough level (0.8 ng/mL)
POD 6	AST down to 58 U/L, total bilirubin 34 µmol/L; patient started eating solid food
POD 7–10	Secondary rise in aminotransferases, GGT, and direct bilirubin
POD 8	Doppler ultrasonography and CT: no vascular thrombosis, no biliary dilatation, mild hepatic artery anastomotic narrowing; drain fluid tests did not suggest a bile leak
POD 10	Total bilirubin peaked at 118 µmol/L; suprahepatic drain removed; walking without a walker; tacrolimus level 6.9 ng/mL
POD 11–16	Gradual improvement in liver biochemistry; tacrolimus levels 6.9–9.3 ng/mL
POD 12	Donor discharged without complications
POD 16	End of follow-up in this report; platelets 122 × 10^9/L; prothrombin 82%, albumin 42.7 g/L

2.4. Postoperative course in the recipient

The patient was admitted to the intensive care unit at 17:00 on 22/08/2026. He was alert, with a pulse of 120 beats/min, blood pressure of 140/80 mmHg, and SpO_2 of 99% on oxygen at 1 L/min via nasal cannula. The suprahepatic drain yielded 20 mL and the subhepatic drain 170 mL

of bloody fluid. The postoperative course was divided into three phases based on clinical and laboratory changes. Trends in liver biochemistry, tacrolimus trough levels, and INR are shown in Figure 2.

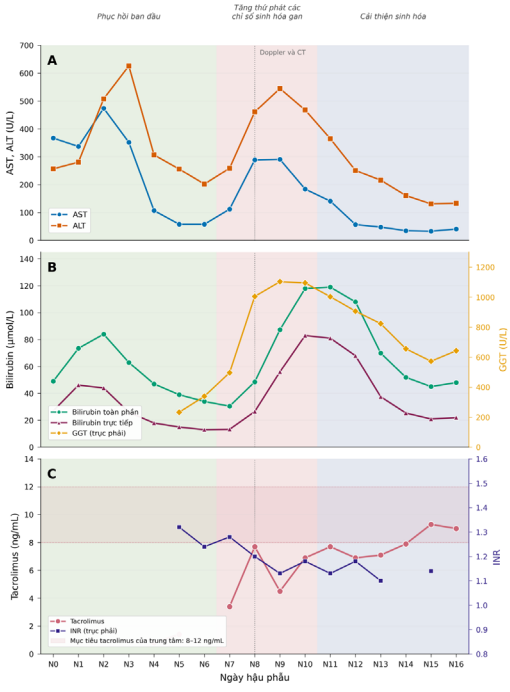


Figure 2. Trends in liver biochemistry, tacrolimus trough levels, and INR (POD 0 to POD 16). (A) AST and ALT. (B) Total and direct bilirubin (left axis) and GGT (right axis). (C) Tacrolimus (left axis) and INR (right axis); horizontal band: target range of 8 to 12 ng/mL. Shaded backgrounds mark the three clinical phases. Vertical dotted lines: Doppler ultrasonography and CT (POD 8) and removal of the suprahepatic drain (POD 10). Daily values were taken from the sample closest to 6:00 (POD 0 and POD 11: 9:00 samples).

2.4.1. Phase 1: Ischemia–reperfusion injury and early recovery (POD 0–6)

Blood lactate was initially 9.6 mmol/L, decreased to 2.49 mmol/L after 12 hours, and remained below 2 mmol/L from POD 2. Hemodynamics were stable throughout this phase, and imaging confirmed preserved graft perfusion. Liver enzymes rose in the typical pattern of ischemia–reperfusion injury: AST peaked at 474 U/L on POD 2 (6:00, 24/08) and ALT at 626 U/L on POD 3 (6:00, 25/08), then decreased gradually to 58 U/L and 202 U/L, respectively, on POD 6. Total bilirubin rose from 49 µmol/L (day of surgery) to 93.5 µmol/L (12:00, POD 1) and then fell steadily to 34 µmol/L on POD 6. The platelet count reached its nadir of 36 × 10^9/L (6:00, POD 1) and began to recover from POD 7. Fluid balance was negative from POD 3, and body weight was stable at 52 kg. Tacrolimus was started according to the protocol. The first trough level, measured on POD 4 (6:00, 26/08), was 0.8 ng/mL, and the level on POD 5 was 1.4 ng/mL, below the target therapeutic range of 8 to 12 ng/mL for the early post-transplant period.

2.4.2. Phase 2: Secondary rise in liver enzymes and bilirubin (POD 7–10)

From POD 7, graft function parameters worsened. AST rose from 58 (POD 6) to 112 (POD 7) and 289 (POD 8), peaking at 291 U/L on POD 9. ALT rose from 202 (POD 6) to 259 (POD 7) and 461 (POD 8), peaking at 545 U/L on POD 9. Total bilirubin rose from 30.5 (POD 7) to 48.5 (POD 8) and 87.3 (POD 9), peaking at 118 µmol/L on POD 10, with direct bilirubin predominating (83/118 µmol/L; 70%). GGT rose sharply from 339 (POD 6) to 1,103 U/L (POD 9). The rise in aminotransferases was accompanied by marked increases in GGT and direct bilirubin: GGT exceeded 20 times the upper limit of normal, and direct bilirubin accounted for 70% of total bilirubin (83/118 µmol/L). During this phase, tacrolimus trough levels remained low: 3.4 ng/mL (POD 7), 7.7 ng/mL (POD 8), 4.5 ng/mL (POD 9), and 6.9 ng/mL (POD 10). The tacrolimus dose was kept at 10 mg/day (in 2 divided doses at 8:00 and 20:00, taken 2 hours after meals), and trough levels were measured at 7:30 each day. Doppler ultrasonography on 30/08 (POD 8) showed a peak systolic velocity (PSV) of 80 cm/s and a resistive index (RI) of 0.65 in the hepatic artery at the anastomosis; the intraparenchymal hepatic artery branches had a PSV of 46 cm/s and an RI of 0.73. Maximum portal vein velocity was 60 cm/s at the anastomosis and 38 and 56 cm/s proximal and distal to the anastomosis, respectively; the anastomotic diameter was 9.7 mm, with an estimated flow of 1,659 mL/min. The right hepatic vein showed a biphasic waveform; the reconstructed middle hepatic vein was patent (48 cm/s); and the V5 and V8 branches were patent, with maximum anastomotic velocities of 58 and 54 cm/s, respectively. CT on the same day showed mild narrowing of the hepatic artery anastomosis and relatively homogeneous enhancement of the graft

parenchyma, with no hepatic artery, portal vein, or hepatic vein thrombosis. The intrahepatic bile ducts were not dilated. Associated findings included periportal edema and a small right pleural effusion. Drain fluid analysis (18:00, POD 8) showed LDH 263 U/L, albumin 34.1 g/L, bilirubin 61.7 µmol/L, glucose 12.6 mmol/L, triglycerides 0.612 mmol/L, and amylase 66.1 U/L. Drain fluid bilirubin was lower than the simultaneous serum bilirubin (79 µmol/L), which did not suggest a bile leak. CRP was mildly elevated (10.4 mg/L on POD 8), but the patient was afebrile and procalcitonin was normal. CMV prophylaxis with intravenous ganciclovir had been started on POD 2 according to the protocol and was switched to oral valganciclovir on POD 9 (both donor and recipient were CMV IgG-positive before transplantation; intermediate-risk category).

2.4.3. Phase 3: Improvement in liver biochemistry and stabilization of graft function (POD 11–16).

From POD 11 to POD 16, tacrolimus trough levels ranged from 6.9 to 9.3 ng/mL and reached the center's target range on POD 15–16. At the same time, liver biochemistry improved markedly. AST decreased from 184 (POD 10) to 57 (POD 12), 33 (POD 15), and 41 U/L (POD 16). ALT decreased from 468 (POD 10) to 365 (POD 11), 251 (POD 12), 161 (POD 14), and 131 (POD 15). Total bilirubin decreased from 118 (POD 10) to 108 (POD 12), 70 (POD 13), 52 (POD 14), and 45 (POD 15). GGT decreased from 1,095 (POD 10) to 906 (POD 12), 656 (POD 14), and 573 (POD 15). Prothrombin remained stable at 78% to 86%, with an INR of 1.10 to 1.18. Albumin stayed above 40 g/L (42.7 g/L on POD 15). The platelet count showed a recovering trend: 96 (POD 10), 117 (POD 13), 110 (POD 15), and 122 × 10^9/L (POD 16). Clinically, the patient remained afebrile throughout the 16 postoperative days. Pruritus appeared on POD 10 (night of 01/09), responded poorly to antihistamines, and gradually subsided from POD 12 as bilirubin decreased. The suprahepatic drain was removed on POD 10 (18:00, 01/09), and subhepatic drain output decreased gradually from 630 mL/24 h (POD 8) to 245 mL/24 h on POD 16. Blood glucose was controlled with intravenous insulin infusion (2 to 4 mL/h) and ranged from 5.2 to 18.1 mmol/L. The patient began mobilizing with a walker on POD 4, started eating solid food on POD 6, walked without a walker from POD 10, and did physical therapy with elastic resistance bands 2 times/day from POD 10. Inspiratory volume measured by spirometer reached approximately 1,500 mL on POD 10 (18:00, 01/09).

The immunosuppressive regimen consisted of induction therapy with basiliximab (20 mg intravenously on POD 0 and POD 4) combined with high-dose intravenous methylprednisolone (240 mg on the first day, tapered to 40 mg/day from POD 12). Maintenance therapy consisted of tacrolimus (started on POD 4, with the dose increased according to trough levels; target 8 to 12 ng/mL) and mycophenolate mofetil (started at 500 mg/day on POD 5 and increased to 1,000 mg/day from POD 8). Prophylaxis against HBV recurrence consisted of continued tenofovir alafenamide 25 mg/day. Antibacterial prophylaxis consisted of meropenem combined with vancomycin (vancomycin was stopped on POD 2). Antifungal prophylaxis consisted of intravenous caspofungin, switched to oral fluconazole from approximately POD 12. CMV prophylaxis consisted of intravenous ganciclovir from POD 2, switched to oral valganciclovir from POD 9 (both donor and recipient were CMV IgG-positive before transplantation).

2.5. Postoperative course in the donor

The donor recovered uneventfully after surgery, with no bleeding, bile leak, infection, or major surgical complications during hospitalization. He was discharged on POD 12 and continued outpatient follow-up.

2.6. Patient perspective

On POD 16, the recipient reported that he was satisfied with his treatment, felt that his physical strength had clearly improved compared with before transplantation, and was able to manage his own personal care. The donor reported a good recovery after discharge and had no abnormal symptoms at the follow-up visit.

3. DISCUSSION

The first LDLT at Thong Nhat Hospital was performed at a time when liver trans-

plantation in Vietnam is developing rapidly. From the first liver transplantation in 2004 [3] to early 2026, more than 750 liver transplantations have been performed nationwide [4]. However, most liver transplantation centers in southern Vietnam are concentrated in large tertiary hospitals. With this case, Thong Nhat Hospital became the seventh institution in southern Vietnam to perform liver transplantation [5].

Regarding the case characteristics, the recipient had a complex clinical picture of HCC on a background of cirrhosis due to hepatitis B and C virus coinfection, together with type 2 diabetes mellitus. In a patient with cirrhosis who is not suitable for liver resection, a solitary 4.8-cm HCC without macrovascular invasion or extrahepatic metastasis falls within the Milan criteria and is a classic indication for liver transplantation [7]. However, the explanted liver showed poorly differentiated HCC with intrahepatic vascular invasion, perineural invasion, and capsular invasion. These findings indicate that the pretransplant morphological assessment did not fully reflect the adverse histopathological features of the tumor. The pretransplant AFP of 4.4 ng/mL and PIVKA-II of 111 mAU/mL need to be interpreted together with imaging and the clinical context and were not sufficient to identify invasive features in advance. Tumor biopsy may be considered when imaging does not meet the noninvasive diagnostic criteria or when the histopathological result is expected to change the treatment decision after multidisciplinary discussion. However, liver tumor biopsy is subject to sampling error and tumor heterogeneity, so it does not guarantee full identification of the degree of differentiation or invasive features. Because of these limitations, the indication for pretransplant tumor biopsy should be individualized according to the imaging findings, the likelihood that histopathology will change management, and the procedural risk. This case shows the need for pretransplant oncological risk assessment and long-term post-transplant surveillance for recurrence, based on combined clinical data, HCC biomarkers, imaging, and histopathology.

On the donor side, both the vascular and biliary anatomy were standard (portal vein type I, hepatic artery Michel type I), which was favorable for the first transplantation at a new center. The right hepatic artery, 3.8 mm in diameter with an extraparenchymal length of 20.5 mm, provided a stump of sufficient length for microsurgical anastomosis. Although V5 and V8 measured 4.7 and 4.5 mm in diameter, respectively, the decision to reconstruct middle hepatic vein drainage was based on an overall assessment of the drainage territory and the risk of anterior sector congestion. The principle of reconstructing significant middle hepatic vein tributaries to limit congestion of right-lobe grafts has been described in the modified right liver graft technique [8]. The short right hepatic duct (2 mm on MRCP) required precise division of the bile duct at the parenchymal transection plane to avoid insufficient length for the anastomosis. MRI-PDFF showed a mean hepatic fat fraction of 7.3%; this result was considered together with the remnant liver volume, the vascular and biliary anatomy, and other donor evaluation data in selecting the graft.

The postoperative course had three distinct phases. In the first phase (POD 0–6), aminotransferases rose early and then declined gradually, consistent with post-transplant preservation–reperfusion injury. Blood lactate decreased from 9.6 mmol/L to 2.49 mmol/L within the first 12 hours and remained below 2 mmol/L from POD 2, with stable hemodynamics and graft perfusion. When assessed against the widely used definition of early allograft dysfunction (EAD) by Olthoff et al., the patient met none of the criteria: total bilirubin on POD 7 was 30.5 µmol/L (1.78 mg/dL), INR on POD 7 was 1.28, and peak AST and ALT within the first 7 days were 474 and 626 U/L, respectively [9]. Thus, EAD did not occur in the first week after transplantation. However, the secondary rise in liver tests from POD 7 to POD 10 occurred after an initial period of improvement and was assessed as a separate event.

The secondary rise in aminotransferases, GGT, and direct bilirubin from POD 7 to POD 10 raised a differential diagnosis that included vascular complications, biliary complications, acute cellular rejection, drug-induced liver injury, and viral infection [10]. Under the center's protocol, the target tacrolimus trough level in the early period is 8–12 ng/mL. From POD 7 to POD 10, drug levels ranged from 3.4 to 7.7 ng/mL and were unstable, with several values below 6 ng/mL. A meta-analysis showed

that trough levels of 6–10 ng/mL in the first month may balance the risks of rejection and nephrotoxicity better than higher levels [11]. Doppler ultrasonography and CT on POD 8 showed no vascular thrombosis or biliary dilatation; drain fluid bilirubin was lower than the simultaneous serum bilirubin, which did not suggest a bile leak. However, CT showed mild narrowing of the hepatic artery anastomosis, so the possibility that this lesion contributed to the secondary liver biochemical abnormalities must be considered. Doppler showed an anastomotic PSV of 80 cm/s, an anastomotic RI of 0.65, and an intraparenchymal RI of 0.73. Assessment of hepatic artery stenosis after transplantation requires combined evaluation of flow velocity, RI, and waveform; a tardus–parvus waveform is usually defined by an RI <0.5 and a systolic acceleration time >0.08 seconds. The available parameters did not suggest a clear hemodynamic effect of the narrowing at the time of examination; CT also showed relatively homogeneous enhancement of the graft parenchyma. However, the imaging reports did not provide the degree of narrowing, the length of the narrowed segment, the systolic acceleration time, or a full description of the intraparenchymal arterial waveform, so the significance of this lesion cannot be fully assessed. The elevated GGT and direct bilirubin raised the possibility of biliary injury, but the evidence was insufficient to establish ischemic biliary injury. Therefore, a contribution of the hepatic artery anastomotic narrowing to the rise in liver enzymes can be neither confirmed nor completely excluded, and Doppler findings should be followed together with biochemical indices. The biochemical improvement that coincided with optimization of immunosuppressive therapy shows only a temporal association and is not sufficient to establish the cause. Graft biopsy is used to confirm and grade rejection according to the Banff system [12]. Because no biopsy was performed, acute rejection was not confirmed, and the cause of the liver biochemical abnormalities on POD 7–10 remains unclear.

The aminotransferase trend in this case was consistent with the pattern of preservation–reperfusion injury described in liver transplantation, in which AST and ALT usually peak within the first 7 days after surgery [9]. AST peaked at 474 U/L on POD 2 and ALT at 626 U/L on POD 3, both below the 2,000 U/L threshold in Olthoff's diagnostic criteria for early allograft dysfunction [9], indicating mild preservation–reperfusion injury. The reported incidence of acute rejection after liver transplantation is approximately 20% to 40%, mostly in the first month [14]. In LDLT, the multicenter A2ALL study reported biopsy-proven acute rejection in 27% of recipients [15]. Biliary complications are the most important technical challenge after LDLT, with an incidence of 15% to 30%, including bile leak (11.4%) and biliary stricture (20.6%) in an international multicenter study of 3,633 cases [16]. In this case, no clinically significant bile leak or biliary stricture was observed during the 16 days of follow-up; however, late biliary complications need to be assessed during further follow-up.

Experience from centers starting LDLT shows that outcomes improve along a learning curve. Kim et al. analyzed the first 51 LDLTs at a new cancer center in Korea, comparing the first 17 cases performed with technical support from an external team with the next 34 cases performed independently, and reported no in-hospital deaths and a gradual improvement in outcomes over time [17]. Our case, with a favorable early postoperative outcome achieved with technical support from 108 Military Central Hospital [4,13], is similar to the initial phase of the technology transfer model described by Kim et al. [17].

The collaboration between Thong Nhat Hospital and 108 Military Central Hospital, based on the technology transfer agreement signed in 2024, gave the local team direct access to the experience of a center that has performed more than 300 liver transplantations, including LDLT in 51 patients with acute-on-chronic liver failure whose outcomes have been reported in the international literature [4,13]. This model allows a new hospital to start liver transplantation with direct technical support from a highly experienced center. Other healthcare institutions in Vietnam that are building organ transplantation programs may consider a similar approach.

4. CONCLUSION

The first right-lobe LDLT at Thong Nhat Hospital achieved a favorable early postoperative outcome. By POD 16, graft synthetic function was stable, and no major

surgical complications were observed. This case demonstrates the initial feasibility of a model for establishing a liver transplantation program based on multidisciplinary preparation and technology transfer from an experienced center. Combined monitoring of graft function indices, tacrolimus trough levels, and vascular and biliary imaging supported the evaluation of the liver biochemical abnormalities on POD 7–10. The mild hepatic artery anastomotic narrowing on CT requires further follow-up, although the available Doppler parameters did not suggest a clear hemodynamic effect. The adverse histopathological features of the explanted liver point to the need for risk stratification and surveillance for HCC recurrence after transplantation. However, this report has several limitations. The follow-up period was only 16 days, which does not allow assessment of medium- and long-term graft function, late biliary complications, the risk of HCC recurrence, or post-transplant quality of life. In addition, the report describes a single case, so long-term follow-up and more cases are needed to evaluate the long-term safety and effectiveness of the program.

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