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Technical overview of pediatric liver transplantation for congenital absence of the portal vein: a collective review of six cases at a single center

Jung-Man Namgoong, Shin Hwang

Department of Surgery, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

Congenital absence of the portal vein (CAPV) is a rare vascular anomaly in which mesenteric venous blood bypasses the liver and drains directly into systemic circulation. Liver transplantation (LT) is considered for patients who develop severe complications or do not respond to medical therapy. Because splanchnic venous anatomy varies markedly among patients with CAPV, portal vein (PV) reconstruction must be customized to individual vascular patterns. We reviewed surgical approaches to PV reconstruction in six pediatric LT cases with CAPV reported from a single institution. Patients ranged in age from 3 years 5 months to 12 years at the time of LT. Portocaval shunts (PCS) were classified as extrahepatic in five cases and intrahepatic in one case. Portal hypertension was identified in three patients. Sources of portal inflow included the PCS vein, native extrahepatic PV trunk, enlarged PV collateral vein, pericholedochal collateral vein, and confluence of the mesenteric-splenic vein. All patients recovered without vascular complications and remained alive, with follow-up ranging from 3 to 11 years. In conclusion, CAPV is frequently associated with complex vascular anomalies. Careful preoperative and intraoperative assessment is essential, and PV reconstruction must be individually tailored with anatomically appropriate techniques to achieve successful outcomes.

Keywords: Portocaval shunt; Portosystemic shunt; Splenorenal shunt; Portal hypertension; Living donor liver transplantation

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Corresponding author: Shin Hwang
Department of Surgery, Asan Medical Center, University of Ulsan College of Medicine, Olympic-ro 43-gil 88, Songpa-gu, Seoul 05505, Korea
E-mail: shwang@amc.seoul.kr

INTRODUCTION

Congenital absence of the portal vein (CAPV) is a rare vascular anomaly in which mesenteric venous blood bypasses the liver and drains directly into systemic circulation. Most individuals with CAPV are asymptomatic, showing only mild abnormalities in liver function tests without overt signs of portosystemic encephalopathy. Liver transplantation (LT) is generally reserved for patients who fail to respond to medical management, particularly when severe complications arise, such as hyperammonemia, portosystemic encephalopathy, hepatopulmonary syndrome, hepatic neoplasms, or other refractory clinical problems [1–4].

In a congenital portocaval shunt (PCS), mesenteric venous blood is diverted either directly into the inferior vena cava (IVC) or indirectly through a splenorenal shunt (SRS) via the left renal vein. This aberrant vascular path-

HIGHLIGHTS

- We reviewed the surgical approaches to portal vein reconstruction in six pediatric liver transplant recipients with congenital absence of the portal vein reported between 2021 and 2023 from a single institution.
- Thorough preoperative and intraoperative assessment is critical, and portal vein reconstruction must be individually tailored using anatomically appropriate techniques to achieve successful outcomes.

way bypasses the liver, leading to the absence of portal hypertension and, consequently, limited development of collateral circulation [3–5]. In CAPV, the liver receives inadequate portal venous inflow, relying primarily on hepatic arterial perfusion. When medical therapy fails, LT becomes necessary to ensure long-term survival. However, because liver function in patients with CAPV is often relatively preserved, the pediatric end-stage liver disease score is typically low, restricting access to deceased donor liver transplantation (DDLT) under the current allocation system in Korea. As a result, living donor liver transplantation (LDLT) is the preferred option for these patients in Korea.

Given the marked anatomical variability of splanchnic venous structures in patients with CAPV, portal vein (PV) reconstruction must be tailored to each individual's vascular anatomy. In this study, we reviewed surgical strategies for PV reconstruction in six pediatric LT cases with CAPV, reported between 2021 and 2023 at a high-volume transplantation center.

PRESENTATION OF FIVE LIVING DONOR LIVER TRANSPLANTATION CASES

Case 1

A 3-year 5-month-old boy diagnosed with CAPV, hepatopulmonary syndrome, and pulmonary arteriovenous malformation underwent LDLT with a left lobe graft from his mother. Preoperative imaging demonstrated that an intrahepatic PCS was the primary vascular anomaly (Fig. 1A and B). The inferior right hepatic vein was markedly dilated and functioned as the conduit of the shunt, whereas the native PV trunk remained patent (Fig. 1C and D). No significant collateral circulation was observed. After hepatic parenchymal transection, the PV was isolated, and both right and left PV branches were temporarily clamped to preserve shunt flow. Following division of the inferior right hepatic vein shunt, the two PV branches were opened and shaped into a single-branch patch (Fig. 1E). This patch was anastomosed to the graft PV using 6-0 polydioxanone sutures (Fig. 1F and G). The postoperative course was uneventful, with stable portal hemodynamics and persistent patency of the reconstructed PV (Fig. 1H). The patient remained clinically well throughout the 3-year follow-up period [6].

Case 2

A 12-year-old boy with CAPV and hepatopulmonary syndrome underwent LDLT using a modified right lobe graft donated by his father. Preoperative imaging showed complete absence of the native PV with cavernous transformation, resulting in well-developed PV collaterals (Fig. 2A). Among these, a prominent collateral vessel of adequate length and caliber was selected for direct anastomosis (Fig. 2B and C). To reduce the risk of intraoperative injury, the collateral was gently clamped with a vascular clamp cushioned by a Nelaton catheter. The proximal segment of the collateral vein was transected and prepared as a branch patch (Fig. 2D). The vein edges were trimmed and suspended with 6-0 polypropylene to form a patch suitable for anastomosis. Standard PV reconstruction was performed with 6-0 polypropylene, reinforced with a vein patch on the posterior wall to prevent suture-related tearing (Fig. 2E). The anterior wall was anastomosed in a slightly redundant manner to accommodate future somatic growth (Fig. 2F and G). A coronary collateral vein and a SRS were ligated to optimize portal flow. The postoperative course was uneventful. Imaging confirmed stable hepatic and PV flows without hemodynamic compromise (Fig. 2H). The patient has remained well for 3 years after LDLT [7].

Case 3

A 10-year-old boy with CAPV and multiple hepatic masses underwent LDLT using a left lobe graft donated by his father. Preoperative imaging confirmed complete absence of the native PV, with a prominent PCS vein coursing along the caudate lobe (Fig. 3A and B). The PCS vein was too short for direct anastomosis (Fig. 3C). To address this, an iliac vein homograft was anastomosed to the graft PV stump on the back table (Fig. 3D). Because of

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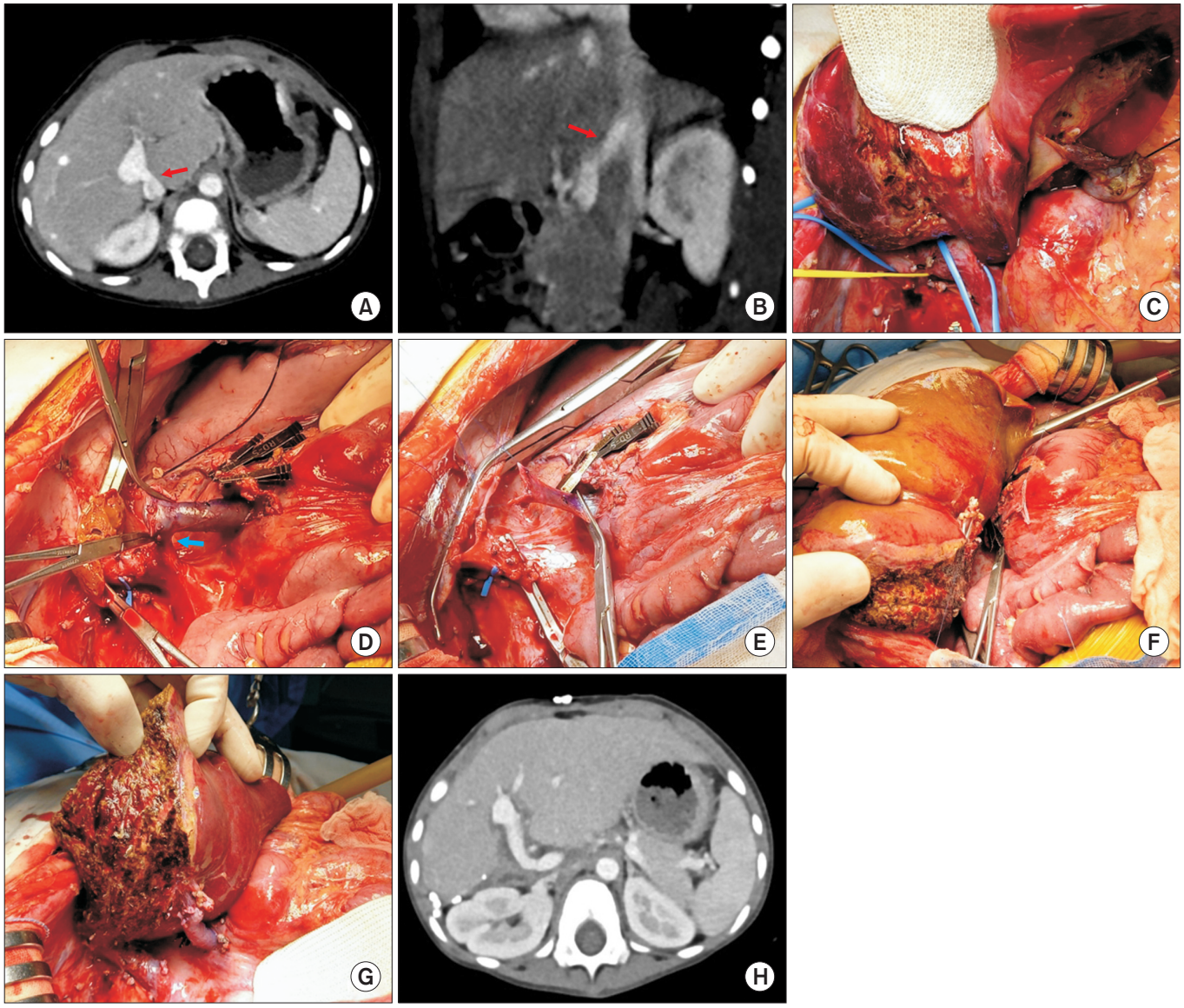

Fig. 1. Image summary of living donor liver transplantation case 1. (A, B) Pretransplant computed tomography (CT) demonstrated normal extrahepatic portal vein anatomy, but a large portocaval shunt through the inferior right hepatic vein (arrows) was identified. (C) The enlarged inferior right hepatic vein was identified and encircled. (D) After removal of the native liver, the communicating inferior right hepatic vein was clamped and ligated. (E) The right and left portal vein stumps were opened to create a branch patch. (F) The recipient portal vein branch patch was anastomosed to the graft portal vein using 6-0 polydioxanone sutures. (G) After graft reperfusion, reperfusion status appeared normal. (H) Posttransplant CT obtained 2 weeks later showed that the graft portal vein was well perfused without evidence of stenosis. Reproduced from Namgoong et al. Ann Liver Transplant 2022;2:144–50. [6]

misalignment between the PCS vein and the graft PV in the standard anatomical position, the graft was rotated into the right subphrenic fossa, enabling dextrorotation and proper alignment of both inflow and outflow vessels. The distal end of the interposed iliac vein conduit was obliquely trimmed to match the orientation and length of the PCS vein. A 1 cm-long wedge-shaped segment of the iliac vein was interposed between the PCS vein and graft PV to complete the anastomosis (Fig. 3E and F). Histopathology of the explanted liver revealed two hepatocellular carcinoma masses arising from a background of multiple unclassified hepatic adenomas (Fig. 3G). The postoperative course was uneventful, with maintained patency of the reconstructed hepatic and PVs and stable hemody-

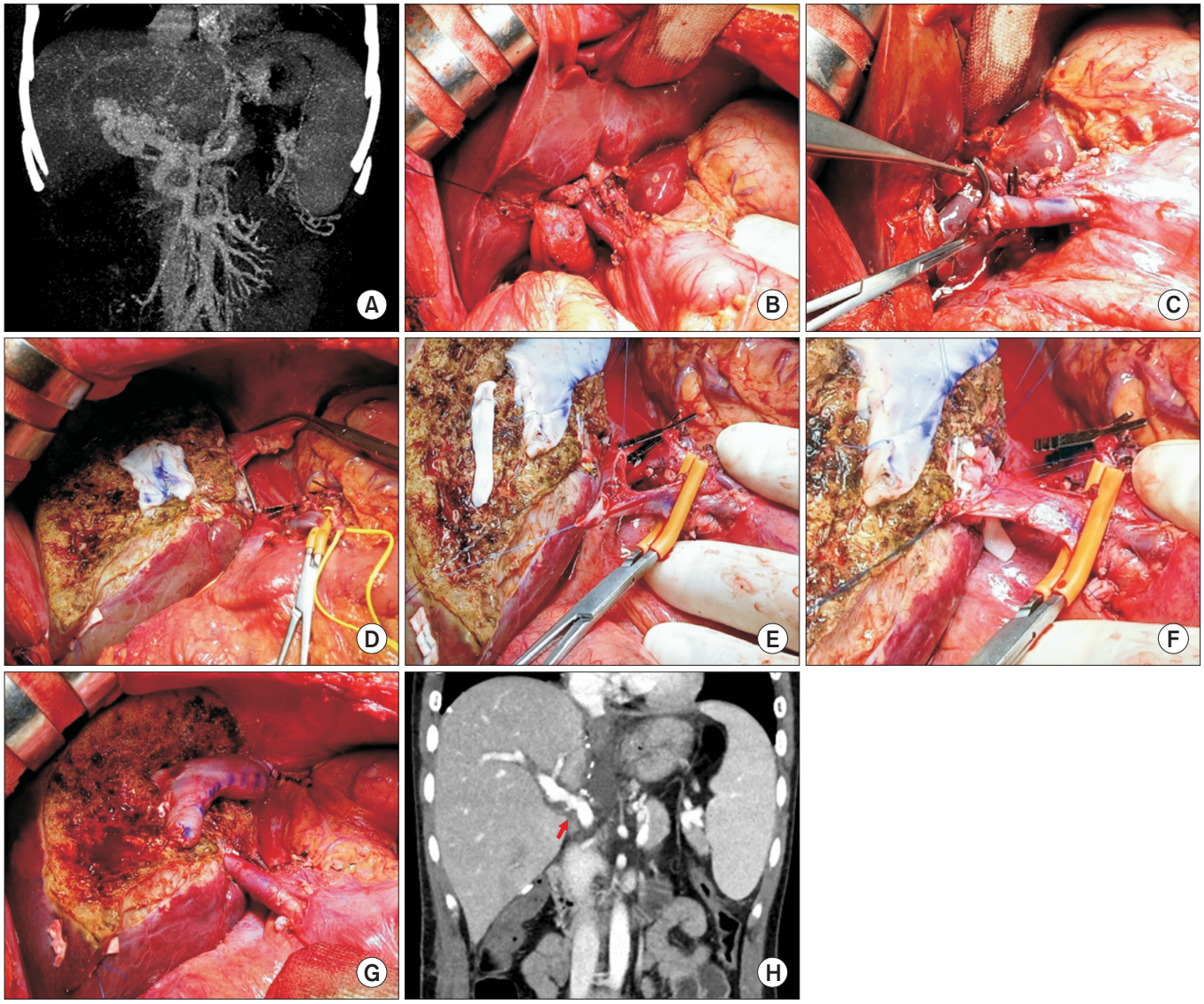

Fig. 2. Image summary of living donor liver transplantation case 2. (A) Pretransplant computed tomography (CT) showed absence of the native portal vein, with development of cavernous, transformed collateral veins. (B) During recipient hilar dissection, a large portal cavernous collateral vein was isolated. (C) This collateral vein was transected with distal cutting of its branches. (D) The transected vein was gently clamped to prevent iatrogenic injury. (E) A branch patch was created at the end of the portal cavernous collateral vein. (F) The posterior wall of the portal vein was anastomosed with a running suture, incorporating a vein patch to prevent stitch-induced wall tearing. (G) The anterior wall was anastomosed redundantly to accommodate future growth. (H) Posttransplant CT obtained 4 days later demonstrated uneventful anastomosis of the graft portal vein. Reproduced from Namgoong et al. Ann Liver Transplant 2023;3:128–35. [7]

namics (Fig. 3H). The patient remains well 3 years after transplantation [8].

Case 4

A 9-year-old girl who presented with hyperammonemia and multiple hepatic masses was diagnosed with CAPV (Fig. 4A) and underwent LDLT with a left liver graft from her mother. Intraoperatively, the native PV was completely absent. After Kocher's maneuver, the left renal vein was exposed and the SRS was carefully isolated. The confluence of the superior mesenteric and splenic veins was meticulously dissected, with all small branches securely controlled (Fig. 4B). A 1.5 cm-sized longitudinal incision was made at the venous confluence, and a cold-pre-

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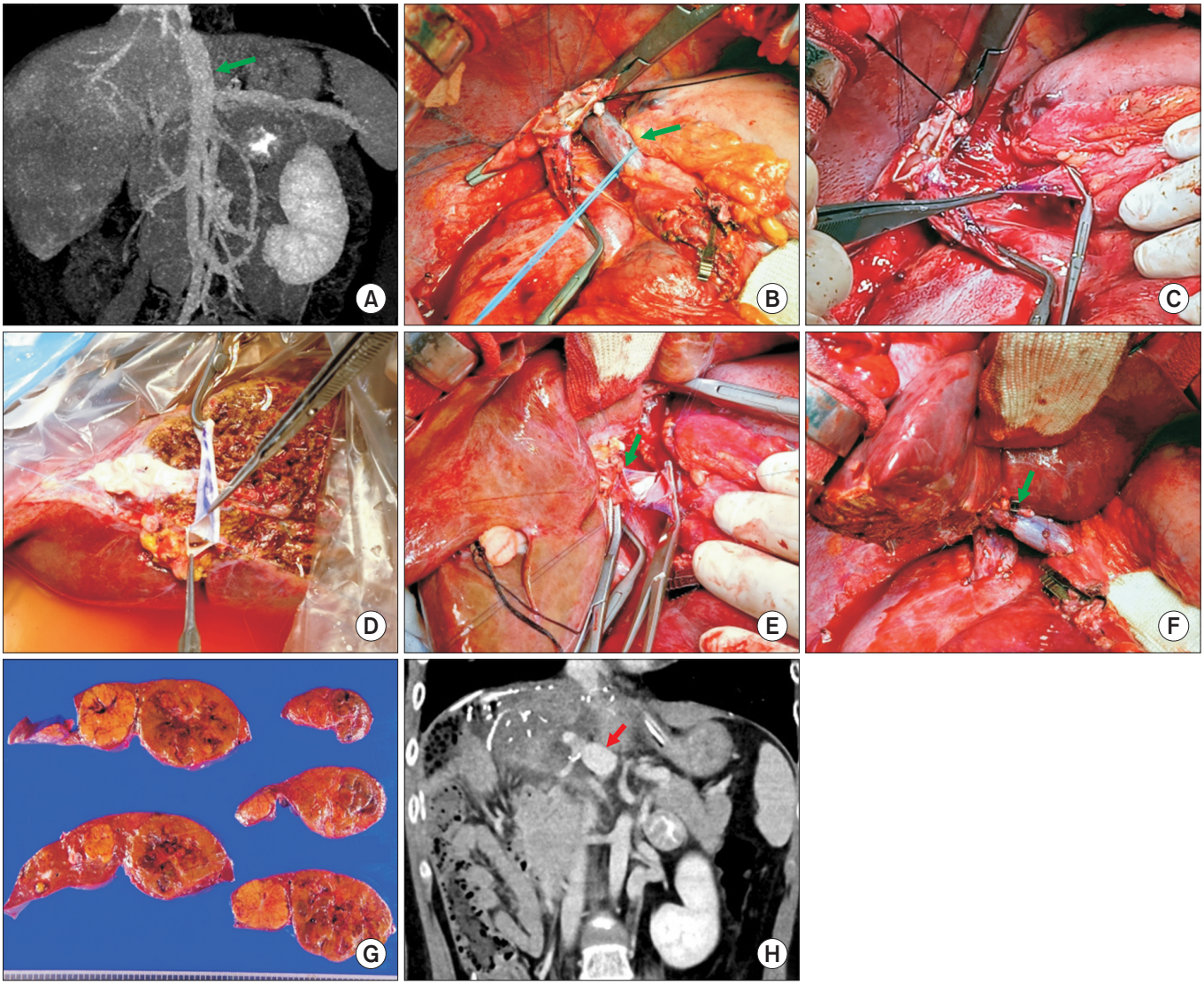

Fig. 3. Image summary of living donor liver transplantation case 3. (A) Pretransplant computed tomography (CT) demonstrated congenital absence of the portal vein with development of a large portocaval shunt to the suprahepatic inferior vena cava (arrow). (B) A large portocaval shunt vein was isolated along the caudate process area (arrow). (C) The isolated shunt vein was transected, but was too short for direct anastomosis to the graft portal vein. (D) An iliac vein segment was anastomosed to the graft portal vein. (E) The interposed iliac vein conduit was obliquely trimmed to match the length and axis of the excised shunt vein. (F) A 1-cm wedge-shaped iliac vein segment (arrow) was interposed between the graft portal vein and shunt vein stumps to avoid redundancy. (G) Gross photograph of the explanted liver showed multiple nodules. (H) Posttransplant CT obtained 14 days later showed a streamlined configuration of the reconstructed portal vein. Reproduced from Namgoong et al. Ann Liver Transplant 2023;3:35–43. [8]

served iliac vein homograft was anastomosed in an end-to-side fashion (Fig. 4C and D). The distal end of the iliac vein conduit was connected to the graft PV (Fig. 4E and F). To accommodate the size mismatch between the conduit and graft PV, small triangular notches were made in both the anterior and posterior walls of the graft PV, ensuring a wide, tension-free anastomosis. The SRS was ligated to improve portal perfusion. Intraoperative direct portography confirmed satisfactory portal flow through the reconstructed conduit (Fig. 4G). The patient recovered uneventfully, and postoperative imaging confirmed durable patency of the reconstructed PV with stable hemodynamics (Fig. 4H). She has remained in good health for 5 years after LDLT [9].

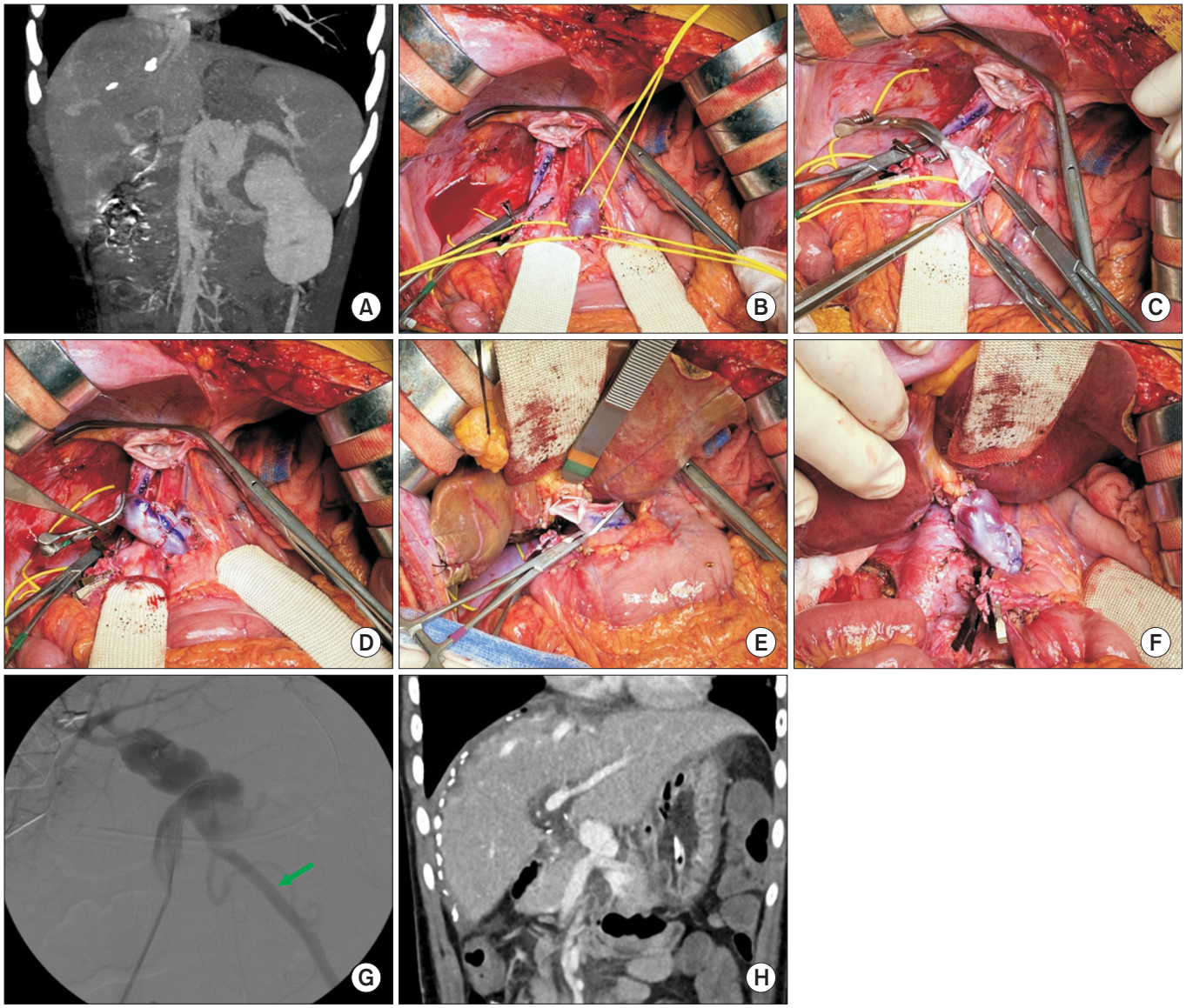

Fig. 4. Image summary of living donor liver transplantation case 4. (A) Pretransplant computed tomography (CT) showed complete absence of the portal vein. (B) The confluence of the mesenteric and splenic veins was meticulously dissected, with branches isolated. (C) A 1.5-cm longitudinal incision was made at the confluence. (D) An iliac vein conduit was anastomosed to the confluence in an end-to-side fashion. (E) The iliac vein conduit was then anastomosed to the graft portal vein. (F) Portal flow increased after ligation of the splenorenal shunt. (G) Intraoperative direct portography showed good portal flow through the conduit and identified a collateral vein to be ligated (arrow). (H) Posttransplant CT obtained 4 days later showed the iliac vein conduit from the mesenteric-splenic vein junction functioning as the main portal trunk. Reproduced from Namgoong et al. Ann Hepatobiliary Pancreat Surg 2021;25:401–7. [9]

Case 5

A 6-year-old boy with CAPV complicated by cavernous transformation of the PV, multiple collateral veins, and splenomegaly (Fig. 5A) underwent LDLT using a left lobe graft from his mother. The operation began with an intraoperative cine-portogram (IOCP), which demonstrated weak hepatopetal flow through variceal collaterals, while most mesenteric venous return drained into a prominently developed SRS (Fig. 5B). Because portal flow improved after proximal clamping of the splenic vein, a collateral vein adjacent to the common bile duct was selected as the source of portal inflow. A cold-stored iliac vein homograft

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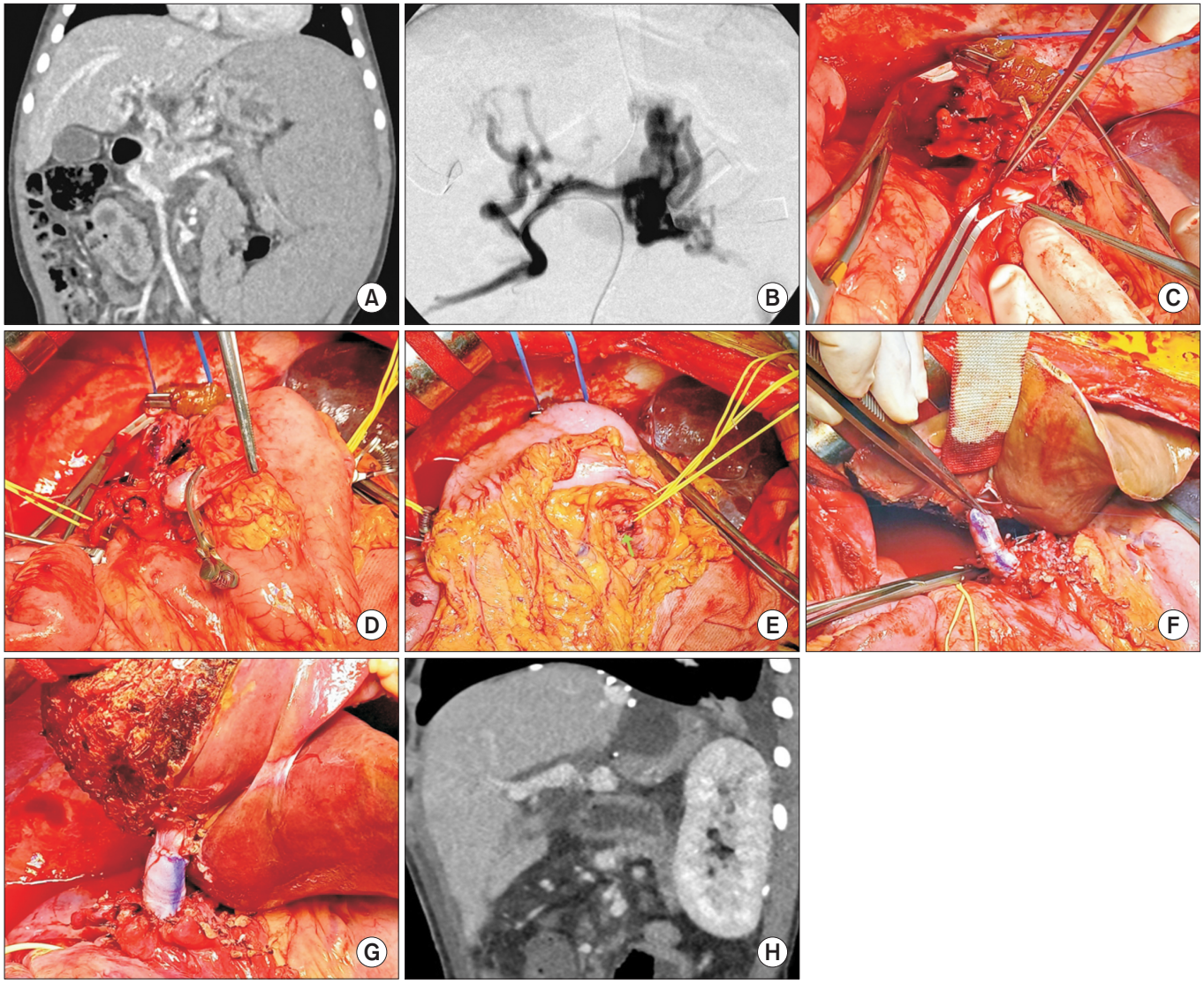

Fig. 5. Image summary of living donor liver transplantation case 5. (A) Pretransplant computed tomography (CT) showed congenital absence of the portal vein with portal hypertension and splenomegaly. (B) The first intraoperative cine-portogram demonstrated that most splanchnic blood drained into the splenorenal shunt. (C) The largest collateral vein adjacent to the common bile duct was side-clamped and opened. (D) An iliac vein homograft was anastomosed to this collateral vein in an end-to-side fashion. (E) The splenic vein (arrow), just distal to the inferior mesenteric vein insertion site, was ligated. (F) The portal vein conduit was connected to the graft portal vein after distance matching. (G) The interposed portal vein conduit was streamlined without redundancy. (H) Posttransplant CT obtained 5 days later showed a well-visualized portal vein conduit. Reproduced from Namgoong et al. Ann Liver Transplant 2022;2:69–77. [10]

was anastomosed to this collateral vein in an end-to-side configuration (Fig. 5C and D). To ensure sufficient portal perfusion, the proximal splenic vein was securely ligated (Fig. 5E). The distal end of the iliac vein conduit was then connected to the graft PV (Fig. 5F and G). The patient recovered uneventfully after transplantation. Postoperative imaging confirmed stable hemodynamics and long-term patency of the reconstructed PV (Fig. 5H). At 13 months after LDLT, partial splenic artery embolization was performed to treat persistent hypersplenism. The patient has remained clinically well for 4 years after LDLT [10].

Table 1. Detailed clinicopathological profiles of six liver transplant recipients for congenital absence of the portal vein.

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Type of LT	LDLT	LDLT	LDLT	LDLT	LDLT	DDLT
Recipient age at LT	3 yr 5 mo	12 yr	10 yr	9 yr	6 yr	4 yr 4 mo
Recipient sex	Male	Male	Male	Female	Male	Male
Recipient body weight (kg)	14	44	45	23	25	15
Pretransplant laboratory findings						
AST/ALT (mU/mL)	40/19	30/15	57/99	102/92	36/23	424/236
Total bilirubin (mg/dL)	0.8	0.8	0.9	0.7	0.4	1.2
Prothrombin time (%/INR)	63%/1.27	70.2%/1.20	100%/1.00	100.1%/1.00	53.6%/1.43	65.0%/1.26
Type of PCS	Intrahepatic shunt to retrohepatic IVC	Cavernous transformation of intra- and extrahepatic PV, SRS	Extrahepatic PCS from mesenteric vein to suprahepatic IVC	Extrahepatic PCS with SRS	Extrahepatic PCS with SRS	Extrahepatic PCS with coronary collaterals
Portal hypertension	Absent	Present	Absent	Absent	Present	Present
Clinical manifestation	Hepatopulmonary syndrome	Esophagogastric varix, hepatopulmonary syndrome	Precocious puberty, liver tumors	Liver nodules, hyperammonemia	Esophageal varix, abdominal distension, splenomegaly	Esophageal and gastric varix bleeding, DiGeorge syndrome
Donor relation	Mother	Father	Father	Mother	Mother	Deceased donor
Donor age (yr)	31	41	45	39	31	5
Graft type	Left liver	Modified right liver	Left liver	Left liver	Left liver	Whole liver
Graft weight (g)	335	640	420	400	420	580
GRWR	2.4	1.45	0.96	1.7	1.7	3.9
Use of vascular graft	No	No	Iliac vein homograft patch	Iliac vein homograft conduit	Iliac vein homograft conduit	Iliac vein homograft conduit
Portal flow source	Native extrahepatic PV trunk	Enlarged PV collateral vein	PCS vein	PCS vein	Pericholedochal collateral vein	Superior mesentric vein-splenic vein confluence
Shunt ligation	None	Coronary collateral, SRS	No	SRS	Proximal splenic vein, coronary collateral veins, splenic artery ligation	Coronary collateral veins
Intraoperative cine-portography	No	No	No	Yes	Yes	No
Liver pathology	Abernethy malformation	Hepatoportal sclerosis	HCC, adenoma	Adenoma, FNH	Hepatoportal sclerosis	Hepatoportal sclerosis
LT operation time (hr)	7	8	7	7	10	7
Estimated intraoperative blood loss (mL)	250	400	350	300	600	400
Complication	No	No	No	No	No	No
Posttransplant intervention	No	No	No	No	Splenic artery embolization after 1 year	No
Follow-up duration (yr)	3	3	3	5	4	11
Survival status	Alive	Alive	Alive	Alive	Alive	Alive

LT, liver transplantation; LDLT, living donor liver transplantation; DDLT, deceased donor liver transplantation; AST, aspartate aminotransferase; ALT, alanine aminotransferase; INR, International Normalized Ratio; PCS, portocaval shunt; IVC, inferior vena cava; PV, portal vein; SRS, splenorenal shunt; GRWR, graft-to-recipient weight ratio; HCC, hepatocellular carcinoma; FNH, focal nodular hyperplasia.

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Summary of Five Living Donor Liver Transplantation Cases

The mean patient age at LDLT was 8.1±3.4 years, with four males and one female. PCS was extrahepatic in four cases and intrahepatic in one. Portal hypertension was present in two patients. Living donors included three mothers and two fathers. Graft types consisted of four left lobe grafts and one modified right lobe graft. Iliac vein homografts were used in three cases, serving as an interposition conduit in two and as a vein patch in one. Portal inflow sources varied: the PCS vein in two cases, a dilated portocaval collateral vein in one, a pericholedochal collateral vein in another, and the native extrahepatic PV trunk in one case. The SRS and large collateral veins were ligated in three patients to enhance portal flow.

No vascular complications occurred following LDLT. One patient required splenic artery embolization for persistent splenomegaly. All five patients survived, with a mean follow-up of 3.6±0.9 years. Detailed clinicopathological findings are summarized in Table 1.

PRESENTATION OF ONE DECEASED DONOR LIVER TRANSPLANTATION CASE

A 4-year-old boy with CAPV and portal hypertension (Fig. 6A) also had DiGeorge syndrome with associated congenital heart anomalies. After a 4-month wait on the DDLT list, he was allocated a liver from a 5-year-old donor weighing 19 kg who was positive for hepatitis B surface antigen (HBsAg). Despite the donor liver's marginal status, liver function tests were acceptable and no significant pathological abnormalities were identified. Considering the very low probability of receiving another DDLT allocation, the decision was made to proceed with transplantation using this graft.

The whole liver graft weighed 580 g, yielding a

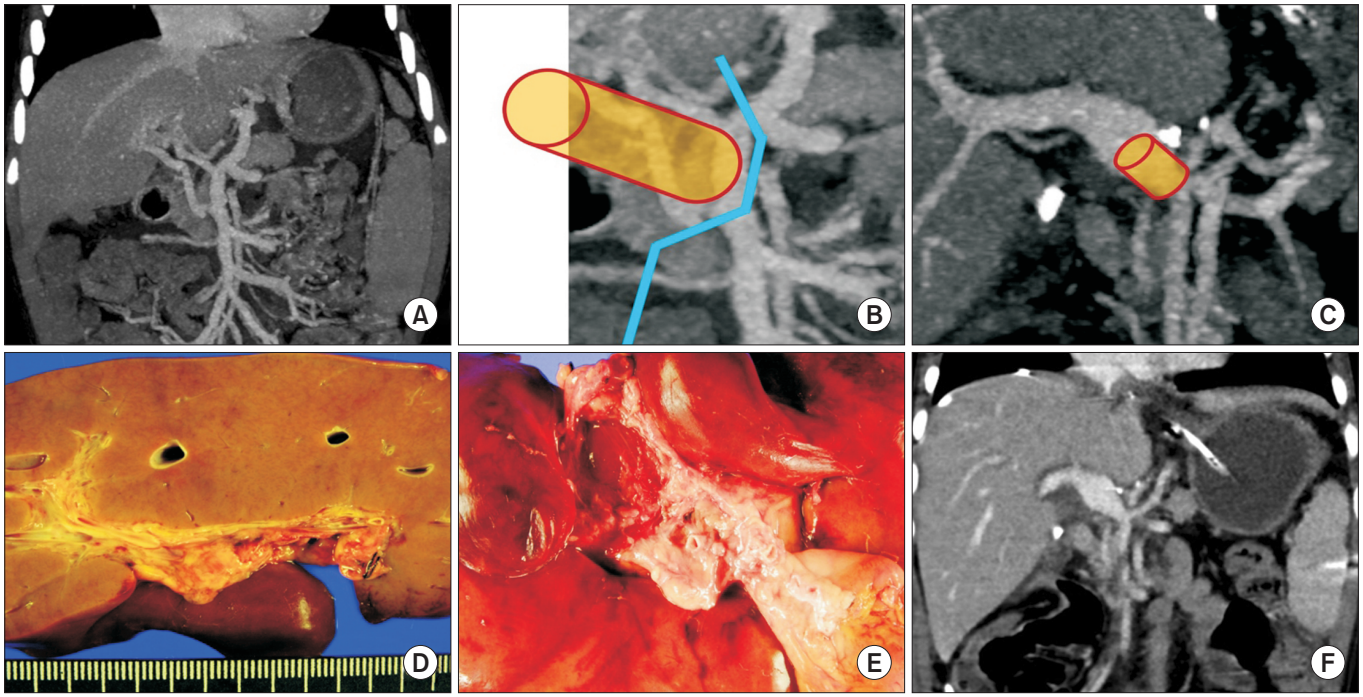

Fig. 6. Image summary of the deceased donor liver transplantation case. (A) Pretransplant computed tomography (CT) showed agenesis of the portal vein with cavernous transformation and secondary portal hypertension, accompanied by gastric and esophageal varices. (B) An external iliac vein graft (cylinder) was anastomosed to the superior mesenteric-splenic vein confluence after deep clamping of this site (blue line). (C) The interposed vascular conduit (cylinder) was positioned between the superior mesenteric-splenic vein confluence and the graft portal vein. (D) Gross photograph of the explanted liver showed non-cirrhotic parenchyma. (E) Magnified view of the portal triad area demonstrated increased vascularity, variable portal venous structures, and intimal fibrosis. (F) Posttransplant CT obtained 7 days later showed smooth, streamlined portal vein reconstruction with resolution of variceal collaterals. Reproduced from Namgoong et al. Korean J Transplant 2021;35:59–65. [11]

graft-to-recipient weight ratio of 3.9%. The recipient procedure followed standard pediatric DDLT techniques. Intraoperatively, the native PV was completely absent. Extensive dissection of the hepatoduodenal ligament was performed to expose the confluence of the superior mesenteric and splenic veins. After deep clamping of this confluence, an external iliac vein homograft harvested from the donor was anastomosed in an end-to-side fashion (Fig. 6B and C). Caval reconstruction was carried out using a modified piggyback technique, and the graft PV was connected with an interposed iliac vein conduit. Coronary venous collaterals supplying gastric and esophageal varices were ligated. Histopathological analysis of the explanted liver confirmed CAPV, with findings of increased vascularity, irregular portal venous structures, and intimal fibrosis (Fig. 6D and E).

The postoperative course was uneventful, with stable portal hemodynamics and durable patency of the reconstructed PV (Fig. 6F). For hepatitis B prophylaxis, the patient received combination therapy with hepatitis B immunoglobulin and antiviral medication for the first 6 months after transplantation, followed by antiviral monotherapy due to sustained high anti-HBs titers. The patient has remained in good health for 11 years following DDLT [11].

DISCUSSION

CAPV is a rare vascular anomaly in which mesenteric venous blood bypasses the liver and drains directly into systemic circulation. Congenital PCS is classified into two major types: intrahepatic and extrahepatic. Intrahepatic PCS involves localized connections between the PV and either the hepatic veins or the retrohepatic IVC [12]. Extrahepatic PCS is further categorized into types I and II, based on the presence or absence of intrahepatic PV perfusion [13]. Type I is defined by complete diversion of mesenteric blood into systemic veins, such as the IVC or left renal vein, due to absence of functional intrahepatic PVs. In contrast, type II PCS retains patent intrahepatic PVs, allowing partial liver perfusion while a portion of mesenteric blood is diverted into systemic circulation. In our case series, one patient had intrahepatic PCS, while the remaining five exhibited extrahepatic PCS of type I.

At present, there is no standardized treatment protocol for CAPV, primarily because of the heterogeneity of its anatomical presentation and associated PCS. PCS is frequently associated with hyperammonemia, and many patients demonstrate mild abnormalities in liver function tests. Most individuals with CAPV are managed conservatively with medical therapy aimed at controlling hyperammonemia. However, surgical interventions, including LT, are considered in select cases—particularly in patients with refractory hyperammonemia or portosystemic encephalopathy. CAPV is also associated with hepatopulmonary syndrome, which is an established indication for LT [14–16].

Although LT for symptomatic CAPV has been reported sporadically, standardized approaches to PV reconstruction remain underdeveloped. Two main techniques are currently employed during LT for CAPV. The first involves direct end-to-end anastomosis between the PCS and the graft PV, as demonstrated in LDLT cases 1 and 2 [2,6,7,13]. The second employs a venous interposition graft anastomosed in an end-to-side fashion to the PCS, as illustrated in LDLT cases 4 and 5 [3,5,9,10].

In most patients with CAPV, pretransplant imaging reveals a prominent communicating vein draining into the IVC via an SRS or PCS. This altered splanchnic circulation typically prevents the development of portal hypertension. However, in our series, three of six patients presented with overt portal hypertension accompanied by gastric and esophageal varices. The clinical manifestations in these patients closely resembled those of decompensated liver cirrhosis. To optimize portal flow to the graft and prevent portal flow diversion, large collateral veins must be effectively managed, either through direct surgical ligation or radiologic intervention.

IOCP is a valuable tool for guiding PV reconstruction and identifying collateral pathways requiring interruption [10,17], as shown in LDLT cases 4 and 5. In our previous study of 31 pediatric LT recipients with biliary atresia, surgical ligation of collateral veins was performed in all cases. IOCP was applied in six patients (19.4%), four of whom demonstrated either absent or minimal residual venous collaterals. Collateral vein embolization and endovascular PV stenting were performed in one patient each. The PV insufficiency-free survival rate was 100% at 1 year and 93.8% at 3 years, underscoring the clinical usability of IOCP in optimizing portal hemodynamics and improving outcomes in pediatric LT recipients [17].

A well-recognized association exists between CAPV and the development of hepatic neoplasms, including focal nodular hyperplasia and hepatocellular adenomas. This association is believed to arise from absent portal

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venous inflow, which results in compensatory arterial hyperperfusion and promotes the formation of regenerative or neoplastic nodules. Although uncommon, these benign lesions may undergo malignant transformation into hepatocellular carcinoma, as presented in LDLT case 3 [8,18–22]. Among 25 reported CAPV cases, 11 developed hepatic tumors [23]. These included focal nodular hyperplasia in four, hepatoblastoma in two, nodular regenerative hyperplasia in two, hepatocellular carcinoma in one, hepatocellular adenoma in one, and nodular hyperplasia in one. Radiologic assessment of these lesions is often challenging because the absence of a functional intrahepatic portal system alters typical enhancement patterns and complicates imaging interpretation.

Transplanting an HBsAg-positive liver graft into an hepatitis B virus (HBV)-naïve pediatric recipient is regarded as a last-resort, life-saving measure. In our experience, LT with HBsAg-positive grafts has been performed in a few critically ill adult patients without prior HBV exposure [24]. In these cases, viral replication was successfully suppressed with oral antiviral therapy. Regardless of the recipient's pretransplant HBV serological profile, rigorous prophylaxis is essential when using HBsAg-positive grafts. This includes the administration of antiviral agents—with or without hepatitis B immunoglobulin—to minimize the risk of HBV transmission and posttransplant reactivation [25,26].

In this study, iliac vein homografts were utilized in four of the six patients. Vascular homografts are indispensable for facilitating complex reconstructions during LDLT. Their availability depends on institutional tissue banks, which serve as the primary source of the vascular materials required for individualized reconstruction. Maintaining an adequate stock of vascular homografts is therefore a critical element of institutional preparedness for LDLT cases requiring customized PV reconstruction [27].

In summary, patients with CAPV frequently present with diverse and complex vascular anomalies. Thorough preoperative and intraoperative evaluations are essential to define these variations accurately. PV reconstruction must be tailored to each patient's anatomy, employing the most anatomically appropriate surgical techniques to achieve optimal outcomes.

ARTICLE INFORMATION

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

ORCID

Jung-Man Namgoong https://orcid.org/0000-0002-9237-7440

Shin Hwang https://orcid.org/0000-0002-9045-2531

Author Contributions

All the work was done by Jung-Man Namgoong and Shin Hwang. All authors read and approved the final manuscript.

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