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ABDOMINAL IMAGING

ORIGINAL ARTICLE

The relationship between preoperative pulmonary hypertension risk in liver transplant and vascular and biliary complications in the postoperative period

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PURPOSE

Liver transplantation (LT) is the gold standard definitive treatment for end-stage liver disease (ESLD). In patients with ESLD, preoperative pulmonary hypertension (PHT) secondary to the primary liver pathology is a common complication that causes high mortality and morbidity. This study aimed to determine whether thrombosis, hemorrhage, stenosis, occlusion, and biliary system complications may be indirectly affected by this vascular complication.

METHODS

This study retrospectively screened patients undergoing LT at our hospital between 2017 and 2024. A total of 265 patients were included in this study, and PHT in the grafts, as well as vascular and biliary pathologies, were identified in all archived computed tomography scans of these patients.

RESULTS

The portal vein complications in the PHT group (17.5%) were higher than those in the non-PHT group (9.4%). In the multivariate logistic regression analysis examining the relationship between PHT level and complications, a significant association with portal vein complications was found. No difference was found between the two groups regarding hepatic vein complications ($P = 0.496$). The incidence of biliary complications in patients with PHT (41.3%) was similar to that in those without PHT (41.1%) ($P = 0.980$). There was no statistically significant relationship between PHT levels (normal, mild, moderate/severe) and the distribution of hepatic artery complications ($P = 0.194$).

CONCLUSION

Moderate-to-severe PHT levels were found to be associated with a significantly increased risk of portal vein complications. We believe caution is warranted, as the presence of preoperative PHT can significantly increase the risk of post-LT complications.

CLINICAL SIGNIFICANCE

Changes in mediator balance in PHT lead to systemic vascular remodeling and, consequently, systemic vasoconstriction. We believe that this condition may negatively affect graft vascularity in patients undergoing LT, thereby increasing vascular and biliary complications. This study found that PHT is associated with the risk of portal vein complications.

KEYWORDS

Preoperative pulmonary hypertension, liver transplantation, complications, biliary system, vascular

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Liver transplantation (LT) is the gold standard definitive treatment for end-stage liver disease (ESLD).[1] In patients with ESLD, pulmonary vascular disorders secondary to the primary liver pathology are common complications that cause high mortality and morbidity. These patients have a 25% prevalence of pulmonary vascular complications. The most common of which is portopulmonary hypertension (PoPHT).[2] Pulmonary hypertension (PHT)

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findings are deemed PoPHT if they develop due to portal venous hypertension. The prevalence of PoPHT ranges from 2.5% to 10.5% in the literature, an extremely high rate.[3,4] The pathogenesis of PoPHT is explained by increased portal venous pressure, leading to hyperdynamic hepatic arterial flow, increased venous blood volume, vasoconstriction in the pulmonary arteries, and neurohumoral activation. Although the condition shares similar histopathological features with other types of PHT, the severity of PoPHT is not related to the severity or etiology of liver disease.[5-7]

The presence of PHT, whether it develops due to PoPHT or any other etiology, significantly worsens the prognosis in patients with liver parenchymal disease who undergo LT.[8] Therefore, identifying PHT in the preoperative period and providing treatment if necessary, as outlined in transplantation guidelines, significantly improves the patient's prognosis.

PHT is a complex systemic disease that affects the entire body and leads to right ventricular failure in its final stage.[9] The disease is caused by a decrease in vasodilator mediators and an increase in vasoconstrictor mediators.[10] In patients with PHT, endothelial dysfunction occurs first, followed by vascular remodeling, and subsequently by apoptosis, neo-adventitial cell proliferation, and thrombosis in arterioles.[11] Regardless of etiology, the imbalance of mediators in patients with PHT leads to vascular remodeling in all systems and, consequently, an increase in mortality and morbidity.[12] Preoperatively, chest X-ray, electrocardiography (ECG), and transthoracic echocardiography (TTE) must be performed. If PHT is suspected, right heart catheterization should be performed to confirm the diagnosis.

TTE is an accurate, reliable, and reproducible tool for estimating systolic pulmonary arterial pressure (sPAP) in the absence of specific pathologies that elevate right heart pressure, such as pulmonary stenosis or right ventricular outflow tract obstruction.[13] Due to its non-invasive and easily accessible nature, the tool has high sensitivity as a screening test.[14] The mean PAP (mPAP) value can be largely predicted using the sPAP value obtained with TTE. The formula used for this is as follows: mPAP: (0.61 * sPAP) + 2 mmHg. The threshold value of 25 mmHg for mPAP used to define PHT is equivalent to 38 mmHg for sPAP.[15] Patients can be grouped as normal (sPAP < 38 mmHg), mild (sPAP: 38–50 mmHg), moderate (sPAP: 50–70 mmHg), or severe (sPAP > 70 mmHg) based on their sPAP values.[16]

Thrombosis generally involves blockage of the lumen of a vessel due to thrombus formation. Stenosis is the narrowing of the vessel's diameter. Occlusion is the complete cessation of blood flow in the vessel.

The primary hypothesis of this study is that thrombosis, hemorrhage, stenosis, occlusion, and biliary system complications may be indirectly affected by these vascular complications. The secondary hypothesis relates to determining the presence of preoperative PHT, superimposed on body mass index (BMI), donor type, cold ischemia time of the graft, presence of early graft dysfunction, need for inotropic support during the perioperative period, total blood loss, and whether there is an increase in the risk of possible vascular and biliary complications when superimposed on the primary etiologies causing liver failure and the presence of preoperative PHT.

Main points

- Preoperative pulmonary hypertension (PHT) increases the risk of portal vein complications in the liver graft, driven by systemic vascular remodeling and vasoconstriction.
- No independent association was found with hepatic artery, hepatic vein, or biliary complications.
- In patients who are candidates for liver transplantation (LT), detecting PHT preoperatively and planning LT following medical treatment reduces postoperative complications.

Methods

This retrospective study was approved by Başkent University Ethics Committee (protocol number: KA25/256, date: 02.07.2025) and was conducted in accordance with the principles outlined in the Declaration of Helsinki. All patients undergoing LT at our hospital between 2017 and 2024 were retrospectively screened. A total of 282 patients were initially included in this study. Patients with missing data in the archives were excluded from the study (n = 10). Additionally, LT procedures performed due to acute liver failure and cancer were excluded from the study as exclusion criteria, with seven patients undergoing LT due to hepatocellular carcinoma excluded. A total of 265 patients were ultimately included (Figure 1), and PHT in the grafts, as well as vascular and biliary pathologies, were identified in all archived computed tomography (CT) scans of these patients. Preoperative TTE findings, BMI, donor type, graft cold ischemia time, presence of early graft dysfunction, need for inotropic support, total perioperative bleeding volume, primary etiologies of liver failure, and demographic data for these patients were determined.

The sPAP values were determined in the TTE findings of the patients included in the study during the preoperative period, and the patients were grouped as normal, mild, moderate, or severe based on these values. All CT scans of the patients were evaluated separately, and all complications in the hepatic artery, portal vein, and hepatic veins (thrombosis, bleeding, stenosis, and occlusion), as well as complications related to the

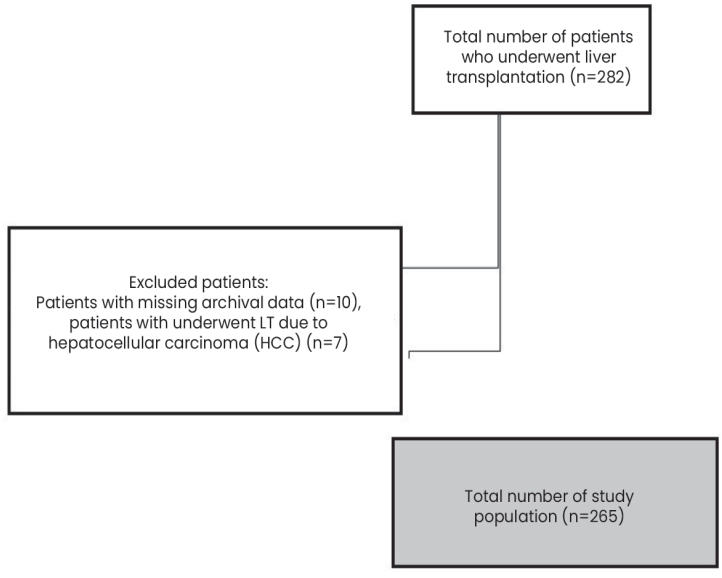

Figure 1. Flow chart of study population. LT, liver transplantation.

biliary system, were identified. All CT scans were evaluated by a single radiologist with 11 years of experience in abdominal and transplantation radiology.

We included all available variables recorded in our hospital's archives. Operative time, transfusion volume, and intraoperative technical difficulties were inconsistently documented throughout the study and were therefore not included in the analysis.

Statistical analysis

All analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as the median (with quartiles 25%–75%; Q1–Q3) for continuous variables and as number (n) and percentage (%) for categorical variables. The Mann–Whitney U test was used to compare two independent groups that did not show a normal distribution. Categorical variables were compared using Pearson's chi-square test when its assumptions were met, and Fisher's exact test or the Fisher–Freeman–Halton exact test via Monte Carlo simulation when expected cell counts were insufficient. A linear-by-linear association test (Cochran–Armitage trend test) was used to evaluate the association between ordinal PHT severity categories and portal vein thrombosis. For risk assessment, separate multivariable logistic regression models were constructed for each complication type (present/absent) as the dependent variable. The primary independent variable was PHT level (reference: normal). All models were adjusted for recipient age, sex (reference: female), BMI, donor type (reference: deceased donor), and cold ischemia time. Model significance (Omnibus test) and goodness-of-fit (Hosmer–Lemeshow test) statistics were reported. Results were presented as adjusted odds ratios (ORs) with 95% confidence intervals (CIs). A P value < 0.05 was considered statistically significant.

Consent to participate and consent to publish

Written consent for participation in the study and for publication of statements was obtained from each patient individually.

Results

A total of 265 individuals were included in the study, with a median age of 8.0 years (range, 1.5–31.0 years), and 66.4% were under 18 years of age. The median BMI was 17.0 kg/m^2 (14.5–20.5), and 55.1% of participants were male patients; 83.8% of transplants were from living donors. The median cold ischemia time was 70 (60.0–85.0) min, and the median perioperative blood loss was 300 (150–660) mL. Delayed graft dysfunction was detected in 7.9% of cases, and inotropic support was required in 14.0%. In recipients, the distribution of etiologies of liver failure ranked biliary pathologies (28.7%), infection (18.5%), and enzyme deficiency (18.1%) in the top three.

Patients with PHT [median: 40.0 (18.0–55.5) years] were older than those without PHT [6.0 (1.0–16.0) years], and the difference between them was statistically significant (P < 0.001). Similarly, 76.2% of those with PHT were over 18 years of age, which was significantly higher than those under 18 years of age with PHT (P < 0.001); BMI [18.0 (15.0–24.5)] was significantly higher in patients with PHT than in those without PHT [16.0 (14.0–20.0)] (P = 0.013). Male gender was significantly more common in the PHT group (66.7%) than in the non-PHT group (51.5%) (P = 0.034). Preoperative PHT was almost twice as common in cadaveric donors (27.0% vs. 12.9%; P = 0.008). The amount of bleeding in patients with perioperative PHT [600.0 (175.0–1,000.0) cc] was significantly higher than in those without perioperative PHT [250.0 (100.0–650.0) cc] (P = 0.001). Cold ischemia time was similar between patients with and without PHT, with no statistically significant difference [65.0 (60.0–82.5) vs. 70.0 (60.0–90.0) min; P = 0.297]. The presence of delayed graft dysfunction (6.3% vs. 8.4%; P = 0.791) and the need for inotropic (norepinephrine) infusion (11.1% vs. 14.9%; P = 0.455) were similar between the PHT and non-PHT groups. No significant difference was found in the distribution of the etiology of liver failure in recipients (P = 0.087). In those with PHT, infection (28.6%) and storage diseases (17.5%) were relatively more common, whereas Budd–Chiari syndrome (0.0%) was not observed. In those without PHT, biliary pathologies (30.2%) and Budd–Chiari syndrome (5.0%) were found to be higher (Table 1).

The distribution of hepatic artery complication types in patients with PHT was similar to that in those without PHT (P = 0.762). Although the rate of thrombosis among portal vein complications in the PHT group (17.5%) was higher than that in the non-PHT group (9.4%), the difference between the groups was not statistically significant (P = 0.211). No difference was found between the two groups regarding hepatic vein complications (P = 0.494). The incidence of biliary complications in patients with PHT (41.3%) was similar to that in those without PHT (41.1%) (P = 0.980). In summary, no significant association was found between preoperative PHT and vascular or biliary complications in the early postoperative period (Table 2).

The relationship between preoperative PHT and postoperative vascular and biliary complications in patients undergoing LT is shown in Table 3. In those without hepatic artery complications, PHT levels were 74.8% in those with normal levels, 66.7% in those with mild levels, and 80.0% in those with moderate/severe levels. In those with hepatic artery thrombosis, the PHT levels were 19.8%, 18.2%, and 20.0%, respectively. In those with hepatic artery bleeding, the levels were 1.0%, 6.1%, and 0.0%, respectively. In those with hepatic artery stenosis, the levels were 4.5%, 9.1%, and 0.0%, respectively. There was no statistically significant relationship between PHT level (normal, mild, moderate/severe) and the distribution of hepatic artery complications (P = 0.192).

In the logistic regression analysis (Table 4 and Figure 2), the model for portal vein complications was statistically significant (model: P = 0.046; Hosmer–Lemeshow: P = 0.793). While no increase in risk was observed for portal vein complications in the mild PHT group (OR: 1.04; 95% CI: 0.39–2.76; P = 0.944), a significant increase in risk was found in the moderate/severe PHT group (OR: 3.67; 95% CI: 1.30–10.36; P = 0.014). No significant association was found between hepatic artery complications and mild PHT (OR: 1.76; 95% CI: 0.76–4.07; P = 0.189) or moderate/severe PHT (OR: 1.25; 95% CI: 0.41–3.80; P = 0.700). Similarly, although the overall model for hepatic vein complications was significant (model: P = 0.014; Hosmer–Lemeshow: P = 0.769), PHT level was not an independent predictor. Hepatic vein complications were not found to be significant in the mild (OR: 0.38, 95% CI: 0.07–1.93, P = 0.241) and moderate/severe (OR: 0.45, 95% CI: 0.08–2.67, P = 0.380) PHT groups. Regarding biliary complications, no significant association was observed in either the mild (OR: 1.02; 95% CI: 0.47–2.20; P = 0.965) or moderate/severe (OR: 0.88; 95% CI: 0.36–2.18; P = 0.783) PHT groups.

Postoperative complications such as bleeding and thrombosis are observed as early complications, detected in almost all patients within the first 30 days. On the other hand, stenosis is a slightly longer-term sub-

Table 1. Demographic and perioperative characteristics according to the presence of PHT in patients [no PHT and PHT present (mild, moderate, and severe)]

	All patients (n = 265)	No PHT (n = 202)	PHT present (n = 63)	*P*
Age (years) Median (Q1–Q3)	8.0 (1.5–31.0)	6.0 (1.0–16.0)	40.0 (18.0–55.5)	**<0.001[M]**
Age; n (%)				
<18 years (years)	176 (66.4%)	161 (79.7%)	15 (23.8%)	**<0.001[K]**
≥18 years (years)	89 (33.6%)	41 (20.3%)	48 (76.2%)	
BMI (kg/m^2) Median (Q1–Q3)	17.0 (14.5–20.5)	16.0 (14.0–20.0)	18.0 (15.0–24.5)	**0.013[M]**
Gender; n (%)				
Male	146 (55.1%)	104 (51.5%)	42 (66.7%)	**0.034[K]**
Female	119 (44.9%)	98 (48.5%)	21 (33.3%)	
Donor type; n (%)				
Living donor	222 (83.8%)	176 (87.1%)	46 (73.0%)	**0.008[K]**
Cadaver	43 (16.2%)	26 (12.9%)	17 (27.0%)	
Cold ischemia time (min) Median (Q1–Q3)	70.0 (60.0–85.0)	70.0 (60.0–90.0)	65.0 (60.0–82.5)	0.297[M]
Bleeding volume (cc) Median (Q1–Q3)	300.0 (150.0–660.0)	250.0 (100.0–650.0)	600.0 (175.0–1,000.0)	**0.001[M]**
Presence of delayed graft function; n (%)				
Absent	244 (92.1%)	185 (91.6%)	59 (93.7%)	0.791[F]
Present	21 (7.9%)	17 (8.4%)	4 (6.3%)	
Need for inotropic (norepinephrine) infusion; n (%)				
Absent	228 (86.0%)	172 (85.1%)	56 (88.9%)	0.455[K]
Present	37 (14.0%)	30 (14.9%)	7 (11.1%)	
Etiology of liver failure in recipients; n (%)				
Infection	49 (18.5%)	31 (15.3%)	18 (28.6%)	0.087[MC]
Accumulation	35 (13.2%)	24 (11.9%)	11 (17.5%)	
Cryptogenic	30 (11.3%)	24 (11.9%)	6 (9.5%)	
Enzyme deficiency	48 (18.1%)	39 (19.3%)	9 (14.3%)	
Biliary pathologies	76 (28.7%)	61 (30.2%)	15 (23.8%)	
Alcoholic cirrhosis	4 (1.5%)	2 (1.0%)	2 (3.2%)	
Budd–Chiari syndrome	10 (3.8%)	10 (5.0%)	0 (0.0%)	
Other rare causes	13 (4.9%)	11 (5.4%)	2 (3.2%)	

[M], Mann–Whitney U test; [K], Pearson's chi-square test, [F], Fisher's exact test; [MC], Monte Carlo exact test (Fisher–Freeman–Halton); PHT, pulmonary hypertension; BMI, body mass index.

acute complication, detected between 1 and 3 months. Occlusion occurs either acutely, due to complete obstruction of the lumen by a thrombus, or chronically, as the stenosis progresses.

In summary, moderate-to-severe PHT levels were only associated with a significant increase in the risk of portal vein complications; no independent association was found with hepatic artery, hepatic vein, or biliary complications.

Discussion

In patients undergoing LT, one of the most common and significant complications during the preoperative period is PHT. In this study, we used sPAP to stratify PHT risk because patients undergoing LT receive routine preoperative ECG examinations. We believed that risk stratification using sPAP would be more effective because it avoids the invasive nature of cardiac catheterization and does not increase contrast or radiation exposure. Krowka and Edwards[6] showed that 5% of patients with cirrhosis met the hemodynamic criteria for PoPHT. Humbert et al.[14] reported a PoPHT prevalence of 9.4% based on data from a French PHT Association registry. A study by Kia et al.[8] demonstrated that high sPAP, as determined by TTE, was associated with a significant increase in mortality and morbidity following LT. Data from the Multicenter Liver Transplant Database showed a 36% mortality rate in patients with PoPHT who underwent LT.[17] In the present study, patients with PHT were older and had higher BMIs than those without PHT. It is expected that age and obesity, in addition to the primary disease, may cause comorbidity and lead to more complications in patients with liver failure, and our study supports this.

Patients undergoing LT from cadaveric donors have a higher risk of complications and PHT compared with patients undergoing LT from living donors. This is because in the latter, the procedure is performed under more elective conditions, with the patient's clinical and laboratory data closely monitored and intervention performed if necessary, ensuring that the procedure is performed under

Table 2. Distribution of PHT status according to vascular and biliary complications

	All patients (n = 265)	No PHT (n = 202)	PHT present (n = 63)	*P*
Hepatic artery complications; n (%)				
Normal	197 (74.3%)	151 (74.8%)	46 (73.0%)	
Thrombosis	52 (19.6%)	40 (19.8%)	12 (19.0%)	0.762[MC]
Bleeding	4 (1.5%)	2 (1.0%)	2 (3.2%)	
Stricture	12 (4.5%)	9 (4.5%)	3 (4.8%)	
Portal vein complications; n (%)				
Normal	207 (78.1%)	161 (79.7%)	46 (73.0%)	
Thrombosis	30 (11.3%)	19 (9.4%)	11 (17.5%)	0.211[K]
Stricture	28 (10.6%)	22 (10.9%)	6 (9.5%)	
Hepatic vein complications; n (%)				
Normal	239 (90.2%)	180 (89.1%)	59 (93.7%)	
Thrombosis	2 (0.8%)	2 (1.0%)	0 (0.0%)	0.494 [MC]
Stricture	24 (9.1%)	20 (9.9%)	4 (6.3%)	
Biliary complications; n (%)				
None	156 (58.9%)	119 (58.9%)	37 (58.7%)	0.980[K]
Present	109 (41.1%)	83 (41.1%)	26 (41.3%)	

[K], Pearson's chi-square test; [MC], Monte Carlo exact test (Fisher–Freeman–Halton; PHT, pulmonary hypertension.

Table 3. Distribution of vascular and biliary complications according to PHT level

	PHT			*P*[K]
	Normal n (%)	Mild n (%)	Moderate/severe n (%)	
Hepatic artery complications				
Normal	151 (74.8%)	22 (66.7%)	24 (80.0%)	
Thrombosis	40 (19.8%)	6 (18.2%)	6 (20.0%)	0.192[MC]
Bleeding	2 (1.0%)	2 (6.1%)	0 (0.0%)	
Stricture	9 (4.5%)	3 (9.1%)	0 (0.0%)	
Portal vein complications				
Normal	161 (79.7%)	27 (81.8%)	19 (63.3%)	**0.037[MC]**
Thrombosis	19 (9.4%)	2 (6.1%)	9 (30.0%)	
Stricture	22 (10.9%)	4 (12.1%)	2 (6.7%)	
Hepatic vein complications				
Normal	180 (89.1%)	31 (93.9%)	28 (93.3%)	0.810[MC]
Thrombosis	2 (1.0%)	0 (0.0%)	0 (0.0%)	
Stricture	20 (9.9%)	2 (6.1%)	2 (6.7%)	
Biliary complications				
Absent	119 (58.9%)	19 (57.6%)	18 (60.0%)	0.981[K]
Present	83 (41.1%)	14 (42.4%)	12 (40.0%)	

[K], Pearson's chi-square test; [MC], Monte Carlo exact test (Fisher–Freeman–Halton); PHT, pulmonary hypertension.

the best possible conditions. Naturally, the graft's cold ischemia time is also shorter in these patients. However, most of these possibilities are not available in LT surgeries performed with cadaveric donors. Therefore, these patients are more prone to developing complications. In parallel with our study, Morioka et al.[18] compared LT from living donors with that from cadaveric donors and found more postoperative complications in the cadaveric LT group. Furthermore, in addition to donor type, this study emphasized that BMI and patient age are extremely important independent predictors of complications, consistent with our results.

No significant differences were found between patient groups in the distribution of primary etiological diseases causing ESLD. As stated separately in the studies by Bozbas and Bozbas[19] and Li et al.,[20] this finding supports the notion that the severity and etiology of liver disease do not affect the severity of PHT.

Meng et al.[21] found that vascular complications, including thrombosis, bleeding, and occlusion, were detected at similar rates in patients with and without PHT, with no significant difference between the groups. Our study yielded similar results, with no significant difference detected between the types of vascular complications.

In the internal evaluation of patients with PHT, a statistically significant increase in the risk of portal vein thrombosis was detected

Table 4. Multiple multivariate logistic regression for vascular and biliary complications: PHT level and covariates (adjusted OR, 95% CI)

Variable	Portal vein complication		Hepatic artery complication		Hepatic vein complication		Biliary complication	
	P	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)
PHT status								
PHT normal (ref)					**0.413**	—	**0.957**	—
PHT mild	0.944	1.036 (0.389–2.761)	0.189	1.756 (0 758–4.068)	0.241	0.377 (0.074–1.926)	0.965	1.017 (0.471–2.196)
PHT moderate and severe	0.014	3.665 (1.296–0.360)	0.700	1.246 (0.408–3.804)	0.380	0.451 (0.076–2.671)	0.783	0.881 (0.356–2.175)
Recipient age (years)	0.162	0.986 (0.967–1.006)	0.077	0.984 (0.966–1.002)	0.048	1.024 (1.000–1.049)	0.709	1.003 (0.988–1.017)
Gender								
Female (ref)								
Male	0.817	0.931 (0.509–1.704)	0.895	1.039 (0.585–1.847)	0.044	0.395 (0.160–0.975)	0.659	0.892 (0.538–1.480)
BMI (kg/m^2)	0.863	1.003 (0.968–1.039)	0.208	1.020 (0.989–1.053)	0.823	1.005 (0.959–1.054)	0.643	0.993 (0.963–1.023)
Donor type								
Cadaveric (ref)								
Living donor	0.922	0.946 (0.316–2.835)	0.747	0.828 (0.262–2.613)	0.997	0.000 (0.00–0.00)	0.951	1.029 (0.419–2.524)
Cold ischemia (min)	0.991	1.000 (0.997–1.003)	0.204	0.998 (0.995–1.001)	0.969	1.000 (0.995–1.005)	0.963	1.000 (0.998–1.002)
Model *P* (Omnibus test)		0.046		0.135		0.014		0.499
Hosmer–Lemeshow *P* (goodness-of-fit)		0.793		0.442		0.769		0.813

PHT, pulmonary hypertension; BMI, body mass index; OR, odds ratio; CI, confidence interval; Ref, reference; min, minimum.

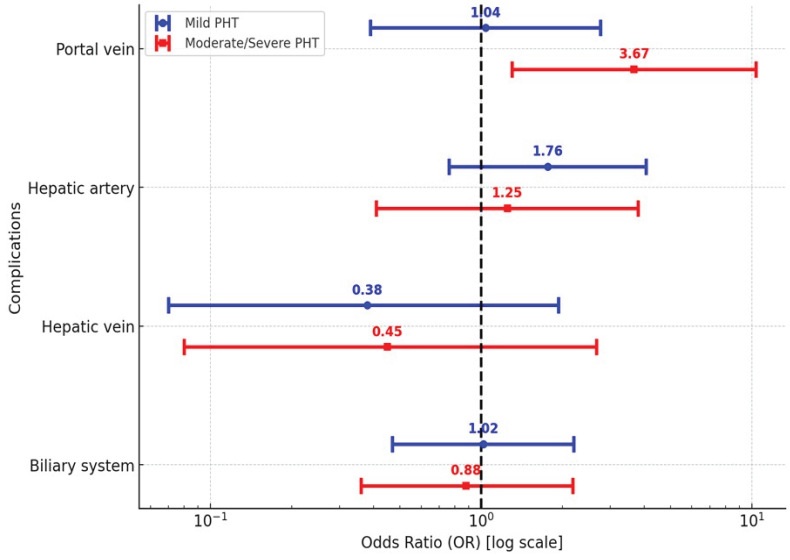

Figure 2. Forest plot showing odds ratios with 95% confidence intervals for vascular and biliary complications according to pulmonary hypertension (PHT) severity.

as the severity of PHT increased. We were unable to find a similar result in our literature review. No significant differences in other types of complications or complications involving other vascular structures were detected according to PHT severity. There are significant gaps in the literature on this subject. The most significant aspect of our study is that our primary and secondary hypotheses address topics that have received little attention in the existing literature. We believe this study will make a valuable contribution to the field; however, randomized controlled trials of these hypotheses involving larger groups are needed.

The most important limitations of this study are inherent to retrospective cohort studies. First, the parameters and information we analyzed are based solely on the completeness of medical records and may contain inaccuracies. Second, due to our small patient group, our evaluation is limited. Therefore, we believe it would be beneficial to re-examine this issue with a larger sample. Another important limitation is that right heart catheterization is not performed prior to LT at our institution in patients with ESLD. In our study, PHT groups were determined based on ECG examination. Furthermore, the lack of standardized operative variables (operational time, transfusion volume, technical difficulties) is a limitation of this retrospective study. While we will prioritize this in future studies, we were unable to assess this in this study. The last limitation is that including adult and pediatric patients in the same cohort may introduce residual confounding, even after adjusting for age. Future studies should be designed prospectively with adequate patient numbers to address this issue.

In conclusion, the presence of preoperative PHT causes a significant increase in risk for most complications. In this study, we found that moderate-to-severe PHT levels were associated only with a significant increase in the risk of portal vein complications. No independent association was found with hepatic artery, hepatic vein, or biliary complications. If preoperative PHT is detected during follow-up, we believe that caution should be exercised regarding portal venous system complications in the postoperative period.

Footnotes

Conflict of interest disclosure

The authors declared that there is no conflict of interest.

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