Hepatopulmonary syndrome increases the postoperative mortality rate following liver transplantation: a prospective study in 90 patients

SCHIFFER, Eduardo, et al.

Abstract

Hepatopulmonary syndrome (HPS) is a frequent pulmonary complication of patients with end-stage liver diseases. HPS is diagnosed by hypoxemia and pulmonary vascular dilatation and is an independent risk factor of mortality. Orthotopic liver transplantation (OLT) is the only factor that modifies the natural course of HPS. Once patients with HPS have been transplanted, their long-term survival rate is similar to transplanted patients without HPS. Consequently, HPS is an indication of OLT whatever the severity of hypoxemia. However, besides the favorable long-term survival of HPS patients with OLT, a high postoperative mortality (mostly within 6 months) has been suggested. The aim of our study was to analyze the incidence of HPS and postoperative outcome after OLT in 90 consecutive patients. All patients were prospectively included and had blood gas analysis to detect HPS. Patients with hypoxemia had contrast echocardiography to confirm HPS. Nine patients had HPS with a 50% increase in mortality compared to non-HPS patients.

Reference


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Hepatopulmonary Syndrome Increases the Postoperative Mortality Rate Following Liver Transplantation: A Prospective Study in 90 Patients


aService d’Anesthésiologie, Département APSI, bUnité de Transplantation, Département de Chirurgie, cLaboratoire de physiopathologie hépatique et imagerie moléculaire and dService de Cardiologie, Hôpitaux Universitaires de Genève, 1206 Geneva, Switzerland
*Département d’Anesthésie et Soins Intensifs, Hôpital Saint-Antoine, Assistance Publique Hôpitaux de Paris, 75012 Paris, France
*Corresponding author: Eduardo Schiffer, eduardo.schiffer@hcuge.ch

Hepatopulmonary syndrome (HPS) is a frequent pulmonary complication of patients with end-stage liver diseases. HPS is diagnosed by hypoxemia and pulmonary vascular dilatation and is an independent risk factor of mortality. Orthotopic liver transplantation (OLT) is the only factor that modifies the natural course of HPS. Once patients with HPS have been transplanted, their long-term survival rate is similar to transplanted patients without HPS. Consequently, HPS is an indication of OLT whatever the severity of hypoxemia.

However, besides the favorable survival of HPS patients with OLT, a high postoperative mortality (mostly within 6 months) has been suggested. The aim of our study was to analyze the incidence of HPS and postoperative outcome after OLT in 90 consecutive patients. All patients were prospectively included and had blood gas analysis to detect HPS. Patients with hypoxemia had contrast echocardiography to confirm HPS. Nine patients had HPS with a 50 ≤ PaO2 ≤ 70 mmHg. Among them 3 (33%) died while the mortality rate was 9.2% in the group without HPS (7 over 76 patients). In the HPS patients who survived, the syndrome completely recovered within 6 months. In conclusion, our study shows a high postoperative mortality rate following OLT even though the preoperative PaO2 was ≥50 mmHg in all HPS patients transplanted.

Key words: Hepatopulmonary syndrome, liver transplantation, perioperative mortality

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without HPS, we analyzed the outcome of 90 consecutive patients with and without HPS transplanted in our institution.

**Methods**

**Patient selection**

Ninety patients [70 males, 20 females, median age 53 years (range: 26–69)] were prospectively included in the study over a period of 4 years and followed for 6 months after OLT. The protocol was approved by the institutional ethics committee of the University of Geneva and written informed consent was obtained from each patient at the time of inclusion in the waiting list for OLT.

**Diagnostic criteria for HPS**

HPS was diagnosed when the following criteria were fulfilled: (1) presence of chronic liver disease (Child–Turcotte–Pugh score ≥6) and/or portal hypertension; (2) alveolar-arterial difference for the partial pressure of oxygen (AaDO₂) ≥ 15 mmHg (normal range, 4–8 mmHg) (20) associated with hypoxemia ≤ 70 mmHg in upright position while breathing room air; (3) intrapulmonary vascular dilatation detected by transthoracic two-dimensional contrast echocardiography and (4) absence of primary cardiac or pulmonary disease, according to history, electrocardiogram and echocardiography including Doppler measurements and chest X-ray.

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**Figure 1: Illustration of a positive contrast-enhanced echocardiography.**

(A) Normal four-chamber view without contrast medium. (B) After one heartbeat, no microbubbles are present in the left cardiac chambers. (C) Four heartbeats after contrast medium injection, microbubbles are detectable in the right atrium and in the right ventricle of the heart. (D) Five heartbeats after injection, microbubbles also appear in the left cardiac chambers. (E) After 10 heartbeats, the left chambers are filled with microbubbles. (F) After 22 heartbeats, microbubbles are progressively disappearing.
Arterial blood gas analysis
Arterial blood gas samples were obtained by percutaneous radial artery puncture, both in the upright (patients being seated in an armchair) and the supine position while breathing room air. Arterial blood gas analysis was performed with an analyzer (Stat Profile Ultra, Nova Biomedical, Waltham, MA, USA) calibrated hourly. Arterial PO2, partial pressure of arterial carbon dioxide (PaCO2), arterial base excess, bicarbonate concentration, pH and AaDO2 were measured or calculated, according to the standard formula as previously described (15,20,21), using daily barometric pressure (PB), water vapor pressure at 37°C (47 mmHg), FiO2 at room air (0.21) and assuming a ventilation/perfusion ratio 0.8:

\[ \text{PAO}_2 = [0.21 \times (P_B - 47)] - (\text{PaCO}_2/0.8) \]

and

\[ \text{AaDO}_2 = \text{PAO}_2 - \text{PaO}_2 \text{ (mmHg)}. \]

Pulmonary hemodynamics
To exclude pulmonary hypertension, pulmonary hemodynamics were measured during a right heart catheterization in steady conditions in all patients via a pulmonary artery catheter (Swan-Ganz catheter, Baxter Healthcare, Irvine, CA, USA). Measurements included mean pulmonary artery pressure (MPAP, mmHg), mean right atrial pressure (RAP, mmHg), cardiac output (L/min, using the thermodilution method), cardiac index (L/min/m²) and pulmonary capillary wedge pressure (PCWP, mmHg). Pulmonary vascular resistances (PVR, dyne s/cm² m⁵) were calculated. Transpulmonary pressure gradient was also measured using free hepatic vein pressure and wedged hepatic vein pressure as previously described (22).

Pulmonary function tests
Vital capacity and forced expiratory volume in 1 second (FEV1) were obtained by a computerized spirometer (Autobox 6200, Sensor Medics, Yorba Linda, CA, USA) using standard procedures. Total lung capacity was measured by a body plethysmograph (Autobox 6200, Sensor Medics, Yorba Linda, CA, USA) and diffusing capacity for carbon monoxide (DLCO) was calculated by either the single-breath technique corrected for serum hemoglobin or the steady-state technique and reported with reference to standard predicted percentages (23).

Enhanced contrast echocardiography
Contrast medium was obtained by agitation of saline solution, which creates a stream of microbubbles after intravenous injection. Transthoracic two-dimensional echocardiography was performed after the iv injection of the contrast medium (5 mL) in a peripheral vein. Echocardiography was performed by an experienced cardiologist using two-dimensional apical four-chamber views (Figure 1). Positive contrast echocardiography for intrapulmonary shunt was defined by the appearance of microbubbles in the left side of the heart chambers within 6 but not before 4 heartbeats after the appearance in the right side of the heart. Thus, microbubbles (diameter: 60–90 μm) opacify the left heart chambers only when they pass through the pulmonary capillaries (24). The distinction between intrapulmonary or intracardiac shunt is made by the time of appearance in the left heart chambers. When intracardiac shunt exists, microbubbles appear within 3 heartbeats in the left heart chambers. In contrast, with intrapulmonary shunts, microbubbles appear within 4–6 heartbeats after the initial appearance in the right side of the heart (4,25).

Data analysis
Comparisons between groups were performed by the Mann-Whitney U test for non-parametric data and the \( \chi^2 \) test when appropriate. Statistical significance was designated as \( p < 0.05 \). The Kaplan-Meier method was used to determine patient survival and the log-rank test to compare survival between groups. Statistical analysis was performed with SPSS (Release 11, SPSS Inc., Chicago, IL, USA).

Results

Prevalence of HPS
We included 90 patients transplanted at a single transplantation center (Hôpitaux Universitaires de Genève, Geneva, Switzerland) between January 1999 and December 2003. Twenty-one additional patients listed during the same period had to be excluded from OLT for various reasons that are explained in Table 1. An overview algorithm that characterizes the patients in terms of HPS assessment and outcome after OLT is provided in Figure 2. Among the 90 patients of the study, blood gas analysis was performed according to the investigation protocol and 18 were suspected to have HPS. Among these 18 patients, 13 had transthoracic contrast echocardiography, which confirmed the syndrome in 9. Thus, 10% of the patients had confirmation of HPS. However, higher percentage of patients with HPS should be expected since among the 5 patients suspected to have HPS based on blood gas analysis and who had no transthoracic contrast echocardiography, 3–4 patients (70% of suspected patients had HPS confirmation) should have HPS. Considering the uncertainty on their clinical status, these 5 patients were finally excluded from the statistical analysis. Thirteen living donor liver transplantations were performed during the study, but none of these patients had HPS. Of note the criteria for HPS was more severe than the one highlighted recently by the European Respiratory Society that considers the threshold value of hypoxemia to be 80 mmHg when we chose a PaO2 ≤ 70 mmHg (16,17). Because all HPS patients had PaO2 ≤ 70 mmHg, we classified the syndrome as very severe (16).

Clinical characteristics of patients
The clinical characteristics of the 9 patients who fulfilled the criteria for HPS diagnosis were compared to those of patients without HPS (Table 2). Mean age, sex and cause

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Figure 2: Overview algorithm of the study patients listed for OLT. OLT = orthotopic liver transplantation; HPS = hepatopulmonary syndrome. White boxes represent HPS patients, gray boxes non-HPS patients.

Table 2: Clinical characteristics of patients (n = 85). Patients were considered HPS+ only when contrast echocardiography was positive (n = 9).

<table>
<thead>
<tr>
<th></th>
<th>With (n = 9)</th>
<th>Without (n = 76)</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) ± SD</td>
<td>56 ± 8</td>
<td>53 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>Male (%)</td>
<td>89</td>
<td>71</td>
<td>NS</td>
</tr>
<tr>
<td>Cause of cirrhosis (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>11.1</td>
<td>32.9</td>
<td>NS</td>
</tr>
<tr>
<td>Hepatitis C virus</td>
<td>88.9</td>
<td>34.1</td>
<td>NS</td>
</tr>
<tr>
<td>Hepatitis B virus</td>
<td>0</td>
<td>13.2</td>
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</tr>
<tr>
<td>Biliary cirrhosis</td>
<td>0</td>
<td>6.8</td>
<td></td>
</tr>
<tr>
<td>Autoimmune hepatitis</td>
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<td>2.6</td>
<td></td>
</tr>
<tr>
<td>Amyloidosis</td>
<td>0</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td>Metabolic inherited disease</td>
<td>0</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>0</td>
<td>5.2</td>
<td></td>
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<tr>
<td>Child–Pugh class (%)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>A</td>
<td>33.3</td>
<td>22.4</td>
<td>NS</td>
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<tr>
<td>B</td>
<td>22.2</td>
<td>40.8</td>
<td>NS</td>
</tr>
<tr>
<td>C</td>
<td>44.4</td>
<td>36.8</td>
<td>NS</td>
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<tr>
<td>Child—Pugh score</td>
<td>8.9 ± 2.8</td>
<td>8.8 ± 2.4</td>
<td>NS</td>
</tr>
<tr>
<td>MELD score</td>
<td>17.2 ± 6.1</td>
<td>15.1 ± 6.4</td>
<td>NS</td>
</tr>
<tr>
<td>Serum creatinine (µmol/L)</td>
<td>124 ± 35</td>
<td>108 ± 43</td>
<td>NS</td>
</tr>
<tr>
<td>Total bilirubin (µmol/L)</td>
<td>50 ± 25</td>
<td>46 ± 28</td>
<td>NS</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>29 ± 6</td>
<td>30 ± 4</td>
<td></td>
</tr>
<tr>
<td>Prothrombin time (INR)</td>
<td>1.5 ± 0.3</td>
<td>1.5 ± 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>FEV1 (% predicted)</td>
<td>89 ± 25</td>
<td>84 ± 15</td>
<td>NS</td>
</tr>
<tr>
<td>FEV1/ventil capacity (%)</td>
<td>73 ± 11</td>
<td>72 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>PaO2 (mmHg)</td>
<td>60 ± 6</td>
<td>86 ± 12</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>AaDO2 (mmHg)</td>
<td>52 ± 10</td>
<td>25 ± 14</td>
<td>&lt;0.0001</td>
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</table>

Table 3: Hepatic and systemic hemodynamic parameters

<table>
<thead>
<tr>
<th></th>
<th>HPS patients (n = 9)</th>
<th>Non-HPS patients (n = 76)</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>HVPG (mmHg)</td>
<td>15.9 ± 3.7</td>
<td>15.8 ± 6.0</td>
<td>0.92</td>
</tr>
<tr>
<td>MPAP (mmHg)</td>
<td>13.8 ± 5.4</td>
<td>15.3 ± 5.1</td>
<td>0.34</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>8.4 ± 4.9</td>
<td>8.6 ± 4.1</td>
<td>0.88</td>
</tr>
<tr>
<td>Cardiac index (L/m²)</td>
<td>3.5 ± 0.9</td>
<td>3.5 ± 1.2</td>
<td>0.84</td>
</tr>
<tr>
<td>PVRI (dyne s/cm⁻⁵ m²)</td>
<td>125 ± 32</td>
<td>162 ± 90</td>
<td>0.22</td>
</tr>
</tbody>
</table>

HVPG = hepatic venous pressure gradient; MPAP = mean pulmonary arterial pressure; PCWP = pulmonary capillary wedge pressure; PVRI = pulmonary vascular resistance index.

of cirrhosis were similar in both groups. More importantly, the severity of liver diseases assessed by Child–Pugh and model of end-stage liver disease (MELD) scores was not related to HPS presence. Accordingly, total bilirubin, albumin and creatinine concentrations in serum and prothrombin time were not different in patients with or without HPS. There was no significant difference in lung function parameters. Hepatic and systemic hemodynamic parameters were also similar in both groups (Table 3). As expected, PaO2 was significantly reduced and AaDO2 was significantly increased in patients with HPS. However, all patients with HPS had a 50 ≤ PaO2 ≤ 70 mmHg (Table 4). The PaO2 ranges for non-HPS patients were 71–104 mmHg and the AaDO2 ranges were 3–31 mmHg. All patients with HPS documented by positive contrast echocardiography had deceased-donor OLT and were followed for 6 months. Median time from HPS diagnosis to OLT was 10.3 months in the 9 patients with HPS. At OLT time, PaO2 had deteriorated in all but 2 patients. Individual clinical details of the 18 patients having blood gas analysis suggesting HPS are also presented in Table 5.

Postoperative mortality rate

The mortality rate was compared between the 76 patients who had no HPS and the 9 patients who had a positive echocardiography. The 5 patients suspected to have HPS without confirmation by contrast transthoracic echocardiography were excluded from the statistics to avoid speculation on their clinical status. Mean survival rates at 6 months were significantly different (p = 0.0012, log-rank test, Figure 3). The mortality rate in patients with HPS was 33% while the mortality rate of patients without HPS was 9.2%. At 6 months, the overall mortality of patients with and
without HPS (n = 90) was 13%. Characteristics of HPS patients who died are shown in Table 6. Child–Pugh score was similar in those who died in comparison to those who survived but MELD score was higher (Table 6). Surprisingly, PaO2 was significantly higher and AaDO2 significantly lower in the HPS patients who died. However, the small number of HPS patients precludes extrapolating these findings to larger groups of patients.

**Evolution of HPS after OLT**

Blood gas analysis was performed at 6 months in the 6 patients with HPS who survived and showed complete resolution of hypoxemia in all patients as revealed by normalization of the AaDO2 gradient (Figure 4). Mean PaO2 after OLT was significantly higher than the pre-OLT value: 92.6 mmHg (72–102 mmHg) versus 57 mmHg (52–63 mmHg). Causes of death in the patients with HPS are listed in Table 7. Three patients died within the first 35 days after OLT of either septic (2) or hemorrhagic shock (1) having never improved blood gas exchange.

**Discussion**

Our prospective study shows that the postoperative mortality rate following OLT in patients with moderate-to-severe HPS (50 ≤ PaO2 ≤ 70 mmHg) is much higher than the postoperative mortality rate of patients without HPS. The reasons for such high mortality remain puzzling.
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Figure 3: Cumulative survival rate of patients screened for HPS who underwent OLT over a 6-month follow-up period. Upper continuous line represents patients without HPS (n = 76) and lower continuous line represents patients with proven HPS (n = 9). Survival was significantly different between patients with or without documented HPS (log-rank test).

Table 6: Demographic and clinical characteristics of patients with HPS

<table>
<thead>
<tr>
<th>Alive (n = 6)</th>
<th>Dead (n = 3)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>59 ± 8</td>
<td>52 ± 1</td>
</tr>
<tr>
<td>Child–Pugh score</td>
<td>7.7 ± 2.6</td>
<td>11.3 ± 1.2</td>
</tr>
<tr>
<td>MELD score</td>
<td>14.0 ± 4.0</td>
<td>23.4 ± 4.5</td>
</tr>
<tr>
<td>PaO2 (kPa)</td>
<td>53 ± 3</td>
<td>62 ± 3</td>
</tr>
<tr>
<td>AaDO2 (kPa)</td>
<td>58 ± 4</td>
<td>41 ± 8</td>
</tr>
<tr>
<td>Liver disease</td>
<td>Alcohol</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Hepatitis</td>
<td>2</td>
</tr>
</tbody>
</table>

However, the syndrome completely recovered within 6 months in patients who survived. Thus, although the favorable long-term survival of HPS patients with OLT may prompt centers to transplant all HPS patients whatever the preoperative hypoxemia, the immediate postoperative mortality rate remains high in this group of patients.

Postoperative mortality

Previous studies have determined that HPS is an independent risk factor of long-term mortality in cirrhosis. In the absence of OLT, the long-term mortality is increased in cirrhotic patients with HPS in comparison to cirrhotic patients without HPS (4,15). Moreover, the 5-year mortality rate in HPS patients who had OLT is 24% versus 77% in HPS patients who do not undergo OLT (15). Following OLT, the long-term mortality is similar in patients with or without HPS (15). This conclusion is true even when preoperative PaO2 was ≤ 50 mmHg (15). Thus, considering a long-term period, OLT is the only factor that modifies the mortality rate in patients with HPS and once patients with HPS have been transplanted, their long-term mortality rate is similar to transplanted patients without HPS (15). Consequently, in the absence of comorbidities, HPS is now an indication of OLT whatever the severity of HPS or hypoxemia (16).

However, besides the good evolution of HPS patients with OLT over 5 years, a high postoperative mortality (mostly within 6 months) has been suggested. In their retrospective study, Swanson et al. (15) showed a 21% (5 over 24 patients) postoperative mortality rate in HPS patients. All 5 patients had a low preoperative PaO2 (≤51 mmHg). Another retrospective analysis also found a 22% postoperative mortality rate in HPS patients (18) while in the prospective study by Arguedas et al. (19), the postoperative mortality rate was 16% (19).
mortality was 29%. Because the immediate postoperative mortality rate of patients with and without HPS has not been previously compared prospectively, we analyzed the outcome of 90 consecutive OLT patients. All patients with HPS were classified as having moderate-to-severe HPS according to recent guidelines, all patients having a PaO2 between 52 and 70 mmHg (17). As suggested by previous studies, we confirmed that the postoperative mortality following OLT was significantly higher in patients with HPS (33%) than in patients without HPS (9.2%). The reasons for the increased postoperative mortality in HPS patients are puzzling. In their study, Taille et al. (18) found that the immediate postoperative mortality was not associated with the severity of HPS. In the 9 HPS patients, Child–Pugh score was similar in those who died in comparison to those who survived but MELD score was higher (Table 6). Surprisingly, PaO2 was significantly higher and AaDO2 significantly lower in the HPS patients who died. However, the small number of HPS patients precludes extrapolating these findings to larger groups of patients. Although the severity of pulmonary shunt was not quantified by echocardiography, the suspicion of HPS was higher when 98 patients of whom 33 (34%) had a positive contrast dilatation (contrast echocardiography vs. scintigraphic perfusion scanning). Under normal conditions, 99mTc albumin macroaggregates that exceed 20 μm in diameter can be visualized in other organs such as the brain or the spleen. Contrast echocardiography was shown to be more efficient than scintigraphic perfusion scanning to diagnose pulmonary vascular dilatation in cirrhotic patients with HPS (14). Guidelines on pulmonary–hepatic vascular disorders recently highlighted echocardiography in the screening algorithm for HPS, scintigraphic scanning being inadequate to differentiate between pulmonary and intracardiac shunts (16).

Prevalence of HPS

The reported prevalence of HPS in patients with liver disease varies from 4% to 19%, probably because various criteria and threshold values defining arterial deoxygenation have been used (2,21,26). Thus, in a study including 98 patients of whom 33 (34%) had a positive contrast echocardiography, the suspicion of HPS was higher when the AaDO2 was >15 mmHg (32%), >20 mmHg (31%), and greater than age-related threshold (28%) than when a PaO2 threshold was chosen <80 mmHg (19%), <70 mmHg (15%), and ‘age-related threshold’ (15%) (26). Moreover, the techniques used to confirm the pulmonary vascular dilatation (contrast echocardiography vs. scintigraphic perfusion scanning) have also to be considered.

The prevalence of HSP among OLT patients was 10% (9 over 90) in our study similarly to the retrospective study of Mohamed et al. (27), who found hypoxemia in 7% of the patients listed for OLT. However, in this study no shunt could be diagnosed either by echocardiography or by scintigraphy. In more recent studies, no prevalence of HSP in OLT candidates was mentioned (15,19,28). Thus, the prevalence of HSP in OLT candidates is difficult to assess because in most studies patients with severe hypoxemia have been excluded from OLT or patients with minimal shunt have not been diagnosed as having HPS. Additionally, considering that 70% (9/13) patients suspected to have HPS have confirmed the diagnosis with contrast echocardiography, the 5 patients suspected to have the syndrome without echocardiography confirmation might increase the prevalence to 14%.

Clinical characteristics of HPS patients

No specific clinical characteristics of HPS patients have been observed in our study. Gender, age and causes of liver disease are similar in patients with or without HPS (Table 2). Hepatic and pulmonary hemodynamic parameters were not different (Table 2). FEV1 and FEV1/CV did not change in patients with and without HPS and although several patients had a decreased DLCO, as observed in another study (29), this finding is not a prerequisite for the diagnosis of HPS.

To document intrapulmonary vascular dilatation, we used contrast echocardiography and found nine positive examinations over the 13 patients who had blood gas abnormalities. An alternative to contrast echocardiography is scintigraphic perfusion scanning. Under normal conditions, 99mTc albumin macroaggregates that exceed 20 μm in diameter are almost completely trapped in the pulmonary circulation. In the presence of cardiac right-to-left shunts or intrapulmonary vascular dilatation, the uptake of 99mTc albumin macroaggregates can be visualized in other organs such as the brain or the spleen. Contrast echocardiography was shown to be more efficient than scintigraphic perfusion scanning to diagnose pulmonary vascular dilatation in cirrhotic patients with HPS (14). Guidelines on pulmonary–hepatic vascular disorders recently highlighted echocardiography in the screening algorithm for HPS, scintigraphic scanning being inadequate to differentiate between pulmonary and intracardiac shunts (16).

Evolution of HPS after OLT

Besides the favorable long-term survival rate, another reason to perform OLT in patients with HPS is the recovery of a normal pulmonary function after OLT (18,27). In our study, correction of hypoxemia occurs as early as 6 months after OLT. The reversibility of HPS is also found within 10 months in other studies (18,30). However, the time of reversibility might be longer when HPS is more severe. An increased recovery time was observed when PaO2 is ≤52 mmHg, AaDO2 is ≥66 mmHg, age is ≥48 years, or if the etiology of liver disease is alcohol intoxication (18). A delayed resolution of HPS might favor complications in the postoperative period (19).

Hypoxemia and outcome in patients with HPS

In the absence of OLT, a PaO2 ≤60 mmHg significantly increases the long-term mortality rate of patients with HPS (4). In contrast, following OLT, the long-term mortality rate of HPS patients is not significantly modified by a PaO2 ≤50 mmHg, OLT interrupting the natural course of the syndrome (15). After the postoperative period, the recovery time of a normal PaO2 might depend on the severity of HPS: the lower the PaO2 in the preoperative period, the longer the delay of recovery. Finally, our study shows a high postoperative mortality rate following OLT in HPS...
patients, even though all patients with HPS had a PaO₂ ≥ 50 mmHg. Identification of risk factors for early mortality is difficult considering the small number of patients with HPS.

In conclusion, although the favorable long-term survival of HPS patients with OLT may prompt centers to transplant all HPS patients whatever the preoperative hypoxemia, the immediate postoperative mortality rate remains high in this group of patients.

References