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# The Incidence and Risk Factors of Low Oxygenation After Orthotropic Liver Transplantation

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**Background:** This study was designed to observe incidence and risk factors of low oxygenation after orthotropic liver transplantation (OLT).

**Material/Methods:** We retrospectively evaluated all adult patients who underwent living-donor OLT between January 1, 2017 and December 31, 2017. Postoperative low oxygenation was defined as $PaO_2/FiO_2$ <300 mmHg within 24 hours after surgery. Early acute kidney injury (AKI) after OLT was also defined when AKI was happened with 24 hours after operative.

**Results:** A total of 301 patients, aged 50.35±10.29 years were enrolled. Of these patients, 100 patients (33.2%) suffered postoperative low oxygenation ($PaO_2/FiO_2$=251.80±35.84). Compared with the normal oxygenation group, body mass index (BMI) (24.48±3.53 versus 23.1±3.27 kg/m², $P$=0.001), preoperative hemoglobin (115.79±29.27 versus 111.52±29.80 g/L, $P$=0.033), preoperative MELD (22.25±6.54 versus 20.24±5.74, $P$=0.008), and intraoperative urinary volume (1.25 [0.76, 1.89] versus 2.04 [1.49, 3.68] mL/kg/h, $P$=0.003) were higher in low oxygenation group. There were more cases of earlier AKIs that occurred after OLT in low oxygenation patients than that in normal group (47% versus 23.4%, $P$<0.001). Logistic analysis showed that the preoperative BMI (hazard ration [HR]=1.107, [1.010, 1.212], $P$=0.029) and early AKI after OLT (HR=2.115, [1.161, 3.855], $P$=0.014) were independent risk factors for postoperative low oxygenation.

**Conclusions:** The incidence of postoperative low oxygenation after liver transplantation in adults was 33.2%. BMI and early AKI after OLT were correlated with postoperative hypoxemia.

**MeSH Keywords:** **Acute Kidney Injury • Acute Lung Injury • Liver Transplantation**

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# Background

Orthotropic liver transplantation (OLT) is a life-saving therapy for patients with end-stage liver disease. However, postoperative pulmonary complications following OLT are associated with high morbidity and mortality rates [1–3]. Pulmonary complications are associated with long-term mechanical ventilation, long hospital stays, and poor outcomes [4,5]. The incidence of pulmonary complications is 42.1–87%, including pleural effusion, atelectasis, pneumonia, acute lung injury (ALI), and acute respiratory distress syndrome (ARDS) [2,3,5–7]. Lin et al. reported the overall mortality of liver transplant recipients was 13.1% and pulmonary causes accounted for 85.7% of the deaths [8].

Due to their chronically immunosuppressed status, liver transplant recipients are continually at risk for infectious pulmonary complications, and there are a number of early noninfectious pulmonary complications that plague the transplant recipient. ALI and its most severe form, the ARDS are common complications after liver transplantation that contribute to the morbidity and mortality of recipients in the acute postoperative stage [5]. Despite advances in surgical techniques and anesthesiologic management, the lung may still suffer throughout the postoperative stage. Patients with ALI are prone to developing ARDS, and mortality rate could be as high as 80–100% [9]. These complications arise because of numerous factors, including the underlying conditions that preceded transplantation, the transplant surgery itself, and post-transplantation liver or kidney dysfunction [7]. Injury may occur because liver transplantation is often associated with prolonged operative time, large volumes of fluid administration, and transfusion, as well as inflammatory responses related to ischemia reperfusion injury [10].

Hypoxemia is an abnormally low level of oxygen in the blood. ALI is defined as the acute onset of hypoxemia (a ratio of the arterial partial pressure of oxygen to fraction of inspired oxygen $PaO_2/FiO_2$ <300 mmHg) according to American-European Consensus Conference (AECC) definition [11]. Low $PaO_2/FiO_2$ indicates hypoxemia and reduction of oxygenation. At present, there is no research on the impact factor of oxygenation after liver transplantation. The object of the present study was to explore the incidence of low oxygenation among OLT patients after operation. Therefore, a comprehensive understanding of the risk factors of low oxygenation after liver transplantation is conducive to further taking effective intervention for improving prognosis.

# Material and Methods

## Patients

We retrospectively evaluated all adult patients who underwent single living-donor OLT between January 1, 2017 and December 31, 2017. Patients who meet the following criteria were enrolled: 1) adult patients over 18 years old; 2) patients undergoing liver transplantation for the first time; 3) patients receiving living-donor liver; and 4) the surgical method was orthotropic liver transplantation for patients. Exclusion criteria were as follows: 1) infant and young children; 2) previous organ transplantation; 3) multi-organ combined transplantation; 4) the surgical method was piggyback or venous transposition; 5) intra-operative cardiac arrest; 6) preoperative AKI patients; and 7) preoperative Chronic Kidney Disease (CKD) and GFR <60 mL/min. Ethical approval for this study was provided by Renji Hospital Ethics Committee, School of Medicine, Shanghai Jiao Tong University (Approved Number: [2018]019). The clinical trial has been registered at Chictr.org (ChiCTR1800018404).

Demographic characteristics, preoperative and postoperative laboratory examination were collected.

Intraoperative parameters included anesthesia and operative times, anhepatic phase, urine output, vasoactive agent use, diuretic administration and fluid management such as volume of intravenous crystalloid, colloids, red-cell concentrates, and plasma.

## Related definitions

Postoperative low oxygenation was defined as $PaO_2/FiO_2$, (P/F) <300 mmHg within 24 hours after operation [11].

Model for end-stage liver disease (MELD) score [12] was defined as MELD=0.967×ln(Scr/88.4 [μmol/L])+0.38× ln(TB[mg/dL])+1.12×ln(INR)+6.43

AKI was defined according to KDIGO 2012 [13] as any of the following: 1) increase in SCr by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours; or 2) increase in SCr to ≥1.5 times baseline within 48 hours; or 3) urine volume <0.5 mL/kg/hour for 6 hours. AKI that occurred within 24 hours after operation was defined as early AKI after OLT.

## Statistical analysis

Data were expressed as percentage, (mean ± standard deviation (SD), or median 25% with 75% interquartile range (IQR) as appropriate. Chi-squared analysis was used to compare categoric data. Continuous data were compared using Student *t*-test. The Mann-Whitney U test was used for data that failed tests of normality. Stepwise logistic regression analysis was performed to identify the risk factors. *P*<0.05 was considered to be statistically significant. We used SPSS 20.0 (SPSS, Chicago, IL, USA) for all analyses.
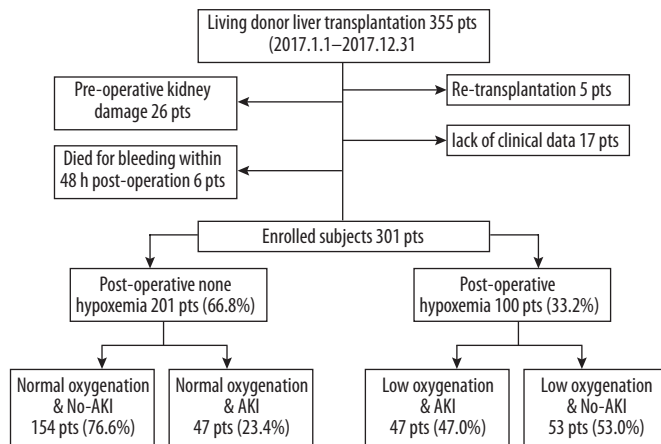
**Figure 1.** Study patient flow chart.



## Results

There were 355 patients with living-organ liver transplantation in 2017 who were screened. Of these, 54 patients met our exclusion criteria, including patients with preoperative kidney damage (n=26), re-transplants (n=5), re-operation or died for bleeding within 48-hour after operation (n=6), and lack of clinical data (n=17) (Figure 1).

In 301 enrolled patients, 238 patients (79.1%) were males. The mean age was 50.35±0.29 years old and body mass index (BMI) was 23.57±3.42kg/m$^2$. Indications for transplantation were hepatoma (n=134; 44.52%), hepatic cirrhosis including hepatitis B virus (HBV), biliary, alcohol, immune, and hepatitis C virus (HCV) (n=127; 42.19%), fulminant hepatic failure (n=31; 10.30%), cholangiocarcinoma (n=6; 1.99%), and other (n=3; 1.00%). The average MELD score was 20.90±6.08. Other baseline characteristics are presented in Table 1.

Within 24 hours after operation, postoperative low oxygenation occurred 100 patients (33.2%). Compared to normal oxygenation group, postoperative low oxygenation patients had higher BMI (24.48±3.53 versus 23.1±3.27kg/m$^2$, $P$=0.001), higher preoperative hemoglobin (115.79±29.27 versus 111.52±29.80g/L, $P$=0.033), higher preoperative MELD (22.25±6.54 versus 20.24±5.74, $P$=0.008) at baseline (Table 1).

Patients with postoperative low oxygenation tended to have longer surgery time and more bleeding during operation although there was no statistic significant. Intraoperative urine volume was 1.25 [0.76, 1.89] and 2.04 [1.49, 3.68] mL/kg/hour ($P$=0.003) in patients with and without postoperative low oxygenation, respectively. There were 47% and 23.4% patients in low oxygenation and normal oxygenation group suffered early AKI after OLT ($P$<0.001). There was no statistically significant with other clinical parameters between these 2 groups (Table 1).

In a multivariate logistic regression, BMI (hazard ratio [HR]=1.127, CI [1.026, 1.239], $P$=0.012) and postoperative AKI (HR=2.053, CI [1.107, 3.808], $P$=0.023) were the independent risk factors for low oxygenation after OLT (Table 2).

## Discussion

In the present study, we found that postoperative low oxygenation was a common complication for liver transplantation with incidence of 33.2%. BMI and postoperative AKI were the independent risk factors for low oxygenation.

The most effective methods for end-stage liver diseases is liver transplantation which takes long operative time and is prone to hemodynamic instability. Although the success of liver transplantation has increased over the last 2 decades, postoperative complications are common and contribute to significant mortality. Pulmonary complications resulting in low oxygenation is one of the most important complication and is often life-threatening with 37.5–45.8% mortality [3,14,15]. Hong et al. reported that 51.9% of patients had pulmonary complications after surgery, including pleural effusion, atelectasis, pneumonia, pulmonary edema, and ARDS [16]. Aduen et al. reported that 52% of patients had different degrees of pulmonary edema within 72 hours after surgery [17]. Recently, Golfieri et al. reported that 86.7% patients had non-infectious pulmonary abnormalities and 44.7% of patients had pulmonary edema appear during the first postoperative week [9]. They also confirmed that persistent non-infectious abnormalities were the major independent predictors of postoperative pneumonia, which contribute to acute respiratory distress syndrome [18]. Most of these studies diagnosed pulmonary complications based on radiology methods. Few of the studies focused on pulmonary function, especially oxygenation marker. Actually, the mortality would be high as 80–100% in liver transplantation

**Table 1.** Baseline and perioperative characteristics in 301 patients undergoing OLT# divided according to low or normal oxygenation after transplantation.

| Variables | All patients (n=301) | | Low oxygenation group (n=100) | | Normal oxygenation group (n=201) | | P |
|---|---|---|---|---|---|---|---|
| Age (y) | 50.35±10.29 | | 51.47±10.84 | | 49.66±9.96 | | 0.099 |
| Male n(%) | 238 | (79.1%) | 81 | (81.0%) | 157 | (78.1%) | 0.681 |
| BMI (kg/m²) | 23.57±3.42 | | 24.48±3.53 | | 23.1±3.27 | | 0.001* |
| **Primary diasease** | | | | | | | 0.825 |
| Hepatoma n (%) | 134 | (44.6%) | 48 | (48.0%) | 86 | (42.8%) | |
| Hepatic cirrhosis n (%) | 127 | (42.2%) | 39 | (39.0%) | 88 | (43.8%) | |
| Fulminant hepatic failure n (%) | 31 | (10.3%) | 12 | (12.0%) | 19 | (9.4%) | |
| Cholangiocarcinoma n (%) | 6 | (2%) | 0 | | 6 | (3.0%) | |
| Other n (%) | 3 | (0.9%) | 1 | (1.0%) | 2 | (1.0%) | |
| **Pre-OLT** | | | | | | | |
| MAP (mmHg) | 92.37±14.19 | | 92.38±15.68 | | 92.36±13.44 | | 0.993 |
| Alanine aminotransferase (U/l) | 40.00 [26.00, 72.75] | | 40.00 [27.00, 88.00] | | 40.00 [25.00, 67.50] | | 0.300 |
| Aspartate aminotransferase (U/l) | 48.00 [30.00, 88.75] | | 50.00 [30.00, 93.00] | | 47.00 [30.00, 87.50] | | 0.795 |
| Blood urea nitrogen (mmol/l) | 5.23±2.39 | | 5.60±2.84 | | 5.05±2.12 | | 0.058 |
| Serum creatinine (µmol/l) | 66.04±18.42 | | 68.64±21.34 | | 64.75±16.68 | | 0.112 |
| Prothrombin time (s) | 18.62±8.33 | | 19.36±8.97 | | 18.25±8.00 | | 0.281 |
| Hemoglobin (g/dl) | 112.95±29.64 | | 115.79±29.27 | | 111.52±29.80 | | 0.033 |
| Hematocrit (%) | 33.51±8.58 | | 34.37±8.34 | | 33.10±8.69 | | 0.236 |
| Platelets (×10⁹/l) | 76.50 [46.25, 127.00] | | 74.00 [43.20, 112.75] | | 78.00 [47.25, 136.50] | | 0.333 |
| Albumin (g/dl) | 36.28±6.57 | | 36.22±6.67 | | 36.31±6.49 | | 0.912 |
| Glucose (mmol/l) | 6.30±3.27 | | 6.42±3.40 | | 6.23±3.21 | | 0.649 |
| MELD | 20.90±6.08 | | 22.25±6.54 | | 20.24±5.74 | | 0.008* |
| **Intra-OLT** | | | | | | | |
| Operative time (min) | 440.52±76.74 | | 452.56±78.60 | | 434.60±75.30 | | 0.057 |
| Anhepatic phase (min) | 45.79±9.32 | | 47.00±9.62 | | 45.19±9.14 | | 0.120 |
| Ascites volume (l) | 0.10 [0, 1.00] | | 0.20 [0, 1.00] | | 0.10 [0, 1.00] | | 0.319 |
| 0 | 142 | (47.2%) | 42 | (42.0%) | 100 | (49.8%) | 0.447 |
| ≤1 l | 107 | (35.5%) | 39 | (39.0%) | 68 | (33.8%) | |
| >1 l | 52 | (17.3%) | 19 | (19.0%) | 33 | (16.4%) | |
| Bleeding volume (l) | 0.50 [0.40, 1.00] | | 0.60 [0.40, 1.00] | | 0.50 [0.40, 0.80] | | 0.081 |
| ≤1 l | 258 | (85.7%) | 82 | (82.0%) | 176 | (87.6%) | 0.194 |
| >1 l | 43 | (14.3%) | 18 | (18.0%) | 25 | (12.4%) | |
| Urine volume (ml/kg/h) | 1.86 [1.13, 3.11] | | 1.25 [0.76, 1.89] | | 2.04 [1.49, 3.68] | | 0.003* |

**Table 1 continued.** Baseline and perioperative characteristics in 301 patients undergoing OLT† divided according to low or normal oxygenation after transplantation.

| Variables | All patients (n=301) | | Low oxygenation group (n=100) | | Normal oxygenation group (n=201) | | P |
|---|---|---|---|---|---|---|---|
| *Crystalloid perfusion* | | | | | | | |
| Perfusion volume (l) | 2.60 [2.00, 3.25] | | 2.50 [1.96, 3.25] | | 2.60 [2.00, 3.44] | | 0.113 |
| ≤3 l n(%) | 201 | (66.8%) | 72 | (72.0%) | 129 | (64.2%) | 0.175 |
| >3 l n(%) | 100 | (33.2%) | 28 | (28.0%) | 72 | (35.8%) | |
| *Colloid perfusion* | | | | | | | |
| Perfusion volume (l) | 2.55 [1.75, 3.60] | | 2.60 [1.98, 3.80] | | 2.50 [1.73, 3.55] | | 0.211 |
| ≤3 l n(%) | 186 | (61.8%) | 59 | (59.0%) | 127 | (63.2%) | 0.482 |
| >3 l n(%) | 115 | (38.2%) | 41 | (41.0%) | 74 | (36.8%) | |
| *Red-cell concentrate perfusion* | | | | | | | |
| Perfusion volume (l) | 0.80 [0, 1.20] | | 0.80 [0, 1.20] | | 0.80 [0, 1.20] | | 0.609 |
| 0 | 108 | (35.9%) | 35 | (35.0%) | 73 | (36.3%) | 0.684 |
| ≤1 l | 112 | (37.2%) | 35 | (35.0%) | 77 | (38.3%) | |
| >1 l | 81 | (26.9%) | 30 | (30.0%) | 51 | (25.4%) | |
| *Plasma perfusion* | | | | | | | |
| Perfusion volume (l) | 0.40 [0, 0.80] | | 0.40 [0, 0.80] | | 0.40 [0, 0.70] | | 0.609 |
| 0 | 127 | (42.2%) | 43 | (43.0%) | 84 | (41.8%) | 0.965 |
| ≤1l | 136 | (45.2%) | 45 | (45.0%) | 91 | (45.3%) | |
| >1l | 38 | (12.6%) | 12 | (12.0%) | 26 | (12.9%) | |
| *Vasoactive agent use* | | | | | | | |
| Number of agent | | | | | | | |
| 0 or 1 | 234 | (77.7%) | 76 | (76.0%) | 158 | (78.6%) | 0.609 |
| >1 | 67 | (22.3%) | 24 | (24.0%) | 43 | (21.4%) | |
| *Diuretic administration* | | | | | | | |
| Furosemide dose | 50.00 [40.00, 80.00] | | 60.00 [32.50, 95.00] | | 50.00 [40.00, 80.00] | | 0.341 |
| 0 n (%) | 49 | (16.3%) | 17 | (17.0%) | 32 | (15.9%) | 0.518 |
| ≤60 mg n(%) | 161 | (53.5%) | 49 | (49.0%) | 112 | (55.7%) | |
| >60 mg n(%) | 91 | (30.2%) | 34 | (34.0%) | 57 | (28.4%) | |
| **Post-OLT** | | | | | | | |
| P/F at 24h | 354.12±96.03 | | 251.80±35.84 | | 404.52±73.63 | | <0.001* |
| Ventilation duration (min) | 275.00 [140.00, 487.50] | | 360.00 [166.25, 673.75] | | 260.00 [127.50, 422.50] | | 0.014* |
| Serum creatinine at 24 h (µmol/l) | 89.05±41.23 | | 105.18±58.64 | | 80.82±24.98 | | <0.001* |
| Serum creatinine at 48 h (µmol/l) | 82.78±52.17 | | 99.71±73.63 | | 74.01±33.37 | | <0.001* |
| AKI n(%) | 94 | (31.2%) | 47 | (47%) | 47 | (23.4%) | <0.001* |

OLT – orthotropic liver transplantation; BMI – body mass index; MAP – mean atrial pressure; MELD – model of end-stage liver disease; P/F – a ratio of the arterial partial pressure of oxygen to fraction of inspired oxygen, $PaO_2/FiO_2$; AKI – acute kidney injury.
* P<0.05 – Compared between low oxygenation and normal oxygenation group

**Table 2.** Stepwise logistic regression showing risk factors of post-OLT low oxygenation.

| Variables | HR | 95% CI | P |
|---|---|---|---|
| Gender (male) | 1.003 | [0.481, 2.089] | 0.994 |
| Age (y) | 1.011 | [0.983, 1.040] | 0.437 |
| BMI (kg/m²) | 1.127 | [1.026, 1.239] | 0.012* |
| Pre-OLT blood urea nitrogen (mmol/l) | 1.088 | [0.947, 1.249] | 0.235 |
| Pre-OLT Hemoglobin (g/l) | 1.005 | [0.994, 1.016] | 0.351 |
| Pre-OLT MELD | 1.021 | [0.970, 1.075] | 0.423 |
| Operation Duration (min) | 1.003 | [0.999, 1.007] | 0.113 |
| Intra-OLT bleeding volume (l) | 1.000 | [1.000, 1.000] | 0.485 |
| Intra-OLT urine volume (ml/kg/h) | 1.000 | [0.999, 1.000] | 0.530 |
| Post-OLT AKI (yes) | 2.053 | [1.107, 3.808] | 0.023* |

OLT – orthotropic liver transplantation; BMI – body mass index; MELD – model of end-stage liver disease; AKI – acute kidney injury.
* P<0.05.

patients with ARDS [19], which suggests that pulmonary function might affect short-term prognosis directly. In the present study, we evaluated pulmonary function by using $PaO_2/FiO_2$ ratio (P/F) within 24 hours after surgery, which was believed to be an oxygenation marker and reduce the effects of rejection and infection. P/F lower than 300 mmHg indicates lung injury. Our results showed that the incidence of low oxygenation was 33.2% after operation. It was much higher than the ARDS incidence reported in liver transplantation patients [19,20], since a mild injury or no severe hypoxemia was included in our cohort.

There are several aspects to consider. First, hemodynamic instability can lead to failure of donor organ procurement in brain-dead donors, which can influence the complications of the recipient. ECMO (extracorporeal membrane oxygenation), provides a mechanical pump for circulation and a membrane gas exchange for oxygenation, can maintain hemodynamically unstable donors for liver transplant recipients. Chen et al. have shown that there were no significant differences in complications of recipients between ECMO for hemodynamically unstable brain-dead donors and no ECOM for hemodynamic stable donors [21]. Second, some research has focused on the risk factors of the recipient itself for pulmonary complications. Preoperative elevated creatinine, hemodialysis, total bilirubin increasing, acute rejection, INR, and restrictive lung ventilation dysfunction might be risk factors for postoperative pulmonary complications [9,16,17]. Third, operation related factors, such as extended cold storage, ischemia/reperfusion-induced graft injury, and transfusion of plasma-containing blood products during surgery have also been documented to increase the occurrence of pulmonary complications [5,22–26]. One of the other rare reasons reported by Li et al. was rituximab-induced ARDS with ABO-incompatible liver transplant recipient, where ECMO support was effectiveness this liver transplant

recipients with poor respiratory functions [27]. In the present cohort in our study, we did not find that any of the aforementioned factors had any difference between low oxygenation patients and normal oxygenation patients. However, BMI and AKI after operation were the independent risk factors for low oxygenation. Obese patients will be at a greater risk of hypoxemia during anesthesia [28,29].There was few of studies that have focused on the relationship between postoperative hypoxemia and BMI. Gallagher et al. found that severe and prolonged episodes of postoperative hypoxemia were a consistent finding in obesity patients with BMI 48±2 kg/m² [30], implicating that obese weight might result in postoperative low oxygenation. In our cohort, the mean BMI in patients with low oxygenation was over 24 kg/m², which was defined as overweight in the Chinese population [31]. Recently, a larger retrospective cohort study investigated 15 238 adult patients who underwent general anesthesia for elective noncardiac surgery and found that adjusted odds ratio of intraoperative hypoxemia and prolonged hypoxemia (>5 minutes) increased 27% and 97% respectively in patients with BMI 25–29.9 kg/m² [32]. The limitation of BMI is well-known. It does not differentiate among muscle mass, fat mass, and edema. For our patients with end stage liver disease, higher BMI might be caused by more pleural and peritoneal effusion, which also contributed to hypoxemia, although there was no difference in albumin concentration or ascites volume between the two groups.

In addition to pulmonary reasons, hepatic and renal function were major impact factors on pulmonary complications. Kidney-lung crosstalk has been studied for a long time [33]. In the early 1950s, it was reported that one patient with acute renal failure had complicated pulmonary congestion and effusion in the chest radiographs [34]. In recent years, some authors have found that the mortality of AKI combined lung injury patients

remained high in the intensive care unit (ICU), and the need for assisted ventilation support for patients with lung injury was twice as high a risk in AKI patients than that in non-AKI patients [35,36]. However, there were few reports of AKI complicated with lung injury after liver transplantation. Our findings showed 47% of low oxygenation patients had postoperative AKI within 24 hours, which was significantly higher than that of normal oxygenation patients. Early AKI was 2.0 times HR for low oxygenation than non-AKI in the patients after OLT. These results indicated lung injury was closely related to the occurrence of AKI after liver transplantation. It was notable that patients with low oxygenation had lower intraoperative urine volume, tended to have more bleeding volume and longer operative time, implicating that operation-related factors might contribute postoperative AKI.

Pulmonary edema plays a major role in AKI complicated lung injury, including cardiogenic pulmonary edema and non-cardiac pulmonary edema. It has long been recognized that cardiogenic pulmonary edema is caused by over-fluid intake. This is the one reported cause of post-AKI related low oxygenation [37]. Hydrostatic pressure is increased due to water clearance disorders after renal injury, introducing the infiltration of water molecules from pulmonary capillaries to interstitial lung, thus resulting in pulmonary edema. The damage of pulmonary endothelial cells directly leads to increased capillary permeability. On this basis, the pulmonary epithelial cells are damaged simultaneously, and the water enters the alveolar cavity, leading to alveolar edema [38]. The key factors causing non-cardiogenic pulmonary edema are the accumulation of pro-inflammatory factors and the damage of endothelium and epithelium caused by inflammatory cell infiltration [39]. Many studies have found that the increase of inflammatory factors was related to AKI, suggesting that AKI can induce inflammatory response [40–43]. Other studies have demonstrated that

pro-inflammatory cytokines can directly damage lung endothelial cells leading to lung injury and non-cardiac pulmonary edema [44,45]. In patients with AKI, the circulation pro-inflammatory cytokinesis increased, and the clearance of inflammatory cytokines is relatively reduced, thus causing the accumulation of inflammatory cytokines and damaging lung vessel endothelial cells, which leads to ALI. Meanwhile ALI might further aggravate renal injury through inflammation, apoptosis, and blood-gas exchange disorders [46].

There were some limitations in this observational study. First, this study was a retrospective study with a small sample size. Second, the selected patients had a shorter observation time which might have created selection bias. Third, we included AKI patients only 48 hours after surgery; we might have missed some AKI patients after 48 hours, which might have led to the deviation of risk factors for AKI occurrence. Fourth, only P/F was used as the diagnostic criteria for ALI, while other relevant evidences, including imaging and pathogenic data, were not available, which might lead to misdiagnosis. Fifth, the causal relationship between AKI and ALI needs to be systematically observed and explored. Larger sample size, perspectives studies with radiology data and longer follow-up should be performed to demonstrate the risk factors for post-OLT hypoxemia.

## Conclusions

Low oxygenation was not uncommon after liver transplantation in adults, with an incidence of 33.2%. BMI and early AKI after OLT were associated with postoperative hypoxemia.

### Conflicts of interest

None.

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