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Original Article

Hospital-Acquired Pneumonia in Adult Living-Donor Liver Transplant Recipients: A Retrospective Cohort Study of Risk Factors and Outcomes

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ABSTRACT

Background: Hospital-acquired pneumonia (HAP) is a frequent and serious complication after liver transplantation. Local data remain limited. Methods: We conducted a retrospective cohort study of adult living-donor liver transplant (LDLT) recipients between 2019 and 2024. HAP was defined as onset ≥48 hours after admission with compatible clinical, radiological, and microbiological evidence. Independent predictors were assessed using multivariable logistic regression including age, sex, Model for End-Stage Liver Disease (MELD) score, chronic kidney disease or end-stage renal disease (CKD/ESRD), diabetes, body mass index, and albumin. Associations between HAP and ventilator days, intensive care unit (ICU) stay, and hospital stay were analysed using adjusted negative binomial regression. Results: Among 163 LDLT recipients, 46 (28%) developed HAP. Independent predictors were CKD/ESRD (adjusted odds ratio [aOR] 4.07, 95% confidence interval [CI] 1.37–12.09, p=0.011) and female sex (aOR 2.78, 95% CI 1.12–6.91, p=0.028). After adjustment, HAP was associated with longer ventilator support (adjusted rate ratio [aRR] 1.96, 95% CI 1.32–2.86, p=0.001), ICU stay (aRR 1.52, 95% CI 1.05–2.17, p=0.027), and hospital stay (aRR 1.59, 95% CI 1.12–2.27, p=0.009). Conclusions: In LDLT recipients, CKD/ESRD and female sex predicted HAP. HAP was linked with prolonged ventilation, ICU stay, and hospitalisation. Focused preventive strategies in high-risk recipients may reduce complications and resource use.

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INTRODUCTION

Hospital-acquired pneumonia (HAP) is one of the most common healthcare-associated infections and contributes substantially to morbidity, mortality, and healthcare resource use [1,2]. Liver transplant recipients are particularly vulnerable because of immunosuppression, invasive devices, and prolonged intensive care exposure [3,4]. Diagnosis is based on compatible clinical features, elevated inflammatory markers such as C-reactive protein (CRP) and leucocyte count [5], new radiological infiltrates [6] and supportive microbiological evidence.

Despite the clinical importance of HAP, data describing its predictors and outcomes after living-donor liver transplantation (LDLT) in South Asia remain scarce. Previous studies in deceased donor liver transplant cohorts have identified comorbidities and multidrug-resistant organisms as key determinants of post-transplant pneumonia [7,8,9,10,11]. However, these findings may not be directly applicable to LDLT, where recipient profiles and perioperative practices differ.

This study aimed to: (i) determine the incidence of HAP among adult LDLT recipients; (ii) identify baseline predictors using a prespecified multivariable model; and (iii) assess the adjusted association between HAP and durations of ventilator support, intensive care unit (ICU) stay, and total hospital stay.

MATERIALS AND METHODS

Study design and setting

We conducted a retrospective cohort study. All adult LDLT recipients aged ≥18 years transplanted between 2019 and 2024 were eligible. Data were extracted from electronic medical records between July and December 2024.

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Ethics

Institutional review board approval was obtained (IRB 240-24). All identifiers were anonymised in accordance with institutional policy.

Eligibility criteria

We included all consecutive adult LDLT recipients during the study period. Exclusion criteria were: paediatric recipients; pneumonia at admission or within 48 hours of hospitalisation; active malignancy; HIV infection; pregnancy or breastfeeding; and incomplete clinical data. A total of 506 patient files were reviewed, of which 163 were included in this study.

Definitions

HAP was defined as a new infection developing ≥48 hours after transplant with compatible clinical, radiological, and microbiological evidence [5]. Clinical indicators included fever, increased respiratory secretions, suction requirement, or new or worsening hypoxaemia. Laboratory and imaging features included raised inflammatory markers, positive tracheal cultures, and new radiographic infiltrates [5,6].

Variables

We collected demographic data, body mass index, comorbidities (with emphasis on CKD/ESRD [6,12]) and diabetes, hypertension, MELD score [13], serum albumin, inflammatory markers, radiological findings, and microbiological culture results. Outcomes were ventilator days, ICU stay, and total hospital stay.

Radiology

Chest radiographs at the time of suspected HAP were categorised as: (i) infiltrates with or without consolidation; (ii) atelectasis; (iii) consolidation with pleural effusion; or (iv) pleural effusion alone [5]. Radiological findings were compared descriptively between groups but excluded from predictor models to prevent incorporation bias.

Statistical Analysis

Continuous variables were tested for normality using the Shapiro–Wilk test. As stay outcomes were non-normally distributed, medians with interquartile ranges (IQRs) were reported and compared using the Mann–Whitney U test. Categorical variables were compared using the Chi-square test. A two-sided p-value <0.05 was considered statistically significant.

Predictors of HAP were evaluated using multivariable logistic regression with a prespecified model including age, sex, MELD score, CKD/ESRD, diabetes, body mass index, and albumin.

This prespecified multivariable model was used to avoid data-driven variable selection, reduce overfitting, and ensure adjustment based on established clinical relevance. The included covariates are well-known confounders that influence both infection risk and hospital outcomes: age (infection susceptibility), sex (immune differences), MELD score (illness severity), CKD/ESRD (impaired immunity), diabetes (higher infection risk), BMI (respiratory and metabolic effects), and albumin (nutritional/inflammatory status). Adjusting for these factors improves the validity and interpretability of the associations examined. Model calibration was assessed with the Hosmer–Lemeshow test.

Associations between HAP and length-of-stay outcomes were estimated using negative binomial regression with a log link, adjusted for the same covariates. Results are presented as adjusted rate ratios (aRR) with 95% confidence intervals (CIs). Analyses were performed using IBM SPSS Statistics v25.

RESULTS

Cohort characteristics

A total of 163 adult LDLT recipients were included. HAP developed in 46 (28%) patients. The study flow is shown in Figure 1.

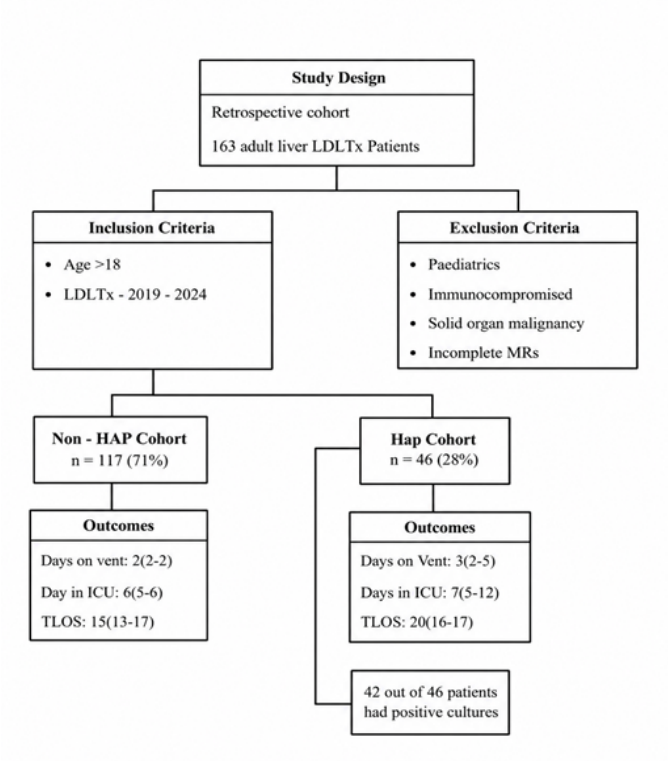


Figure 1. Flow diagram showing screening (506), exclusions (343), and final inclusion (163) of adult LDLT recipients.

Baseline characteristics

The mean age was comparable between groups. Males predominated in the non-HAP group (87% vs 65%, p<0.001). CKD/ESRD was more frequent among HAP cases (26% vs 5%, p=0.001). Median MELD score was slightly higher in the HAP group (18 vs 17, p=0.02), while albumin levels were similar (p=0.15). Detailed baseline characteristics are presented in Table 1.

Variable	HAP (n = 46)	Non-HAP (n = 117)	p-value
Age (years) (Mean ± SD)	49.1 ± 9.9	47.8 ± 10.7	0.458
Gender (Male), n (%)	30 (65%)	102 (87%)	<0.001
Any Comorbidity, n (%)	38 (83%)	80 (68%)	<0.001
Diabetes Mellitus	24 (52%)	55 (47%)	0.337
Hypertension	19 (41%)	35 (29%)	0.115
CKD/ESRD	12 (26%)	7 (5%)	0.001
BMI Category, n (%):			0.244
Underweight	2 (4%)	6 (5%)	
Normal	18 (39%)	61 (52%)	
Overweight	19 (41%)	34 (29%)	
Obese	7 (15%)	16 (13%)	
Lab Parameters (Median [IQR])			
– MELD Score	18 [13–23]	17 [14–21]	0.02
– Albumin (g/dL)	2.9 [2.5–3.4]	3.0 [2.7–3.3]	0.15

Clinical, radiological and laboratory parameters

Indicators of infection, including increased oxygen requirement, suction need, fever, shock, elevated CRP, leukocytosis, and positive cultures, were all significantly more frequent in patients with HAP (all p<0.001). Infiltrates and consolidation with effusion were the dominant findings in HAP, whereas atelectasis and isolated effusion were frequent in non-HAP cases (p<0.001). Full details are shown in Table 2.

Table 2. Clinical, radiological, and laboratory parameters among recipients with and without HAP

Clinical Parameters	HAP (n = 46)	Non-HAP (n = 117)	p-value
Oxygen Requirement, n (%)	38 (83%)	22 (18%)	<0.001
Suction Requirement, n (%)	43 (93%)	9 (7%)	<0.001
Fever Spikes, n (%)	38 (83%)	5 (4%)	<0.001
Shock, n (%)	29 (63%)	9 (7%)	<0.001
Laboratory Parameters			
CRP Positive, n (%)	34 (73%)	17 (14%)	<0.001
WBC > 11,000, n (%)	36 (78%)	52 (44%)	<0.001
Tracheal Culture Positive, n (%)	42 (91%)	2 (1%)	<0.001
Radiological Finding			
Infiltrates ± Consolidation, n (%)	20 (43%)	14 (11%)	<0.001
Atelectasis, n (%)	3 (6%)	53 (45%)	<0.001
Consolidation with Pleural Effusion, n (%)	16 (34%)	3 (2%)	<0.001
Pleural Effusion, n (%)	6 (13%)	26 (22%)	<0.001

Microbiology

Klebsiella pneumoniae was the most frequent isolate (30%), followed by Pseudomonas aeruginosa (11%), Acinetobacter baumannii (11%), and Candida albicans (11%). Other pathogens included Candida tropicalis, Stenotrophomonas maltophilia, Methicillin-resistant Staphylococcus aureus, Aspergillus fumigatus, and Escherichia coli (Table 3).

Table 3. Distribution of causative organisms in recipients with HAP

Organism	n (%)
Klebsiella pneumoniae	13 (31%)
Pseudomonas aeruginosa	5 (12%)
Acinetobacter baumannii	5 (12%)
Candida albicans	5 (12%)
Candida tropicalis	4 (9%)
Stenotrophomonas maltophilia	4 (9%)
MRSA (Methicillin-resistant S. aureus)	4 (9%)
Aspergillus fumigatus	1 (2%)
Escherichia coli	1 (2%)

[a]Percentages are based on culture-positive patients only (n = 42). Four patients had no growth on respiratory cultures despite meeting clinical and radiological criteria for hospital-acquired pneumonia.

Respiratory cultures demonstrated no growth in four patients despite a clinical diagnosis of HAP. These patients met predefined clinical and radiological criteria for HAP, and antimicrobial therapy had been initiated before culture acquisition. Consequently, microbiological confirmation was not obtained in all cases.

Predictors of HAP

Table 4 summarizes independent predictors from multivariable analysis. CKD/ESRD (adjusted odds ratio [aOR] 4.07, 95% confidence interval [CI] 1.37–12.09, p=0.011) and female sex (aOR 2.78, 95% CI 1.12–6.91, p=0.028) were independent predictors of HAP. Other variables were not statistically significant. The Hosmer–Lemeshow goodness-of-fit test was not statistically significant (p = 0.806), indicating adequate calibration and good model fit.

Table 4. Multivariable logistic regression analysis of predictors of HAP

Predictor	Adjusted OR	95% CI for OR	p-value
Age (years)	1.019	0.979 – 1.061	0.363
Body Mass Index	1.271	0.783 – 2.064	0.331
Female Gender (ref: Male)	2.780	1.119 – 6.909	0.028
Diabetes Mellitus	1.420	0.410 – 4.850	0.576
CKD/ESRD (Yes vs No)	4.071	1.371 – 12.094	0.011
MELD Score	1.046	0.986 – 1.109	0.138
Albumin (g/dL)	0.622	0.306 – 1.264	0.189

Impact of HAP on clinical outcomes

Recipients who developed HAP had significantly longer durations of ventilatory support, ICU stay, and hospitalisation compared with those without HAP. Median ventilator days were 3 versus 2 (p<0.001), ICU stay 7 versus 6 days (p<0.001), and hospital stay 20 versus 15 days (p<0.001) (Table 5).

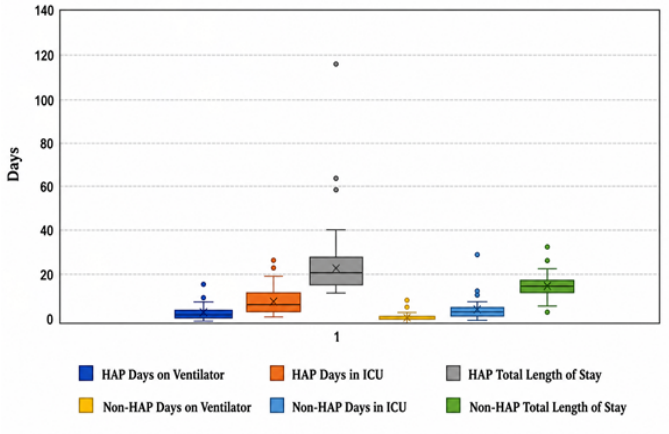
Table 5. Post-transplant outcomes among recipients with and without HAP

Outcome	HAP (n = 46) Median [IQR]	Non-HAP (n =117)	p-value
Days on Ventilator	3 [2–5]	2 [2–2]	<0.001
ICU Stay (days)	7 [5–12]	6 [5–6]	<0.001
Total Length of Stay (days)	20 [16–27]	15 [13–17]	<0.001

These differences are illustrated in Figure 2, showing the distribution of ventilator days, ICU stay, and hospital stay by HAP status.

Figure 2. Box-and-whisker plots comparing ventilator days, ICU stay, and hospital stay between HAP and non-HAP groups

In multivariable analysis adjusting for baseline characteristics, HAP



remained an independent determinant of prolonged stay. It was associated with longer ventilator support (adjusted rate ratio [aRR] 1.96,

95% CI 1.32–2.86, p=0.001), extended ICU stay (aRR 1.52, 95% CI 1.05–2.17, p=0.027), and longer hospitalisation (aRR 1.59, 95% CI 1.12–2.27, p=0.009) (Table 6).

Table 6. Adjusted effect of HAP on clinical outcomes

Outcome	Adjusted Rate Ratio (aRR)	95% CI	p-value
Ventilator days	1.96	1.32 – 2.86	0.001
ICU stay	1.52	1.05 – 2.17	0.027
Total hospital stay	1.59	1.12 – 2.27	0.009

DISCUSSION

In this cohort of adult LDLT recipients, HAP developed in over one quarter of patients and was independently associated with prolonged ventilatory support, ICU stay, and total hospitalisation. CKD/ESRD and female sex were the principal baseline predictors.

The observed incidence of 28% is comparable with reports from other transplant centres, where rates range from 20% to 35% depending on patient selection, surveillance intensity, and diagnostic criteria [11,14,15]. A recent Turkish single-centre study found a 25.6% rate of post-transplant pneumonia, while an Egyptian series reported similar findings and highlighted the impact of preventive bundles [14,15].

The strong association between CKD/ESRD and HAP supports evidence that renal impairment increases infection risk through reduced immune competence, fluid overload, and uraemic toxicity [6,12,16]. A recent European analysis of solid-organ recipients also identified CKD as a major determinant of pneumonia and adverse outcomes [16]. The association with female sex has been less consistently reported. While biological sex may influence immune responses, hormonal regulation, and pharmacokinetics of immunosuppressive therapy, this finding should be interpreted cautiously given the marked sex imbalance in our cohort, with females underrepresented in the non-HAP group. Residual confounding and instability from the small female subgroup may have influenced this association. Validation in larger, more balanced cohorts is required.

The microbiological spectrum in this cohort was dominated by Klebsiella pneumoniae, Pseudomonas aeruginosa, and Acinetobacter baumannii, reflecting global and regional patterns of multidrug resistance in nosocomial pneumonia [17]. Fungal pathogens, including Candida and Aspergillus, were also identified, emphasising the need for early detection and antifungal stewardship [18]. These findings reinforce the need for empiric antimicrobial regimens informed by local resistance profiles, followed by timely de-escalation once cultures are available.

Radiological findings were consistent with previous descriptions, where infiltrates and consolidation with pleural effusion are more characteristic of infection, while atelectasis or isolated effusion are commonly non-infective [5]. Recognition of these patterns can assist in early diagnostic stratification and help optimise antibiotic use.

Recipients with CKD/ESRD should be recognised as a high-risk subgroup and prioritised for preventive measures such as ventilator care bundles, strict oral hygiene, and early mobilisation [5,15]. Early recognition and targeted empirical therapy may reduce complications and resource utilisation. The additional ventilator and ICU days attributable to HAP represent a significant burden, particularly in resource-limited settings.

These findings contribute to the limited literature on HAP in LDLT and highlight areas for preventive intervention. Future work should focus on multicentre prospective studies to validate these predictors, explore interventional bundles for high-risk recipients, and incorporate time-dependent factors such as ventilation duration and immunosuppressive regimens.

Strengths of this study include a complete dataset with no missing variables, prespecified multivariable modelling to minimise bias, use of appropriate regression techniques for skewed outcomes, and detailed microbiological and radiological review. However, the single-centre retrospective design, moderate sample size, absence of detailed antimicrobial resistance profiling, and lack of external validation may limit generalisability. Future multicentre studies are needed to validate these findings.

CONCLUSIONS

In LDLT recipients, CKD/ESRD and female sex independently predicted the development of HAP. HAP was associated with significantly longer ventilator use, ICU stay, and total hospitalisation. Focused infection-prevention strategies and early identification of high-risk recipients may improve outcomes and reduce healthcare burden after LDLT.

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Declaration

The authors used a language-editing tool to improve readability; all scientific content and interpretation remain author-generated. No external funding was received for this study.

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