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The Role of Pretransplant Infections in Pediatric Receiving LDLT in Indonesia: A 7-y Retrospective Study

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Background. Liver transplantation is the definitive treatment for pediatric end-stage liver disease. Infections post-transplantation might significantly affect the outcome of the surgery. This study aimed to identify the role of pretransplant infection among children who underwent living donor liver transplantation (LDLT) in Indonesia. **Methods.** This is an observational, retrospective cohort study. A total of 56 children were recruited between April 2015 and May 2022. Patients were categorized into 2 according to the presence of pretransplantation infections requiring hospitalization before the surgery. Diagnosis of posttransplantation infection was observed for up to 1 y based on the clinical features and laboratory parameters. **Results.** The most common indication for LDLT was biliary atresia (82.1%). Fifteen of 56 patients (26.7%) had a pretransplant infection, whereas 73.2% of patients were diagnosed with a posttransplant infection. There was no significant association between pretransplant and posttransplant infection in all 3-time points (≤ 1 mo, 2–6 mo, and 6–12 mo). The most common organ involvement posttransplantation was respiratory infections (50%). The pretransplant infection did not significantly affect posttransplant bacteremia, length of stay, duration of mechanical ventilation, initiation of enteral feeding, hospitalization cost, and graft rejection. **Conclusions.** Our data showed that pretransplant infections did not significantly affect clinical outcomes in post-LDLT procedures. A prompt and sufficient diagnosis and treatment before and after the LDLT procedure is the best way to obtain an optimal outcome.

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Liver transplant is the standard therapeutic modality for many cases of acute and chronic end-stage liver diseases.^{1,2} Because of cultural and religious appropriation, living donor liver transplantation (LDLT) is a preferred approach over deceased donor liver transplantation in the Asia region.³

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In the past years, a significant improvement in the survival of posttransplant patients was seen. This progress was mainly due to the development of a more potent immunosuppressive agent that prevents rejections.² However, the rate of opportunistic infection has increased in accordance with the rise in the survival rate, hindering the effort to lower the mortality and morbidity posttransplantation.^{2,4} A study among postorthotopic liver transplantation patients revealed that posttransplant infections occurred in more than half of the transplant recipients, with 70% of them being bacterial infections.^{2,4,5}

Various studies attempted to analyze the impact of pretransplant infections on the patients' clinical outcomes following transplantation. A study by Kim et al reported the effect of infection that occurred 1 mo before LDLT in 357 recipients showed a significant longer length of stay (LOS) in the ICU of >7 d, a higher rate of posttransplant infections, and a higher number of bacteremia cases posttransplant.⁶ In 2019, Heldman et al supported a previous study that reported that pretransplant infection significantly affects the incidence of posttransplant infection and longer LOS in hospitals.⁷ However, conflicting results were found in a study by Lin et al, which demonstrated no significant difference in posttransplant infection rates, the length of ICU stay, 1-y survival, and graft rejection between with and without pretransplant infection group.⁸ In Indonesia, Cipto Mangunkusumo Hospital (CMH) was only 1 of 2 active hospitals that performed LDLT

since 2010. Eight years since its participation, CMH had been performed 45 LDLTs in pediatrics, with biliary atresia as the most common cause.⁹ However, there are no updated data about the effect of pretransplant infection on the posttransplant outcome. Thus, our study aimed to determine the role of pretransplant infections on the clinical course among pediatric liver transplantation recipients.

MATERIALS AND METHODS

A retrospective study on the medical records of post-LDLT recipients between April 2015 and May 2022 was conducted in 1 of the 2 transplantation centers in Indonesia, CMH. The patients' demographics, including distribution of age and gender, presence of pretransplant infection, and laboratory findings, were recorded. The primary outcome comprised of posttransplant infection occurring within the first month, the second to sixth month post-LDLT, and beyond the sixth month.^{4,10} In addition, the secondary outcome consisted of bacteremia, the LOS (PICU and in hospital), length of mechanical ventilation usage, number of days until the patients started enteral feeding, hospitalization cost, and graft rejection. The patients were divided into 2 groups according to the presence of infection before LDLT surgery. Pretransplant infection was defined as any infection occurring up to 3 mo before LDLT surgery that required hospitalization. Diagnosis of posttransplant infection was marked by the presence of fever, elevated leukocytes, procalcitonin, and CRP, as well as pathogen isolated from the culture of infection site after transplantation.

IBM SPSS, version 26.0, was used to perform the analysis. The normality of numerical data was assessed using the Kolmogorov-Smirnov test and presented as the mean \pm SD for data with normal distribution and the median (interquartile range) for abnormal distribution. Numerical data with normal distribution were analyzed using the unpaired *t* test, whereas abnormal distribution was analyzed with the Mann-Whitney test. The chi-square test and Fisher's exact test were used whenever appropriate. The study was approved by the Ethics Committee Faculty of Medicine, Universitas Indonesia Cipto Mangunkusumo Hospital, Jakarta, Indonesia, on May 2021 with approval number KET-497/UN2.F1/ETIK/PPM.00.02/2021. The institutional review board waived the need for written consent as long as we kept retrospective data confidential.

RESULTS

Among 56 patients who received LDLT between 2015 and 2022, 46 patients were diagnosed with biliary atresia (82.1%), and 9 underwent the Kasai procedure before LDLT. The common source of infection before LDLT based on the organ involved was gastrointestinal infection (35%), respiratory infection (29%), urinary infection (24%), and others (12%) (Figure 1). All patients with pretransplant infection received adequate therapy before the LDLT procedure.

The characteristics of patients, laboratory findings, and parameters denoting the clinical outcome in both groups are shown in Table 1. Pretransplant infection occurred in 15 patients (26.8%); 13 of 15 patients (86.7%) had posttransplant infections. Of 15 patients with pretransplant infection, 11 patients with bacterial infections had antibiotics.

Meanwhile, patients with viral or fungal infections did not get antibiotic treatment. There was no increase of antibiotic-resistant bacteria in patients with pretransplant infection compared with patients without pretransplant infection ($P = 0.488$). The median ages between patients with pretransplant infection and without pretransplant infection were 16.0 mo (10.0–33.0) and 16.5 mo (11.5–22.5), respectively. No significant differences in age ($P = 0.862$) and gender ($P = 0.129$) were noted between the 2 groups.

Based on the laboratory results, no significant difference in bilirubin levels was found between the recipients with pretransplant infection and without pretransplant infection ($P = 0.982$). The difference in ALT and AST levels between the 2 groups was also not statistically significant, with P values of 0.215 and 0.124, respectively. In addition, there was no significant difference in procalcitonin levels between patients with and without posttransplant infection (4.61 ug/dL [1.22–7.67] versus 2.34 ug/dL [0.35–7.01]; $P = 0.233$).

Recipients with pretransplant infection had a higher percentage of posttransplant infection within all 3 phases (86.7% versus 70.7%, $P = 0.307$; 66.7% versus 63.4%, $P = 1.000$; and 46.7% versus 39.0%, $P = 0.760$, respectively), as shown as in Table 2. However, no significant difference was found between the 2 groups.

The primary source of infection after the LDLT procedure in our study were respiratory infection (50%), urinary infection (28%), gastrointestinal infection (14%), and others (8%) (Figure 1). However, there was no effect of pretransplant pneumonia on posttransplant pneumonia ($r = 0.105$, $P = 0.427$). The most common pathogen isolated from respiratory infection was *Pseudomonas aeruginosa*, whereas *Escherichia coli* was frequently found in urinary tract infection and spontaneous bacterial peritonitis (Table 3).

In addition, the frequency of bacteremia was not significantly different between the 2 groups (26.7% versus 19.5%; $P = 0.715$) (Table 4). Of the 12 patients with posttransplant bacteremia, *Acinetobacter sp.* was the most frequently found microorganisms in blood culture, followed by *S. epidermidis*, *E. coli*, *Enterobacter sp.*, and *S. Maltophilia*.

The LOS in the hospital (40 d [33.0–78.0] versus 39 d [28.5–55.0]; $P = 0.345$) and PICU (20 d [13.0–26.0] versus 15 d [9.5–22.0]; $P = 0.287$) were higher in the group with pretransplant infection; however, the differences were not statistically significant. Furthermore, the length of mechanical ventilation and initiation of enteral feeding were similar between the 2 groups. In addition, there was no significant difference between the cost of hospitalization and graft rejection between the 2 groups ($P = 0.127$ and $P = 0.668$, respectively). In this study, 8 patients had rejection, 3 from the pretransplant infection group and 5 without the pretransplant infection group (20.0% versus 12.2%). The rejection incidence rate was 14.28% in total.

DISCUSSION

The primary etiology of liver transplants in our study was biliary atresia. This result is similar to the previous research, which reported that >50% of patients with biliary atresia required a liver transplant.^{9,11} The most common source of infection before liver transplant in this study was gastrointestinal infection, followed by respiratory infection and UTI. Moreover, previous studies demonstrated that the most

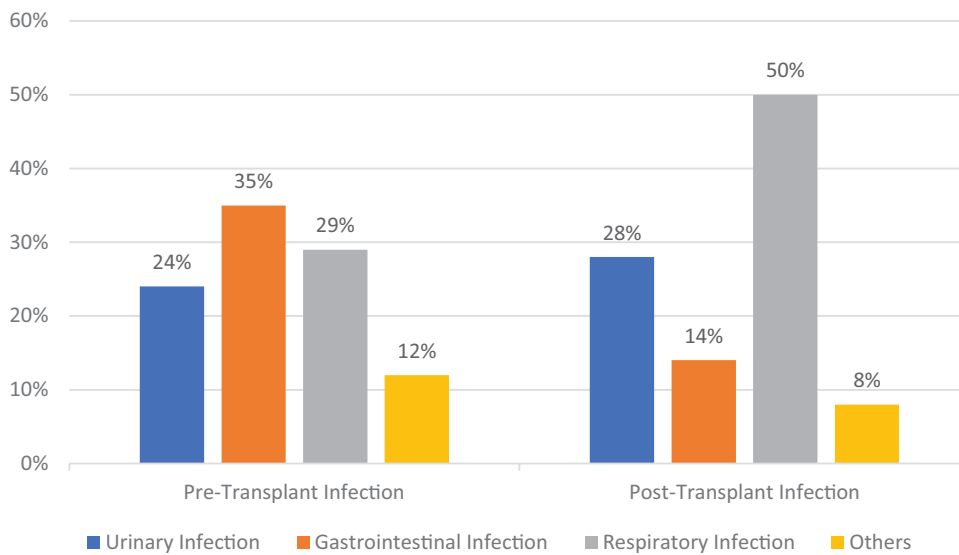


FIGURE 1. Source of infection before and after LDLT. Gastrointestinal infection was the major source of pretransplant infection. Furthermore, respiratory infection was the main source of posttransplant infection. LDLT, living donor liver transplantation.

TABLE 1.

Comparison of patient characteristics and laboratory findings between patients with and without pretransplant infection

Characteristics	Patients with pretransplant infection (n = 15)	Patients without pretransplant infection (n = 41)	P
Age (median, IQR), mo	16.0 (10.0–33.0)	16.5 (11.5–22.5)	0.862
Male, n (%)	11.0 (73.3)	19.0 (46.3)	0.129
Bilirubin (median, IQR), mg/dL	7.04 (5.90–22.10)	9.19 (4.97–20.07)	0.982
ALT (median, IQR), U/L	227.0 (131.5–332.5)	164.0 (74.0–302.0)	0.215
AST (median, IQR), U/L	341.50 (216.25–460.75)	233.00 (121.00–422.00)	0.124
Procalcitonin (median, IQR), µg/dL	4.61 (1.22–7.67)	2.34 (0.35–7.01)	0.233

Categorical data were analyzed using Fisher's exact test and presented as n (%). Numerical data were analyzed using the Mann-Whitney test and presented as median (IQR). ALT, alanine transaminase; AST, aspartate transaminase; IQR, interquartile range.

frequent pretransplant infections were UTI and chest infection.^{8,12,13} The difference between studies could be due to the possibility of 1 patient experiencing >1 infection before the LDLT procedure and the different exposure in their environment, causing the varying results.

The laboratory findings in this study showed no significant differences between the 2 groups. This might be because patients indicated for liver transplant often have high levels of liver markers and infection markers due to the excessive destruction of liver cells and other factors of inflammatory stimuli.^{14,15}

The incidence of posttransplant infection in our study was about 39% to 86.7%. However, contrary to other studies, the result reported lower posttransplant infection, around 24.6% to 82.4%.^{6–8} Our finding suggested that there was no significant difference in the presence of posttransplant infections between patients with and without pretransplant

TABLE 2.

Comparison of posttransplant infection in patients with and without pretransplant infection

Characteristics	Patients with pretransplant infection (n = 15)	Patients without pretransplant infection (n = 41)	P
Posttransplant infection (within 1st mo), n (%)	13.0 (86.7)	29.0 (70.7)	0.307
Posttransplant infection (2nd–6th mo), n (%)	10.0 (66.7)	26.0 (63.4)	1.000
Posttransplant infection (>6 mo–1 y), n (%)	7.0 (46.7)	16.0 (39.0)	0.760

Categorical data were analyzed using Fisher's exact test and presented as n (%).

TABLE 3.

Pathogen isolated from posttransplant infections within the first month

Infection site	n	Pathogen isolated
Respiratory infection	25	<i>Klebsiella pneumoniae</i> (10), <i>Acinetobacter baumannii</i> (8), <i>Acinetobacter lwoffii</i> (1), <i>Staphylococcus epidermidis</i> (2), <i>Escherichia coli</i> (2), <i>Enterobacter sp.</i> (2), <i>Streptococcus viridans</i> (7), <i>Staphylococcus aureus</i> (3), <i>Pseudomonas aeruginosa</i> (14), <i>Candida sp.</i> (5)
Urinary infection	14	<i>Klebsiella pneumoniae</i> (15), <i>Acinetobacter baumannii</i> (1), <i>Acinetobacter lwoffii</i> (1), <i>Staphylococcus epidermidis</i> (1), <i>Escherichia coli</i> (16), <i>Enterobacter sp.</i> (5), <i>Proteus vulgaris</i> (4), <i>Staphylococcus saprophyticus</i> (1), <i>Proteus mirabilis</i> (2)
Spontaneous bacterial peritonitis	2	<i>Escherichia coli</i> (2)
Unknown source	1	Not applicable

infection within the first month. This could be due to the adequate treatment received by the groups with the pretransplant infection before the transplantation.⁸ This finding differs from the previous study, which reported that patients with

TABLE 4.**Comparison of patient's outcome between patients with and without pretransplant infection**

Characteristic	Patients with pretransplant infection (n = 15)	Patients without pretransplant infection (n = 41)	P
Posttransplant bacteremia, n (%)	4.0 (26.7)	8.0 (19.5)	0.715
Length of stay in hospital (median, IQR), d	40.0 (33.0–78.0)	39.0 (28.5–55.0)	0.345
Length of stay in PICU (median, IQR), d	20.0 (13.0–26.0)	15.0 (9.5–22.0)	0.287
Length of mechanical ventilation (median, IQR), d	1 (1–4)	2 (1–5)	0.381
Initiation of enteral feeding (IQR), d	6.50 (5.25–7.00)	6.00 (5.00–8.00)	0.758
Hospitalization cost (median, IQR), rupiah	546 361 371.0 (383 070 186.0–885 482 325.0)	420 031 480.0 (352 284 524.0–586 463 622.5)	0.127
Graft rejection, n (%)	3.0 (20.0)	5 (12.2)	0.668

Categorical data were analyzed using Fisher's exact test and presented as n (%). Numerical data were analyzed using the Mann-Whitney test and presented as median (IQR). IQR, interquartile range.

pretransplant infection have a significantly higher incidence of posttransplant infection.⁷ The contrary findings might be caused by impaired immunity, frequent hospitalization, and translocation of bacteria from the intestine in patients with end-stage liver disease.^{16–19} In our study, no significant difference was found in the second and third phases post-LDLT between the 2 groups. Several factors predisposed to posttransplant infection in the second and third phases of our study consist of high disobedient antibiotic consumption, contamination of drinking water, uncooked meal, air pollution, lack of ventilation in the house, and lack of awareness in keeping a distance from unhealthy people or animals.

In our study, respiratory infection was the frequent cause of posttransplant infection. However, there was no association between pretransplant pneumonia and posttransplant pneumonia in this study. This result differs from a previous study, which identified bacterial peritonitis as the most common etiology of posttransplant infection.^{8,12} As we previously mentioned, a patient might have had >1 infection before LDLT, and infection posttransplant might have happened in a different organ because of various exposure. It may increase the possibility that patients who did not have pretransplant pneumonia developed posttransplant pneumonia. Several factors may promote the occurrence of respiratory infection in postoperative patients, such as the prolonged use of mechanical ventilation and surgical duration, as well as the history of malnutrition.^{20–22} Based on our study, the most frequent pathogen isolated from respiratory infection was gram-negative microbes such as *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and *Acinetobacter baumannii*. This finding is supported by another study that explained that gram-negative bacteria were the primary pathogen causing posttransplant infections, related to hospital-acquired pneumonia and ventilator-associated pneumonia.^{21,23}

Notably, our study found that gram-negative bacteria, particularly *Acinetobacter sp.*, were the leading cause of bacteremia. This result is in line with other studies.^{24–26} *Acinetobacter* is known to have a low virulence, but it may cause infection, mainly in immunocompromised patients, with the use of a central venous catheter, and with prolonged use of a mechanical ventilator.²⁷

We did not find any significant difference in LOS in the hospital and PICU between the 2 groups. This finding contradicts previous studies that reported that the length of hospital stay is significantly longer in patients with pretransplant infections.^{7,8} However, the extended stay may have been attributed to undergoing treatment for the pretransplant infection or the waiting period for transplant schedule.⁸ In addition, different

medical care habits between physicians, as well as the progression of patient condition and management between ICU and wards, may contribute to the increase of PICU LOS.²⁸ Similar to the LOS, we found that the hospitalization cost was not significantly different between the 2 groups.

Our findings showed that the length of mechanical ventilation did not significantly differ between the 2 groups, which is supported by another study.⁷ The median lengths of mechanical ventilation between the 2 groups were 1 versus 2 d. This is consistent with previous studies, which stated that, in stable patients, extubation could be conducted in 48 h to avoid respiratory complications.^{29,30}

The initiation of enteral feeding between the 2 groups in this study did not significantly differ. However, this result may have been attributed to the fact that nutrition is only 1 factor contributing to the patient's susceptibility toward infection. There are other variables, such as preoperative malnutrition and postoperative complications, that we did not link in this study, which might cause different results.¹⁹

Our findings showed no differences in graft rejection between the 2 groups, which is in line with the previous study.⁸ In 2014, Kim stated a higher incidence of posttransplant infection related to graft rejection.²¹ All patients in this study were given immunosuppression to prevent rejection, which may increase the risk of infection. Thus, clinicians enhanced immunosuppressive choices, dose monitoring, infection control, and attention to modifiable factors, such as pretransplant nutrition.³¹

STRENGTHS AND LIMITATIONS

This is the first study to address the relation between pretransplant infection and posttransplant outcomes in pediatric liver transplant recipients in Indonesia. Knowing the role of pretransplant infection in the posttransplant outcome allows for better judgment and management of pediatric liver transplant recipients. However, our study has limitations that need to be acknowledged. Our study design was observational and depended on the medical records. We cannot assume any association between pre- and posttransplant infection because of the lack of pretransplant cultured data. In addition, pretransplant infection was fully managed before transplantation. Only some of the patients were cultured because of empirical antibiotic responsiveness. Only those not responsive to the empirical antibiotic were considered to be cultured. The organ-specific pretransplant infection was not cultured because of the nature of this study and limited funding.

Furthermore, this study have no detailed data that can determine the precise period of infection in hospital-acquired or community-acquired infection and its correlation with immunosuppression. Thus, further prospective studies should be conducted to identify and confirm the pretransplant infection.

CONCLUSION

Our study found that there was no significant association between pretransplant infections that were treated adequately with the occurrence of posttransplant infections, presence of bacteremia LOS in hospital and PICU, length of mechanical ventilation, number of days until the patients were started on enteral feeding, hospitalization cost, and graft rejection. The best approach to reach an optimal outcome in LDLT is a prompt and adequate diagnosis and treatment before and after the LDLT procedure, including 3 critical times after LDLT.

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REFERENCES

1. Laici C, Gamberini L, Bardi T, et al. Early infections in the intensive care unit after liver transplantation-etiology and risk factors: a single-center experience. *Transpl Infect Dis*. 2018;20:e12834.
2. Hernandez Mdel P, Martin P, Simkins J. Infectious complications after liver transplantation. *Gastroenterol Hepatol (N Y)*. 2015;11:741–753.
3. Barbeta A, Butler C, Barhouma S, et al. Living donor versus deceased donor pediatric liver transplantation: a systematic review and meta-analysis. *Transplant Direct*. 2021;7:e767.
4. Fishman JA. Infection in solid-organ transplant recipients. *N Engl J Med*. 2007;357:2601–2614.
5. Sun HY, Cacciarelli TV, Singh N. Identifying a targeted population at high risk for infections after liver transplantation in the MELD era. *Clin Transplant*. 2011;25:420–425.
6. Kim YJ, Yoon JH, Kim SI, et al. Impact of pretransplant infections on clinical course in liver transplant recipients. *Transplant Proc*. 2018;50:1153–1156.
7. Heldman MR, Ngo S, Dorschner PB, et al. Pre- and post-transplant bacterial infections in liver transplant recipients. *Transpl Infect Dis*. 2019;21:e13152.
8. Lin KH, Liu JW, Chen CL, et al. Impacts of pretransplant infections on clinical outcomes of patients with acute-on-chronic liver failure who received living-donor liver transplantation. *PLoS One*. 2013;8:e72893.
9. Oswari H, Rahayatri TH, Soedibyo S. Pediatric living donor liver transplant in Indonesia's National Referral Hospital. *Transplantation*. 2020;104:1305–1307.
10. Fishman JA. Infection in organ transplantation. *Am J Transplant*. 2017;17:856–879.
11. Sasse M, Boehne M, Forstmeyer I, et al. Relevance of SIRS and sepsis in pediatric liver transplantation. *J Biosci Med*. 2021;9:131–145.
12. Saleh AM, Hassan EA, Gomaa AA, et al. Impact of pre-transplant infection management on the outcome of living-donor liver transplantation in Egypt. *Infect Drug Resist*. 2019;12:2277–2282.
13. Thévenot T, Bureau C, Oberti F, et al. Effect of albumin in cirrhotic patients with infection other than spontaneous bacterial peritonitis. A randomized trial. *J Hepatol*. 2015;62:822–830.
14. Sharma A, Nagalli S. *Chronic Liver Disease*. StatPearls [Internet]; StatPearls Publishing; 2021.
15. Jensen JU, Lundgren JD. Procalcitonin in liver transplant patients--yet another stone turned. *Crit Care*. 2008;12:108.
16. Fernández J, Acevedo J, Castro M, et al. Prevalence and risk factors of infections by multiresistant bacteria in cirrhosis: a prospective study. *Hepatology*. 2012;55:1551–1561.
17. Bonnel AR, Bunchorntavakul C, Reddy KR. Immune dysfunction and infections in patients with cirrhosis. *Clin Gastroenterol Hepatol*. 2011;9:727–738.
18. Chavez-Tapia NC, Torre-Delgado A, Tellez-Avila FI, et al. The molecular basis of susceptibility to infection in liver cirrhosis. *Curr Med Chem*. 2007;14:2954–2958.
19. Kim JM, Joh JW, Kim HJ, et al. Early enteral feeding after living donor liver transplantation prevents infectious complications: a prospective pilot study. *Medicine (Baltim)*. 2015;94:e1771.
20. Weiss E, Dahmani S, Bert F, et al. Early-onset pneumonia after liver transplantation: microbiological findings and therapeutic consequences. *Liver Transpl*. 2010;16:1178–1185.
21. Kim SI. Bacterial infection after liver transplantation. *World J Gastroenterol*. 2014;20:6211–6220.
22. Yuan H, Tuttle-Newhall JE, Chawa V, et al. Prognostic impact of mechanical ventilation after liver transplantation: a national database study. *Am J Surg*. 2014;208:582–590.
23. Shebl E, Gulick PG. *Nosocomial Pneumonia*. StatPearls [Internet]; StatPearls Publishing; 2021.
24. Singh N, Wagener MM, Obman A, et al. Bacteremias in liver transplant recipients: shift toward gram-negative bacteria as predominant pathogens. *Liver Transpl*. 2004;10:844–849.
25. Al-Hasan MN, Razonable RR, Eckel-Passow JE, et al. Incidence rate and outcome of Gram-negative bloodstream infection in solid organ transplant recipients. *Am J Transplant*. 2009;9:835–843.
26. Sganga G, Spanu T, Bianco G, et al. Bacterial bloodstream infections in liver transplantation: etiologic agents and antimicrobial susceptibility profiles. *Transplant Proc*. 2012;44:1973–1976.
27. Brady MF, Jamal Z, Pervin N. *Acinetobacter*. StatPearls [Internet]; StatPearls Publishing; 2017.
28. Pan ZY, Fan YC, Wang XQ, et al. Pediatric living donor liver transplantation decade progress in Shanghai: Characteristics and risks factors of mortality. *World J Gastroenterol*. 2020;26:1352–1364.
29. Tannuri U, Tannuri AC. Postoperative care in pediatric liver transplantation. *Clinics (Sao Paulo)*. 2014;69(Suppl 1):42–46.
30. Pudjadi AH. Intensive care management to reduce morbidities following pediatric liver transplantation in Indonesia. *Transplant Rep*. 2020;5:100064.
31. Shepherd RW, Turmelle Y, Nadler M, et al; SPLIT Research Group. Risk factors for rejection and infection in pediatric liver transplantation. *Am J Transplant*. 2008;8:396–403.