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# Clinical implications of prompt ascitic drain removal in cirrhosis with refractory ascites

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**INTRODUCTION** Large-volume paracentesis (LVP) is the first-line treatment for decompensated cirrhosis with refractory ascites. While ascitic drain removal (ADR) within 72 hours of the procedure was once considered safe, it was uncertain whether ADR within 24 hours could further reduce the risk of ascitic drain-related bacterial peritonitis (AdBP). This study aimed to investigate the association between the timing of ADR and the presence of AdBP.

**METHODS** All patients with cirrhosis with refractory ascites who underwent LVP in our institution from 2014 to 2017 were studied. AdBP was diagnosed based on an ascitic fluid neutrophil count ≥ 250 cells/mm$^3$ or positive ascitic fluid culture following recent paracentesis within two weeks.

**RESULTS** A total of 131 patients who underwent LVP were followed up for 1,806 patient-months. Their mean age was 68.3 ± 11.6 years, and 65.6% were male. Their mean Model for End-Stage Liver Disease score was 15.2. The overall incidence of AdBP was 5.3%. ADR beyond 24 hours was significantly associated with a longer median length of stay (five days vs. three days, p < 0.001), higher risk of AdBP (0% vs. 8.9%, p = 0.042) and acute kidney injury (AKI) following LVP (odds ratio 20.0, 95% confidence interval 2.4–164.2, p = 0.021). The overall survival was similar in patients who underwent ADR within and beyond 24 hours of LVP.

**CONCLUSION** ADR within 24 hours of LVP is associated with a reduced risk of AdBP and AKI. As AdBP is associated with resistant organisms and AKI, we recommend prompt ADR within 24 hours, especially in patients who have Child-Pugh class C alcoholic cirrhosis.

*Keywords: acute kidney injury, ascites, paracentesis, peritonitis*

## INTRODUCTION

Refractory ascites, a common complication among patients with decompensated cirrhosis, is associated with poor survival and significantly impaired quality of life from abdominal bloating, immobility, development of hernia and sarcopenia.[1] Refractory ascites is defined as ascites that cannot be mobilised, either owing to diuretic-induced complications (diuretic-intractable ascites) or a lack of response to diuretics (diuretic-resistant ascites).[2] While liver transplantation is curative, the high cost of organ transplantation and organ scarcity limits access for patients with refractory ascites. The majority of cirrhosis patients with refractory ascites are, thus, dependent on palliative options such as large-volume paracentesis (LVP) or transjugular intrahepatic portosystemic shunt for symptom relief. Current guidelines by the American Association for the Study of Liver Diseases and European Association for the Study of the Liver (EASL) recommend LVP as the first-line treatment for patients with refractory ascites.[2,3]

Although LVP is an effective treatment option for cirrhosis patients with refractory ascites and is associated with lower readmission risk when compared to diuretics,[4] complications such as paracentesis-induced circulatory dysfunction (PICD), acute kidney injury (AKI) and ascitic drain-related bacterial peritonitis (AdBP) can occur with LVP.[5-7] For instance, PICD can occur in up to 75% of cirrhosis patients undergoing LVP as a result of haemodynamic changes following LVP in the setting of excessive systemic arterial vasodilatation. PICD has also been associated with rapid re-accumulation of ascitic fluid, AKI and hepatorenal syndrome. The development of AKI was shown to adversely affect the outcome of cirrhosis patients, resulting in extended hospitalisation, increased inpatient stays and increased 90-day mortality. Current evidence indicates that intravenous albumin is effective in preventing PICD, especially when ascitic drainage greater than 5 L is performed.[6,8,9] A slower rate and small amount of ascitic drainage was also shown to reduce the risk of PICD and AKI.[10-13]

To prevent PICD and AKI following LVP, some physicians may allow slower ascitic drainage over a more extended period. While delayed ascitic drain removal (ADR) can theoretically increase the risk of AdBP, ADR within 72 hours was previously considered safe among patients with decompensated cirrhosis.[14,15] Kathpalia et al reported a higher risk of AdBP and poorer survival in patients who underwent ADR after 72 hours of LVP.[14] However, the literature on the association between the timing of ADR and the occurrence of AdBP remains limited. It is unclear whether ADR within 24 hours of LVP, when compared to ADR beyond 24 hours, could further reduce the risk of AdBP without increasing the risk of AKI or readmission rates. Hence, this study aimed to determine the association between the timing of ADR and AdBP as well as the clinical impact of AdBP among cirrhosis patients with refractory ascites. We hypothesised that ADR within 24

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hours of LVP could further reduce the risk of AdBP as compared to ADR beyond 24 hours.

## METHODS

This was a retrospective study conducted in Changi General Hospital, a 1,000-bed teaching hospital serving a population of 1.3 million from the eastern and northeast regions of Singapore. All hospitalised patients with decompensated cirrhosis who had undergone LVP in Changi General Hospital from January 2014 to December 2017 were included.

Patients were first identified from electronic medical records using the procedure code for paracentesis and ICD-9 (International Classification of Diseases, ninth revision) codes for liver cirrhosis and ascites. To ensure the accuracy and consistency of our data, two authors (WYJ and LHM) separately reviewed the electronic medical records of all patients as well as the relevant data on patients' baseline characteristics, laboratory results, clinical outcomes, and duration and complications of LVP. Cases were censored at death, liver transplant or last follow-up. A standard dose of albumin infusion was administered during LVP, as per EASL guidelines. Our institutional review board approved the study protocol.

All adults (age ≥ 21 years) who were diagnosed with liver cirrhosis and underwent paracentesis were consecutively included. The exclusion criteria were: (a) patients with spontaneous bacterial peritonitis (SBP) diagnosed upon admission or during insertion of a peritoneal drain; (b) patients with ascites secondary to malignancy, pancreatitis, nephrotic syndrome, non-cirrhotic portal hypertension and infection, including tuberculous peritonitis; and (c) patients who had undergone a diagnostic abdominal tap. After excluding patients with missing data, we identified a total of 131 patients with refractory ascites who had undergone LVP for analysis.

Cirrhosis was defined based on clinical, biochemical, radiological or histological findings. Non-alcoholic steatohepatitis (NASH) was defined as cirrhosis with metabolic syndrome after exclusion of other liver diseases such as chronic viral hepatitis, autoimmune liver disease and Wilson's disease. LVP was defined as ascitic drainage to relieve symptoms arising from refractory ascites.[16] AdBP was diagnosed based on ascitic fluid analysis, especially a neutrophil count ≥ 250 cells/mm$^3$ or positive ascitic fluid culture following recent paracentesis within two weeks. AKI was defined as an increase in serum creatinine of > 26.5 μmol/L from baseline within 48 hours following LVP.[2] For the diagnosis of alcoholic liver cirrhosis, clinically significant alcohol intake was defined as alcohol consumption of > 20 g/day for women and > 30 g/day for men.[17]

Statistical analysis was conducted using IBM SPSS Statistics version 22.0 (IBM Corp, Armonk, NY, USA). Descriptive statistics were computed for all variables. Categorical variables were expressed as frequencies and percentages, while mean and standard deviation were expressed as continuous variables. Categorical variables were analysed using chi-square or Fisher's exact test. Continuous variables were analysed using Student's *t*-test. All p-values quoted were two-sided, and $p < 0.05$ was considered statistically significant. ADR within 24 hours was the dependant variable. Binary logistic regression was performed by adjusting for covariates that were clinically relevant to AdBP. Cox proportional hazards regression was used for mortality analysis.

## RESULTS

The analysis included 131 patients with cirrhosis who had undergone LVP. The mean age of the patients was 68.3 ± 11.6 years. The mean Model for End-Stage Liver Disease (MELD) score was 15.2 ± 5.3 years. The commonest cause of underlying liver cirrhosis was NASH (29.0%), followed by alcohol (26.0%), cryptogenic (19.8%) and chronic hepatitis C (16.0%). A total of 17 (13.0%) patients received antibiotics for secondary prophylaxis while undergoing LVP. The median duration of ADR was 2 (interquartile range 2–10) days, and 65.6% of the patients underwent ADR within two days.

The baseline demographics of patients who underwent ADR within and beyond 24 hours of LVP are summarised in Table I. Both patient groups were similar in terms of age, gender, underlying aetiology of cirrhosis, severity of liver cirrhosis, biochemistry and the proportion of patients receiving antibiotic prophylaxis for SBP. ADR beyond 24 hours was associated with a higher risk of AdBP (0% vs. 8.9%, p = 0.042) and AKI (1.9% vs. 29.1%, p < 0.001). The amount of intravenous albumin administered was similar between patients who had undergone ADR within and beyond 24 hours of LVP (80 g vs. 70 g, p = 0.184).

ADR beyond 24 hours of LVP was associated with a higher risk of AKI (odds ratio [OR] 20.0, 95% confidence interval [CI] 2.4–164.2; p = 0.005) after adjusting for MELD score, Child-Pugh score, AdBP and alcoholic liver cirrhosis (Table II). ADR beyond 24 hours was also associated with a longer median length of stay (OR 1.3, 95% CI 1.1–1.5; p < 0.001). Unscheduled 30-day readmissions were similar in both groups (48.1% vs 43.0%, p = 0.595). Overall survival was similar in patients who underwent ADR within and beyond 24 hours of LVP after adjusting for MELD score, AKI, Child-Pugh score and the timing of ADR (hazard ratio [HR] 3.0, 95% CI 0–6.3 months vs. HR 5.0, 95% CI 2.8–7.2 months; p = 0.335).

The overall incidence of AdBP in our cohort was 5.3% (7/131). The baseline characteristics of patients with and without AdBP are summarised in Table III. The AdBP rate in patients who underwent ADR within 24 hours and 48 hours of LVP was 0%, while that in patients who underwent ADR within 72 hours was 4.3%. The rate of AdBP was significantly lower in patients who underwent ADR within 24 hours (0% vs. 8.9%, p = 0.042) and within 48 hours (0% vs. 15.6%, p < 0.001) of LVP (Fig. 1). Patients who developed AdBP had higher MELD scores (17.1 ± 1.8 vs. 15.1 ± 5.4, p = 0.028) and were more likely to have Child-Pugh Class C cirrhosis (100.0% vs. 42.7%, p = 0.003). As all cases of AdBP occurred in patients with Child-Pugh Class C cirrhosis, we performed a subgroup analysis of this group and found that the rate of AdBP remained significantly lower in patients who underwent ADR within 24 hours (0% vs. 17.9%, p = 0.040) and 48 hours (0% vs. 28.0%, p < 0.001) of LVP (Fig. 2).

**Table I. Baseline demographics of patients with decompensated cirrhosis who underwent ascitic drain removal (ADR) within and beyond 24 hours of large-volume paracentesis.**

| Characteristic | No. (%)/mean ± SD | | | p-value |
|---|---|---|---|---|
| | Total (n = 131) | ADR ≤ 24 hr (n = 52) | ADR > 24 hr (n = 79) | |
| **Age (yr)** | 68.3 ± 11.6 | 69.4 ± 11.8 | 67.4 ± 11.5 | 0.359 |
| **Gender** | | | | 0.251 |
| Male | 86 (65.6) | 31 (59.6) | 55 (69.6) | |
| Female | 45 (34.4) | 21 (40.4) | 24 (30.4) | |
| **Aetiology** | | | | 0.386 |
| NASH | 38 (29.0) | 15 (28.8) | 23 (29.1) | |
| Alcohol | 34 (26.0) | 14 (26.9) | 20 (25.3) | |
| Hepatitis B | 8 (6.1) | 2 (3.8) | 6 (7.6) | |
| Hepatitis C | 21 (16.0) | 9 (17.3) | 12 (15.2) | |
| Cryptogenic | 26 (19.8) | 9 (17.3) | 17 (21.5) | |
| Cardiac | 1 (0.8) | 0 (0) | 1 (1.3) | |
| PBC | 3 (2.3) | 3 (5.8) | 0 (0) | |
| **Serum albumin (g/L)** | 26 ± 5 | 25 ± 6 | 26 ± 5 | 0.559 |
| **Serum bilirubin (μmol/L)** | 51.5 ± 72.7 | 45.1 ± 65.2 | 55.7 ± 77.3 | 0.129 |
| **Serum creatinine (mmol/L)** | 158 ± 135 | 164 ± 140 | 144 ± 113 | 0.383 |
| **Platelet (× $10^9$)** | 185 ± 225 | 169 ± 159 | 195 ± 260 | 0.516 |
| **INR** | 1.20 ± 0.24 | 1.1 ± 0.2 | 1.2 ± 0.3 | 0.331 |
| **Child-Pugh class** | 9.5 ± 1.2 | 9.4 ± 1.2 | 9.6 ± 1.1 | 0.518 |
| B | 71 (54.2) | 31 (59.6) | 40 (50.6) | 0.313 |
| C | 60 (45.8) | 21 (40.4) | 39 (49.4) | |
| **MELD score** | 15.2 ± 5.3 | 14.5 ± 4.1 | 15.7 ± 5.9 | 0.199 |
| **SBP prophylaxis** | 17 (13.0) | 4 (7.7) | 13 (16.5) | 0.144 |
| **Albumin administered (g)** | 74 ± 42 | 80 ± 48 | 70 ± 37 | 0.184 |
| **Ascitic fluid drained (L)** | 7.9 ± 2.9 | 7.6 ± 2.5 | 8.1 ± 3.1 | 0.366 |
| **Median LOS* (day)** | 4 (2–10) | 3 (2–5) | 5 (3–12) | < 0.001‡ |
| **Complication** | | | | |
| AdBP | 7 (5.3) | 0 (0) | 7 (8.9) | 0.042‡ |
| AKI | 24 (18.3) | 1 (1.9) | 23 (29.1) | < 0.001‡ |
| 30-day readmission rate | 59 (45.0) | 25 (48.1) | 34 (43.0) | 0.595 |
| Overall survival† (mth) | 5 (2.7–7.3) | 3 (0.0–6.3) | 5 (2.8–7.2) | 0.876 |

*Data presented as median (interquartile range) and †hazard ratio (95% confidence interval). ‡p < 0.05 considered statistically significant. AdBP: ascitic drain-related bacterial peritonitis; AKI: acute kidney injury; INR: international normalised ratio; LOS: length of stay; NASH: non-alcoholic steatohepatitis; PBC: primary biliary cholangitis; SBP: spontaneous bacterial peritonitis; SD: standard deviation

**Table II. Association between clinical outcomes and ascitic drain removal (ADR) within and beyond 24 hours of large-volume paracentesis.**

| Covariate | No. (%)/mean ± SD | | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|---|---|
| | ADR ≤ 24 hr (n = 52) | ADR > 24 hr (n = 79) | Unadjusted OR (95% CI) | p-value | Adjusted OR (95% CI)* | p-value |
| Secondary bacterial peritonitis | 0 (0) | 7 (8.9) | ND | 0.042 | ND | ND |
| Acute kidney injury | 1 (1.9) | 23 (29.1) | 20.9 (2.7–60.7) | < 0.001† | 20.0 (2.4–164.2) | 0.005† |
| MELD score | 14.5 ± 4.1 | 15.7 ± 5.9 | 1.05 (1.0–1.1) | 0.199 | 0.99 (0.9–1.1) | 0.818 |
| Child-Pugh score | 9.4 ± 1.2 | 9.6 ± 1.1 | 1.11 (0.8–1.5) | 0.518 | 0.99 (0.7–1.4) | 0.951 |
| Alcoholic cirrhosis | 14 (26.9) | 20 (25.3) | 0.9 (0.4–2.0) | 0.837 | 1.4 (0.5–3.3) | 0.521 |

*Multivariate analysis (logistic regression or Cox regression as appropriate) was performed with adjustment for MELD score, secondary bacterial peritonitis, Child-Pugh score, acute kidney injury and alcoholic cirrhosis (vs. non-alcoholic cirrhosis), with ADR ≤ 24 hr as the reference group. †p < 0.05 is considered statistically significant. CI: confidence interval; MELD: Model for End-Stage Liver Disease; ND: analysis could not be done, as no cases of secondary bacterial peritonitis were observed in the ADR ≤ 24 hr group; OR: odds ratio; SD: standard deviation

**Table III. Baseline demographics of patients with and without ascitic drain-related bacterial peritonitis (n = 131).**

| Characteristic | No. (%)/mean ± SD | | p-value |
|---|---|---|---|
| | AdBP (n = 7) | No AdBP (n = 124) | |
| **Age (yr)** | 71.6 ± 7.3 | 68.1 ± 11.8 | 0.270 |
| **Male gender** | 6 (85.7) | 80 (64.5) | 0.251 |
| **Aetiology** | | | 0.719 |
| Alcoholic | 2 (28.6) | 36 (29.0) | |
| NASH | 3 (42.9) | 31 (25.0) | |
| Hepatitis B | 0 (0) | 8 (6.5) | |
| Hepatitis C | 2 (28.6) | 19 (15.3) | |
| Cryptogenic | 0 (0) | 26 (21.0) | |
| Cardiac | 0 (0) | 1 (0.8) | |
| PBC | 0 (0) | 3 (2.4) | |
| **Timing of ADR** | 78.9 ± 11.7 | 53.8 ± 53.8 | 0.045‡ |
| ≤ 24 hr (vs. > 24 hr) | 0 (0) | 52 (100.0) | 0.026† |
| ≤ 48 hr (vs. > 48 hr) | 0 (0) | 86 (100.0) | < 0.001† |
| ≤ 72 hr (vs. > 72 hr) | 5 (4.3) | 112 (95.7) | 0.163 |
| **Albumin (g/L)** | 22 ± 4 | 26 ± 5 | 0.075 |
| **Bilirubin (mmol/L)** | 55.5 ± 26.7 | 51.3 ± 74.5 | 0.882 |
| **Creatinine (mmol/L)** | 140 ± 22 | 160 ± 139 | 0.443 |
| **Platelet (× $10^9$)** | 140 ± 64 | 187 ± 231 | 0.592 |
| **International normalised ratio** | 1.23 ± 0.16 | 1.19 ± 0.24 | 0.693 |
| **Child-Pugh score** | 10.3 ± 0.8 | 9.5 ± 1.2 | 0.065 |
| **Child-Pugh class** | | | 0.003† |
| B | 0 (0) | 71 (57.3) | |
| C | 7 (100.0) | 53 (42.7) | |
| **MELD score** | 17.1 ± 1.8 | 15.1 ± 5.4 | 0.028† |
| **Albumin given (g)** | 74 ± 56 | 74 ± 41 | 0.986 |
| **SBP prophylaxis** | 2 (28.6) | 15 (12.1) | 0.207 |
| **Complication** | | | |
| AdBP | 7 (100.0) | 0 (0) | NA |
| 30-day readmission rate | 4 (57.1) | 55 (44.4) | 0.700 |
| AKI | 4 (57.1) | 20 (16.1) | 0.021† |
| Median LOS* (day) | 21 (4–21) | 4 (2–8) | 0.313 |
| Median survival* (mth) | 2 (0–14.0) | 2 (0–53.0) | 0.443 |

*Data presented as median (interquartile range). †p < 0.05 considered statistically significant. AdBP: ascitic drain-related bacterial peritonitis; ADR: ascitic drain removal; AKI: acute kidney injury; INR: international normalised ratio; LOS: length of stay; MELD: Model of End-Stage Liver Disease; NA: not applicable; NASH: non-alcoholic steatohepatitis; PBC: primary biliary cirrhosis; SBP: spontaneous bacterial peritonitis; SD: standard deviation

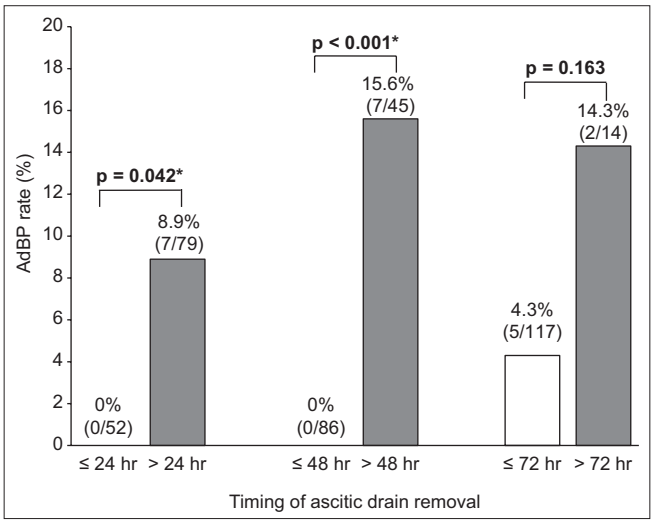


**Fig. 1** Chart shows the rates of ascitic drain-related bacterial peritonitis and the timing of ascitic drain removal in all patients. *p < 0.05 is considered statistically significant. AdBP: ascitic drain-related bacterial peritonitis

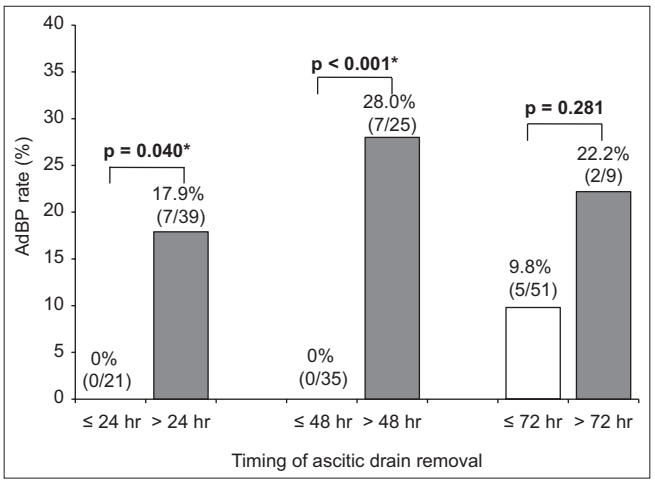


**Fig. 2** Chart shows the rates of ascitic drain-related bacterial peritonitis and the timing of ascitic drain removal in patients with Child-Pugh class C cirrhosis. AdBP: ascitic drain-related bacterial peritonitis

AdBP was also associated with a higher risk of AKI following LVP (57.1% vs. 16.1%, p = 0.021) (Fig. 3). Although AKI was independently associated with the timing of ADR and AdBP, AKI during LVP was not associated with the total volume of ascitic drainage (7.8 L vs. 7.9 L, p = 0.845) or the amount of intravenous albumin infusion administered (66 g vs. 76 g, p = 0.279). The average length of stay (15.8 days vs. 7.3 days, p = 0.149) and 30-day readmission rate (45.8% vs. 44.9%, p = 0.931) were also similar in patients with and without AKI. Almost all (95.8%) of the 24 patients with AKI had Grade 1 AKI, except for one patient who had Grade 3 AKI. The median length of stay and unscheduled 30-day readmission rates were similar between the AdBP and non-AdBP groups (57.1% vs. 44.4%, p = 0.700). Overall survival was similar in patients with and without AdBP after adjusting for MELD score, AKI, Child-Pugh score, alcoholic cirrhosis and timing of ADR (p = 0.972). Notably, patients with alcoholic cirrhosis had a two-fold higher risk of mortality after adjusting for MELD score, AdBP, Child-Pugh score, AKI and timing of ADR (HR 2.2, 95% CI 1.4–3.5; p = 0.002).

Among all patients with AdBP, 71.4% (5/7) had resistant organisms from ascitic fluid cultures (Extended-spectrum beta-lactamases-producing *Escherichia coli*: n = 3, Methicillin-resistant

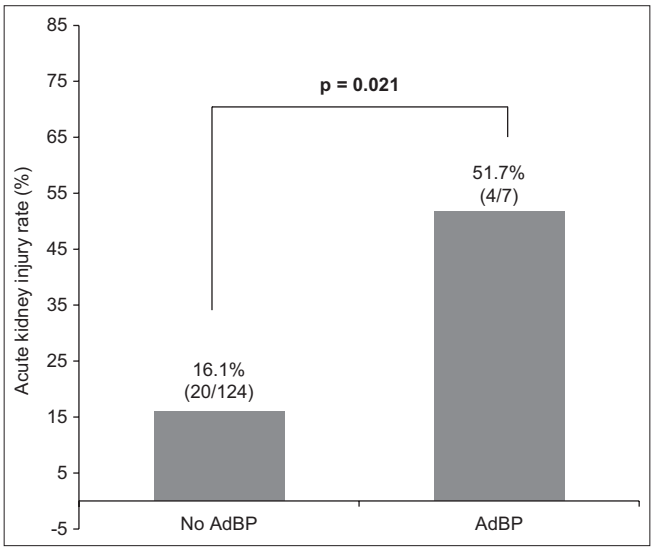


**Fig. 3** Chart shows the rates of acute kidney injury with and without ascitic drain-related bacterial peritonitis. AdBP: ascitic drain-related bacterial peritonitis

*Staphylococcus aureus*: n = 2, *Bacteroides ovatus*: n = 2). The proportion of patients receiving SBP prophylaxis was similar among patients with and without AdBP (28.6% vs. 12.2%, p = 0.207).

## DISCUSSION

This study demonstrates that ADR beyond 24 hours of LVP increases the risk of AdBP and AKI in patients undergoing LVP. While none of the patients who underwent ADR within 24 hours had AdBP, ADR beyond 24 hours and 48 hours was associated with a significantly higher risk of AdBP (8.9% and 15.6%, respectively) (Fig. 1). Previous literature on the association between the timing of ADR and the presence of AdBP is limited. In practice, some physicians may choose to delay the timing of ADR for up to 48 hours,[18] either to minimise PICD and AKI or to prioritise complete ascitic drainage over timing of ADR in hopes of reducing readmissions in patients with refractory ascites. However, there is no substantial evidence that such practice is beneficial to patient care. While it is unlikely that a prospective randomised study will be conducted to address this clinical dilemma owing to ethical reasons, the present study highlights that ADR beyond 24 hours of LVP significantly increases the risk of AdBP, with a concomitant 20-fold higher risk of AKI. As the majority of patients had early-stage AKI that was reversible with intravenous albumin infusion, no significant difference in mortality was observed between patients with and without AdBP.

AdBP has important clinical implications among patients undergoing LVP. AdBP is not only preventable but is also associated with a higher risk of AKI and prolonged hospitalisation, particularly among cirrhosis patients with higher MELD scores. Prolonged hospitalisation, in turn, increases the direct healthcare costs for patients and predisposes them to a higher risk of nosocomial infections by multidrug-resistant organisms (MDROs).[19] The emergence of MDROs in AdBP significantly impacts the survival of hospitalised cirrhosis patients and is associated with a higher risk of acute-on-chronic liver failure (ACLF). It is now clear that ACLF carries a high 90-day mortality of 50.4%–56.1% among hospitalised patients with decompensated cirrhosis.[17,20] As a palliative procedure, the goal of LVP should be symptom relief rather than complete drainage of ascites. Our data supports prompt ADR within 24 hours to mitigate the risk of AdBP and AKI from LVP.

Despite a higher risk of AdBP and AKI, overall survival was similar between patients who underwent ADR within 24 hours and beyond 24 hours of LVP, after adjusting for various clinically relevant confounding factors. Meanwhile, patients with alcoholic cirrhosis had a two-fold higher risk of mortality as compared to patients with non-alcoholic cirrhosis (HR 2.2, 95% CI 1.4–3.5, p = 0.002). The association between alcoholic cirrhosis and a higher incidence of ascites and SBP has been reported in previous studies.[21,22] Postulated mechanisms of these findings included increased gut permeability, impaired immunity and gut dysbiosis.[23-25] Alcohol abstinence may improve liver function, thus potentially improving transplant-free survival among alcoholic cirrhosis patients with ascites.[26] Therefore, physicians should be vigilant in performing prompt ADR among alcoholic cirrhosis patients who require LVP.

We acknowledge that this study must be interpreted within its limitations. Firstly, our study had a retrospective and non-randomised design. Owing to the small number of events, logistic regression analysis to assess the OR of AdBP could not be conducted. The small number of events also precluded multivariate analysis for confounders of AdBP. As all the events were observed in patients with Child-Pugh Class C cirrhosis, it is conceivable that the severity of liver disease is a significant contributor to the development of AdBP. A subgroup analysis within patients with Child-Pugh Class C cirrhosis showed that ADR within 24 hours of LVP remained significantly associated with AdBP (Fig. 2). Secondly, ascitic fluid cell count was measured only in patients who had symptomatic bacterial peritonitis, which may potentially underestimate the prevalence of AdBP. Lastly, data on comorbidities such as diabetes mellitus, hypertension and medication history was not available in this study. Future studies evaluating AKI following LVP should account for these factors.

The results of this study should be widely applicable, because we included all patients with decompensated liver cirrhosis with refractory ascites undergoing LVP, regardless of the severity of the liver cirrhosis and the underlying aetiology. The baseline characteristics were also comparable between the patients who underwent ADR within and beyond 24 hours of LVP. Further, ADR beyond 24 hours of LVP was significantly associated with a higher risk of AdBP and AKI, even after adjusting for clinically significant confounding factors.

In conclusion, ADR beyond 24 hours of LVP increases the risk of AKI and AdBP, and is linked to a significant risk of AdBP. As AdBP is associated with more resistant organisms and a higher risk of AKI, prompt ADR within 24 hours is recommended, especially among patients with alcoholic cirrhosis with Child-Pugh Class C or a higher MELD score. While it is unlikely that a prospective study will be performed, these findings should serve as a reminder to clinicians that leaving a drain for longer durations increases the risk of AdBP and that there is no merit in protracted ascitic drainage.

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