

Original Research Article

ARTERIAL BLOOD LACTATE LEVEL AS A MORTALITY MARKER IN POST OPERATIVE PATIENTS WITH HOLLOW VISCUS PERFORATION

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ABSTRACT

Background: Serum lactate has been utilized for numerous years to evaluate perfusion status and as an indicator of disease severity. Perforation of a hollow viscus is a critical, life-threatening illness associated with significant morbidity and death due to multiple organ failure and subsequent sepsis. In the clinical context, assessing the severity of hollow viscus perforation in patients, which may result in sepsis, and identifying prognostic signs is essential to mitigate the elevated death risk. The aim is to elucidate that postoperative arterial blood lactate level is a predictor of mortality in patients with hollow viscus perforation. **Materials and Methods:** This was a prospective cohort study conducted from September 2022 to June 2024 among patients undergoing surgery for hollow viscus perforation at Ramkrishna Care Hospital, Raipur. The above-mentioned patients were divided into mortality and survivor groups, and mortality was assessed at 48 hours, 7 days, and 28 days.

Results: Arterial blood lactate level of 2.0 mmol/L or more was found in 30.8% of the study population. The mortality rate in patients with lactate >2 mmol/L was 71.4% as compared to 10.8% in those with lactate ≤ 2 mmol/L, and the difference was found to be highly statistically significant ($p < 0.001$). In our study, sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) for this test were 83.33% (95% CI: 62.62%–95.26%), 80.49% (95% CI: 65.13%–91.18%), 71.43% (95% CI: 56.69%–82.68%), and 89.19% (95% CI: 76.91%–95.34%), respectively. The results of univariate analysis showed that the APACHE-II and SOFA scores were significantly higher and eTIME was significantly longer in the mortality group than in the survivor group. The postoperative LAC level was significantly higher in the mortality group (5.35 ± 3.07 mmol/L) than in the survivor group (1.26 ± 0.93 mmol/L; $p < 0.001$), and the preoperative WBC count was significantly lower in the mortality group than in the survivor group. Multivariate logistic regression analysis using the mortality risk factors determined by univariate analysis, including eTIME, APACHE-II score, SOFA score, preoperative WBC count, and postoperative LAC, demonstrated that postoperative LAC level was an independent risk factor for mortality.

Conclusion: High postoperative lactate level is a useful factor for independently predicting high mortality rates in patients with hollow viscus perforation.

Keywords: Hollow viscus perforation, Arterial blood lactate, Prognosis, Sepsis.

INTRODUCTION

Hollow viscus perforation is a major surgical emergency characterized by disruption of the gastrointestinal tract with leakage of luminal contents into the peritoneal cavity. Perforation may involve

the stomach, duodenum, small intestine, appendix, colon, or other portions of the gastrointestinal tract. Once contamination occurs, patients may rapidly develop diffuse peritonitis, systemic inflammatory response, sepsis, circulatory instability, and multiple-organ dysfunction. Emergency surgical source

control, adequate resuscitation, broad-spectrum antimicrobial therapy, and postoperative monitoring therefore form the basis of management. Despite advances in diagnostic imaging, anesthesia, operative techniques, antimicrobial treatment, and critical care, postoperative morbidity and mortality remain important concerns after surgery for gastrointestinal perforation.^[1] The clinical course after hollow viscus perforation is influenced by several patient- and disease-related factors. Age, comorbid illness, anatomical site of perforation, extent of contamination, duration of symptoms, shock at presentation, hematological abnormalities, renal dysfunction, nutritional status, and severity of systemic inflammation may contribute to postoperative outcome.^[2] Delay between perforation and definitive source control may permit continued contamination and bacterial translocation, intensifying peritoneal inflammation and physiological derangement. Patients may also reach surgery with dehydration, electrolyte imbalance, acidosis, tissue hypoperfusion, or established sepsis. Accurate early risk assessment is therefore important for identifying patients who require aggressive resuscitation, closer postoperative observation, and timely organ support. Perforated peptic ulcer disease remains an important component of hollow viscus perforation requiring emergency laparotomy. Although the spectrum of perforation varies with geographic region, etiology, age, and access to healthcare, gastroduodenal perforations remain frequently encountered in emergency surgical practice.^[3] Other causes include appendicular, ileal, colonic, malignant, inflammatory, ischemic, traumatic, and iatrogenic perforations. The postoperative course is not determined by the anatomical defect alone; physiological reserve, degree of peritoneal contamination, timing of intervention, and response to resuscitation also influence recovery. Recent clinical research has therefore focused increasingly on objective perioperative markers that can supplement conventional assessment and assist postoperative risk stratification.^[4] Several scoring systems are used for prognostication in critically ill surgical patients, including the Acute Physiology and Chronic Health Evaluation II and Sequential Organ Failure Assessment scores. These integrate physiological and laboratory variables to estimate illness severity, but require multiple parameters and may change during the perioperative period. In emergency surgery, a rapidly available biochemical marker reflecting global physiological stress may offer additional practical value. Blood lactate is particularly attractive because it can be obtained quickly from arterial blood gas analysis and measured repeatedly during resuscitation and postoperative care. Hyperlactatemia has consequently become an important parameter in assessment of critically ill patients and patients with secondary peritonitis.^[5] Circulating lactate reflects the balance between its production and clearance. In severe intra-abdominal

infection, elevated lactate may occur with impaired tissue perfusion, increased metabolic demand, adrenergic stimulation, microcirculatory dysfunction, altered oxygen utilization, or reduced clearance. Lactate may therefore provide information not completely captured by routine bedside hemodynamic measurements. In the perioperative setting, lactate can be measured before, during, and after surgery, allowing assessment of physiological response across different stages of treatment. Studies in emergency gastrointestinal surgery have examined perioperative lactate as a practical prognostic biomarker.^[6]

MATERIALS AND METHODS

This prospective observational cohort study was conducted in the Department of Critical Care, Ramkrishna Care Hospital, Raipur, Chhattisgarh, from September 2022 to June 2024 after obtaining approval from the Institutional Scientific Committee and Institutional Ethics Committee. The study included adult patients aged >18 years who underwent elective or emergency surgery for hollow viscus perforation in the Department of Surgery. Patients with coronary artery disease, diabetic ketoacidosis, diabetes mellitus receiving metformin therapy, liver failure, corrosive ingestion, or intraoperative cardiac arrest were excluded from the study. Written informed consent was obtained from the patients or their legally authorized representatives before enrolment, wherever applicable.

Sample Size and Study Groups: The sample size was calculated on the basis of the postoperative mortality rate of 22.00% obtained from previous hospital records. Considering the expected mortality proportion and an absolute precision of 10%, the minimum sample size was estimated using the Cochran formula. Accordingly, a total of 65 patients fulfilling the eligibility criteria were included in the study. Based on survival status at the end of 28 days, the enrolled patients were categorized into two groups. Group M included patients who did not survive up to 28 days following surgery, whereas Group S included patients who survived at the end of the 28-day follow-up period. Mortality occurring within 48 hours and at the end of 7 days was also recorded.

Clinical Assessment and Study Procedure: After enrolment, demographic and clinical information including age, sex, site of hollow viscus perforation, and elapsed time to surgery (eTIME) was recorded. The elapsed time to surgery was defined as the interval between the onset of clinical symptoms and signs suggestive of perforation, particularly guarding and rigidity, and the commencement of surgical intervention. Postoperatively, hemodynamic and clinical parameters were recorded one hour after extubation. These included oral temperature, heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, respiratory rate, peripheral

oxygen saturation (SpO₂), and Glasgow Coma Scale score. Laboratory parameters including total white blood cell count, platelet count, hematocrit, serum creatinine, serum bilirubin, serum sodium, and serum potassium were also recorded. Acute Physiology and Chronic Health Evaluation II (APACHE II), Sequential Organ Failure Assessment (SOFA), and Systemic Inflammatory Response Syndrome (SIRS) criteria were assessed during the postoperative period. For calculation of the APACHE II score, the worst value of each physiological parameter recorded during the first 24 postoperative hours was considered.

Arterial Blood Gas and Lactate Measurement: An arterial blood sample was collected immediately after completion of surgery using a sterile heparinized 2-mL syringe under aseptic precautions. The sample was analyzed using a Radiometer ABL80 FLEX blood gas analyzer. Arterial blood gas parameters including pH, partial pressure of arterial oxygen (PaO₂), partial pressure of arterial carbon dioxide (PaCO₂), bicarbonate (HCO₃⁻), and arterial blood lactate level were measured. Postoperative arterial lactate was considered the principal study variable for evaluating its association with mortality. The clinical, biochemical, arterial blood gas, APACHE II, SOFA, and SIRS parameters were subsequently compared between the mortality and survivor groups. The primary outcome was mortality at 28 days, while mortality within 48 hours and at 7 days was assessed as additional outcome measures.

Statistical Analysis: The collected data were entered into a master chart and analyzed using appropriate statistical methods. Continuous variables were expressed as mean $\pm$ standard deviation, whereas categorical variables were presented as frequencies and percentages. The unpaired Student's t-test was used to compare normally distributed continuous variables between the survivor and mortality groups, while categorical variables were compared using the Chi-square test or Fisher's exact test, wherever appropriate. Univariate analysis was performed to identify variables associated with postoperative mortality. Variables found to be significantly associated with mortality on univariate analysis were subsequently entered into a multivariate logistic regression model to determine independent predictors of mortality. Receiver operating characteristic (ROC) curve analysis was performed to assess the ability of postoperative arterial blood lactate levels to predict mortality and to determine an appropriate cut-off value. The area under the ROC curve (AUC), sensitivity, and specificity were determined. A p-value <0.05 was considered statistically significant, while a p-value <0.001 was considered highly significant.

RESULTS

[Table 1] Demographic Characteristics and Distribution of Study Participants

A total of 65 patients with hollow viscus perforation were included in the study. Of these, 24 (36.92%) patients belonged to Group M, comprising patients who did not survive at the end of 28 days, whereas 41 (63.08%) patients belonged to Group S, comprising patients who survived at the end of 28 days. The mean age of patients in Group M was 51.0 ± 14.05 years, compared with 48.0 ± 9.41 years in Group S. The difference in mean age between the two groups was not statistically significant ($p=0.8816$). Among the 65 study participants, 53 were males and 12 were females. Group M consisted of 19 males and 5 females, giving a female-to-male ratio of 5:19, while Group S consisted of 34 males and 7 females, with a female-to-male ratio of 7:34. The overall female-to-male ratio was 12:53. There was no statistically significant difference in sex distribution between Group M and Group S ($p=0.748$).

[Table 2] Clinical Characteristics According to Elapsed Time to Surgery and Site of Perforation

The mean elapsed time from onset of symptoms to surgery was 57.67 ± 24.47 hours in Group M and 18.54 ± 13.63 hours in Group S. The difference in mean elapsed time between the two groups was statistically significant ($p<0.001$). Among patients who underwent surgery within 12 hours of onset, all 23 patients belonged to Group S and none belonged to Group M. Among patients with an elapsed time of ≥ 12 hours, 24 (57.1%) belonged to Group M and 18 (42.9%) belonged to Group S. The distribution according to elapsed time was statistically significant ($p<0.0001$), with a reported relative risk of 2.1 (95% CI: 1.6–3.3). Regarding the anatomical site of perforation, prepyloric perforation was the most frequently observed site in both groups. It was present in 14 (58.3%) patients in Group M and 32 (78.0%) patients in Group S. Duodenal perforation was observed in 4 (16.7%) patients in Group M and 2 (4.9%) patients in Group S. Ileal perforation was recorded in 2 (8.3%) patients in Group M and was not observed in Group S. Caecal perforation was not recorded in Group M and was present in 1 (2.4%) patient in Group S. Appendicular perforation was noted in 4 (16.7%) patients in Group M and 3 (7.3%) patients in Group S. Transverse colonic perforation was absent in Group M and was observed in 3 (7.3%) patients in Group S.

[Table 3] Comparison of Hematological Parameters Between Mortality and Survivor Groups

The mean total leukocyte count was $8294.58 \pm 5066/\text{mm}^3$ in Group M and $7828.72 \pm 8320/\text{mm}^3$ in Group S. The difference in mean total leukocyte count between the two groups was not statistically significant ($p=0.780$). Among patients with a total leukocyte count $<4000/\text{mm}^3$, 10 (52%) belonged to Group M and 9 (48%) belonged to Group S. Among those with a total leukocyte count $\geq 4000/\text{mm}^3$, 14 (30%) belonged to Group M and 32 (69%) belonged to Group S. The categorical distribution of total leukocyte count was statistically significant ($p<0.001$), with a relative risk of 3.2 (95% CI: 1.6–6.3) for the $<4000/\text{mm}^3$ category. The mean platelet

count was $164 \pm 60 \times 10^3/\mu\text{L}$ in Group M compared with $227 \pm 170 \times 10^3/\mu\text{L}$ in Group S. Although the mean platelet count was numerically lower in Group M, the difference between the two groups was not statistically significant ($p=0.08$).

[Table 4] Comparison of Postoperative Severity Scores Between Mortality and Survivor Groups

The mean APACHE II score was 24.79 ± 7.31 in Group M, whereas it was 13.48 ± 3.07 in Group S. The difference in APACHE II scores between the two groups was statistically significant ($p<0.001$). The mean SOFA score was also higher in Group M, with a value of 9.96 ± 3.81 , compared with 6.24 ± 2.39 in Group S. This difference was statistically significant ($p<0.001$). Thus, both postoperative severity scores showed statistically significant differences between the mortality and survivor groups, with higher mean values recorded in Group M.

[Table 5] Arterial Blood Lactate Levels in Mortality and Survivor Groups

The mean postoperative arterial blood lactate level was 5.35 ± 3.07 mmol/L in Group M compared with 1.26 ± 0.93 mmol/L in Group S. The difference in mean arterial lactate levels between the two groups was statistically significant ($p<0.001$). A lactate level >2 mmol/L was recorded in 28 patients. Of these, 20 (71.4%) belonged to Group M and 8 (28.6%) belonged to Group S. In contrast, among the 37 patients with arterial lactate levels ≤ 2 mmol/L, 4 (10.8%) belonged to Group M and 33 (89.2%) belonged to Group S. The difference in distribution according to the 2 mmol/L lactate cut-off was statistically significant ($p<0.001$). The relative risk associated with an arterial lactate level >2 mmol/L was 3.1, with a 95% confidence interval of 1.7–5.6.

[Table 6] Diagnostic Performance of Arterial Blood Lactate Level for Prediction of Mortality

Using the selected arterial blood lactate cut-off, the sensitivity for identifying mortality was 83.33% (95% CI: 62.62%–95.26%), while the specificity was 80.49% (95% CI: 65.13%–91.18%). The positive predictive value was 71.43% (95% CI: 56.69%–82.68%), whereas the negative predictive value was 89.19% (95% CI: 76.91%–95.34%). The positive likelihood ratio was 4.27, with a 95% confidence interval of 2.24–8.16. The negative likelihood ratio was 0.21, with a 95% confidence interval of 0.08–0.51. These values represented the calculated diagnostic performance parameters of postoperative arterial blood lactate for mortality prediction in the study population.

[Table 7] Multivariate Logistic Regression Analysis of Independent Risk Factors for Mortality

Multivariate logistic regression analysis was performed using elapsed time to surgery, APACHE II score, SOFA score, white blood cell count, and arterial lactate level as variables. Elapsed time to surgery had an odds ratio of 1.012 (95% CI: 0.95–1.23) with a p-value of 0.170 and was not statistically significant. The APACHE II score had an odds ratio of 1.004 (95% CI: 0.86–1.16) with a p-value of 0.604, while the SOFA score had an odds ratio of 1.009 (95% CI: 0.91–1.09) with a p-value of 0.549; neither variable remained statistically significant in the multivariate model. White blood cell count showed an odds ratio of 0.988 (95% CI: 0.84–1.08) with a p-value of 0.740 and was also not statistically significant. Arterial lactate level had an odds ratio of 3.69 (95% CI: 2.6–4.1) with a p-value of 0.0001. Among the variables included in the multivariate analysis, arterial lactate level was the only variable that remained statistically significant.

Table 1: Demographic Characteristics and Distribution of Study Participants

Parameter	Group M (n=24)	Group S (n=41)	Total (n=65)	p-value
Distribution of patients, n (%)	24 (36.92%)	41 (63.08%)	65 (100%)	—
Age (years), Mean $\pm$ SD	51.0 $\pm$ 14.05	48.0 $\pm$ 9.41	—	0.8816
Female, n	5	7	12	0.748
Male, n	19	34	53	
Female : Male ratio	5:19	7:34	12:53	

Table 2: Clinical Characteristics According to Elapsed Time to Surgery and Site of Perforation

Parameter	Group M (n=24)	Group S (n=41)	p-value	Relative Risk (95% CI)
Elapsed time from onset to surgery (hours), Mean $\pm$ SD	57.67 $\pm$ 24.47	18.54 $\pm$ 13.63	<0.001	—
Elapsed time <12 hours, n (%)	0	23 (100%)	<0.0001	2.1 (1.6–3.3)
Elapsed time ≥ 12 hours, n (%)	24 (57.1%)	18 (42.9%)		
Site of perforation				
Prepyloric	14 (58.3%)	32 (78.0%)	—	—
Duodenal	4 (16.7%)	2 (4.9%)	—	—
Ileal	2 (8.3%)	0	—	—
Caecal	0	1 (2.4%)	—	—
Appendix	4 (16.7%)	3 (7.3%)	—	—
Transverse colon	0	3 (7.3%)	—	—
Total	24 (100%)	41 (100%)		

Table 3: Comparison of Hematological Parameters Between Mortality and Survivor Groups

Parameter	Group M (n=24), Mean ± SD / n (%)	Group S (n=41), Mean ± SD / n (%)	p-value	Relative Risk (95% CI)
Total leukocyte count (/mm ³), Mean ± SD	8294.58 ± 5066	7828.72 ± 8320	0.780	—
TLC <4000/mm ³	10 (52%)	9 (48%)	<0.001	3.2 (1.6–6.3)
TLC ≥4000/mm ³	14 (30%)	32 (69%)		
Platelet count (×10 ³ /μL), Mean ± SD	164 ± 60	227 ± 170	0.08	—

Table 4: Comparison of Postoperative Severity Scores Between Mortality and Survivor Groups

Parameter	Group M (n=24), Mean ± SD	Group S (n=41), Mean ± SD	p-value
APACHE II score	24.79 ± 7.31	13.48 ± 3.07	<0.001
SOFA score	9.96 ± 3.81	6.24 ± 2.39	<0.001

Table 5: Arterial Blood Lactate Levels in Mortality and Survivor Groups

Parameter	Group M (n=24)	Group S (n=41)	p-value / 95% CI
Arterial lactate (mmol/L), Mean ± SD	5.35 ± 3.07	1.26 ± 0.93	<0.001
Lactate >2 mmol/L, n (%)	20 (71.4%)	8 (28.6%)	<0.001
Lactate ≤2 mmol/L, n (%)	4 (10.8%)	33 (89.2%)	—
Relative risk for lactate >2 mmol/L	3.1	—	95% CI: 1.7–5.6

Table 6: Diagnostic Performance of Arterial Blood Lactate Level for Prediction of Mortality

Diagnostic Performance Parameter	Value	95% CI
Sensitivity	83.33%	62.62%–95.26%
Specificity	80.49%	65.13%–91.18%
Positive Predictive Value (PPV)	71.43%	56.69%–82.68%
Negative Predictive Value (NPV)	89.19%	76.91%–95.34%
Positive Likelihood Ratio (PLR)	4.27	2.24–8.16
Negative Likelihood Ratio (NLR)	0.21	0.08–0.51

Table 7: Multivariate Logistic Regression Analysis of Independent Risk Factors for Mortality

Parameter	Odds Ratio	95% CI	p-value
Elapsed time to surgery (eTIME)	1.012	0.95–1.23	0.170
APACHE II score	1.004	0.86–1.16	0.604
SOFA score	1.009	0.91–1.09	0.549
White blood cell count	0.988	0.84–1.08	0.740
Arterial lactate level	3.69	2.6–4.1	0.0001

DISCUSSION

In the present study, 65 patients with hollow viscus perforation were evaluated, of whom 24 (36.92%) died within 28 days and 41 (63.08%) survived. The mean age was 51.0 ± 14.05 years in the mortality group and 48.0 ± 9.41 years in the survivor group, with no statistically significant difference (p=0.8816). Males predominated in both groups, accounting for 19 of 24 patients in Group M and 34 of 41 patients in Group S, while sex distribution was not significantly different (p=0.748). Chandran et al. (2024), in a study of 100 patients with hollow viscus perforation, similarly reported marked male predominance, with 78% males and 22% females and a male-to-female ratio of 3.5:1. Their mean age was 43 years, and the largest proportion of patients (35%) belonged to the 41–50-year age group. Thus, the demographic profile of the present series was comparable with their study in terms of middle-aged presentation and marked male predominance.^[7] A marked difference in elapsed time from onset to surgery was observed between the two outcome groups in the present study. The mean elapsed time was 57.67 ± 24.47 hours among non-survivors compared with 18.54 ± 13.63 hours among survivors (p<0.001). None of the 23 patients operated within 12

hours died, whereas among patients with an elapsed time ≥12 hours, 24 (57.1%) belonged to the mortality group and 18 (42.9%) survived; the relative risk was 2.1 (95% CI: 1.6–3.3; p<0.0001). Azuhata et al. (2014) prospectively evaluated 154 patients with gastrointestinal perforation associated with septic shock and found that the 60-day survival rate was 98% when surgery was initiated within the first 2 hours after admission, whereas survival fell progressively with delay and reached 0% when surgery was delayed beyond 6 hours. In multivariate analysis, time to initiation of surgery remained independently associated with outcome (OR 0.29, 95% CI: 0.16–0.47; p<0.0001). Although their interval was measured from hospital admission rather than symptom onset, both studies demonstrated a strong association between increasing delay and adverse outcome.^[8] In relation to the anatomical site of perforation, prepyloric perforation constituted the largest subgroup in the present study, occurring in 14 (58.3%) non-survivors and 32 (78.0%) survivors. Duodenal perforation was found in 16.7% and 4.9%, appendicular perforation in 16.7% and 7.3%, and ileal perforation in 8.3% and 0% of Group M and Group S, respectively. Caecal and transverse colonic perforations were uncommon. Jhobta et al. (2006), in their series of 504 patients with perforation

peritonitis, also demonstrated predominance of upper gastrointestinal perforation; duodenal perforation accounted for 289 (57%) cases, gastric perforation for 42 (8%), ileal perforation for 76 (15%), and jejunal perforation for 16 (3%). Their overall mortality was 10%. Although the exact distribution differed, both studies showed that gastroduodenal perforations constituted a major proportion of cases, while distal intestinal and colonic perforations were relatively less frequent.^[9] The mean total leukocyte count in the present study was $8294.58 \pm 5066/\text{mm}^3$ among non-survivors and $7828.72 \pm 8320/\text{mm}^3$ among survivors, and the difference in mean values was not significant ($p=0.780$). However, when leukocyte count was categorized, 10 (52%) patients with $\text{TLC} < 4000/\text{mm}^3$ belonged to the mortality group compared with 9 (48%) survivors, and leukopenia was significantly associated with mortality ($p<0.001$; RR 3.2, 95% CI: 1.6–6.3). The mean platelet count was also lower among non-survivors ($164 \pm 60 \times 10^3/\mu\text{L}$) than survivors ($227 \pm 170 \times 10^3/\mu\text{L}$), although this difference did not reach statistical significance ($p=0.08$). Matsuoka et al. (2022), studying 146 patients undergoing emergency surgery for colorectal perforation, reported significantly lower postoperative leukocyte counts among patients who died than among survivors [3.2 versus $8.6 \times 10^3/\mu\text{L}$, $p=0.006$]. Postoperative platelet counts were also lower among those who died [14.5 versus $18.2 \times 10^4/\mu\text{L}$, $p=0.035$], and the postoperative reduction in platelet count was significantly greater in non-survivors (39.3% versus 15.1%, $p<0.001$). These findings were comparable with the present observation that lower leukocyte and platelet values were more prominent in the mortality group, although mean platelet count in the present study did not achieve statistical significance.^[10]

The APACHE II score showed a marked difference between the two outcome groups in the present study. The mean score was 24.79 ± 7.31 among non-survivors compared with 13.48 ± 3.07 among survivors, and this difference was highly significant ($p<0.001$). Bylapudi et al. (2021), in a prospective observational study of 50 patients with peritonitis secondary to hollow viscus perforation, similarly reported a significantly higher mean APACHE II score among non-survivors than survivors (19.43 ± 3.10 versus 7.93 ± 3.91 ; $p<0.001$). Mortality in their study increased across APACHE II categories, reaching 75% among patients with scores of 16–20 and 100% among those with scores >20 . The findings of the present study therefore closely paralleled their observed separation of survivors and non-survivors by APACHE II score, although the mean scores in both outcome groups were higher in the present cohort.^[11] The mean postoperative SOFA score in the present study was 9.96 ± 3.81 in the mortality group and 6.24 ± 2.39 in the survivor group, with a statistically significant difference ($p<0.001$). Hynninen et al. (2008) evaluated 163 consecutive patients with secondary peritonitis and reported hospital mortality of 19% and one-year mortality of

23%. Among patients requiring intensive care, the admission SOFA score was independently associated with mortality ($p<0.0001$), and SOFA demonstrated an area under the ROC curve of 0.78 for discrimination of poor outcome. Their analysis also identified the degree of organ dysfunction measured by SOFA as an important mortality-related variable. These observations corresponded with the present study, in which patients who died had substantially higher postoperative SOFA scores than patients who survived.^[12] Postoperative arterial lactate showed one of the largest differences between the outcome groups in the present study. The mean lactate level was 5.35 ± 3.07 mmol/L among non-survivors compared with 1.26 ± 0.93 mmol/L among survivors ($p<0.001$). Among patients with lactate >2 mmol/L, 20 (71.4%) belonged to the mortality group and only 8 (28.6%) survived, whereas among those with lactate ≤ 2 mmol/L, only 4 (10.8%) died and 33 (89.2%) survived. Lactate >2 mmol/L was associated with a relative risk of 3.1 (95% CI: 1.7–5.6). Jobin et al. (2019), in 113 patients undergoing emergency laparotomy for perforation peritonitis, reported a 28-day mortality rate of 15.04%. Immediate postoperative arterial lactate was significantly higher among non-survivors than survivors [median 3.6 (3.1–4.2) versus 1.4 (1.0–2.2) mmol/L; $p<0.0001$]. They identified an immediate postoperative lactate cut-off of 2.8 mmol/L for predicting 28-day mortality; preoperative and 24-hour postoperative cut-offs were 2.75 and 2.45 mmol/L, respectively. Thus, despite differences in the selected cut-off, both studies demonstrated a clear separation in postoperative lactate between survivors and non-survivors.^[13] In the present study, arterial lactate demonstrated a sensitivity of 83.33% (95% CI: 62.62%–95.26%) and specificity of 80.49% (95% CI: 65.13%–91.18%) for prediction of mortality. The positive predictive value was 71.43%, while the negative predictive value was 89.19%. The positive likelihood ratio was 4.27 and the negative likelihood ratio was 0.21. Subedi et al. (2025), in a prospective cohort of 120 patients undergoing emergency laparotomy for gastrointestinal perforation peritonitis, reported a 30-day mortality rate of 35%. A serum lactate threshold of ≥ 1.88 mmol/L predicted mortality with a sensitivity of 81%, specificity of 69.2%, and an AUC of 0.817 ($p=0.0001$). Their combination of serial lactate and APACHE II achieved sensitivity of 85%, specificity of 82%, and an AUC of 0.919. The sensitivity of lactate alone in their study was therefore close to the 83.33% observed in the present study, while the present study demonstrated a higher specificity of 80.49% at the >2 mmol/L threshold.^[14] On multivariate logistic regression in the present study, elapsed time to surgery (OR 1.012, $p=0.170$), APACHE II score (OR 1.004, $p=0.604$), SOFA score (OR 1.009, $p=0.549$), and white blood cell count (OR 0.988, $p=0.740$) did not remain statistically significant. In contrast, arterial lactate was independently associated with mortality, with an odds ratio of 3.69 (95% CI: 2.6–

4.1; $p=0.0001$). Kang et al. (2022) evaluated 104 patients undergoing surgery for gastrointestinal perforation, of whom 17 (16.3%) died in hospital. Their postoperative lactate level was significantly higher in patients who died than in survivors (7.9 ± 4.9 versus 3.6 ± 3.0 mmol/L; $p<0.001$). On multivariate analysis, postoperative lactate remained independently associated with in-hospital mortality (HR 1.259, 95% CI: 1.084–1.463; $p=0.003$), and postoperative lactate produced an AUC of 0.771 for mortality prediction. This closely corresponded with the present multivariate findings, in which arterial lactate remained the statistically significant mortality-associated variable after adjustment for the other included parameters.^[15]

CONCLUSION

Postoperative arterial blood lactate level was significantly associated with mortality in patients operated for hollow viscus perforation. Higher lactate levels were observed among non-survivors and showed good sensitivity and specificity for predicting 28-day mortality. Multivariate analysis identified postoperative lactate as an independent predictor of mortality. Thus, early postoperative lactate measurement may serve as a simple and useful prognostic marker for identifying high-risk patients requiring intensive monitoring and management.

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