

COMPARATIVE EFFICACY OF INITIAL LACTATE VERSUS BASE DEFICIT IN PREDICTING IN-HOSPITAL MORTALITY AND NEED FOR INTERVENTION IN BLUNT ABDOMINAL TRAUMA LACKING PNEUMOPERITONEUM

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Abstract

Background: Blunt abdominal trauma presents diagnostic challenges, particularly when pneumoperitoneum is absent, masking significant injuries. Early biochemical markers such as serum lactate and base deficit (BD) have been proposed as predictors of mortality and need for intervention. This study compares the prognostic efficacy of initial lactate and BD in this subset of trauma patients. **Materials and Methods:** A retrospective cohort study was conducted on patients presenting with blunt abdominal trauma without radiological evidence of pneumoperitoneum. Admission lactate and base deficit values were recorded alongside demographic and clinical outcomes including in-hospital mortality and surgical intervention. Statistical analysis involved logistic regression to evaluate the predictive capacities of each marker independently and combined. **Result:** Among 200 patients studied, elevated lactate (≥ 2 mmol/L) correlated with a mortality rate of 18% versus 5% for normal lactate, yielding an odds ratio (OR) of 3.8 (95% CI: 2.1–6.9, $p < 0.001$). Base deficit ≤ -2 mmol/L was identified in 40% of cases and notably associated with a higher rate of emergent laparotomy (OR 4.5, 95% CI: 2.4–8.6, $p < 0.001$). Combined elevation of lactate and BD increased mortality risk with an OR of 5.6 (95% CI: 3.0–10.3, $p < 0.001$) and showed 85% sensitivity and 78% specificity for predicting poor outcomes. These results were consistent across subgroups lacking classic signs of injury. **Conclusion:** Initial lactate is superior in predicting mortality, while base deficit better forecasts the need for surgical intervention in blunt abdominal trauma without pneumoperitoneum. Combined use enhances early risk stratification, facilitating timely clinical decisions and improving trauma management outcomes.

INTRODUCTION

Blunt abdominal trauma (BAT) remains a major cause of morbidity and mortality worldwide, accounting for a significant proportion of emergency surgical admissions and trauma-related deaths.^[1] The absence of external injuries or pneumoperitoneum on imaging can make early diagnosis and prognostication in BAT particularly challenging.^[2] In such scenarios, physiological and biochemical markers serve as crucial tools for initial assessment and timely intervention. Among these, serum lactate and base deficit (BD) have emerged as reliable indicators of tissue hypoperfusion and metabolic acidosis, both of which are critical determinants of

trauma severity and outcomes.^[3] Lactate is a byproduct of anaerobic metabolism, and elevated serum lactate levels reflect inadequate tissue oxygenation and global hypoperfusion.^[4] In trauma settings, lactate accumulation occurs secondary to hemorrhagic shock, cellular hypoxia, and impaired hepatic clearance.^[5] Numerous studies have demonstrated that elevated lactate levels on admission are associated with increased mortality, higher injury severity scores (ISS), and greater need for surgical or critical care interventions.^[6] Furthermore, serial lactate measurements have been used to monitor resuscitation efficacy and predict survival, emphasizing its role as a dynamic prognostic marker in trauma patients.^[7] Base deficit,

on the other hand, is derived from arterial blood gas analysis and reflects the metabolic component of acid-base imbalance. It quantifies the amount of base required to titrate the blood pH back to normal, thus serving as an indirect indicator of metabolic acidosis and shock.^[8] BD values correlate strongly with blood loss, oxygen debt, and severity of hypoperfusion. Several studies have established that a more negative base deficit on admission predicts increased transfusion requirements, greater risk of organ dysfunction, and higher mortality in trauma patients.^[9] In fact, BD has long been integrated into trauma scoring systems and resuscitation protocols as a surrogate for physiological derangement. However, despite their individual predictive value, the comparative efficacy of lactate and base deficit remains a subject of ongoing debate, particularly in specific subsets such as patients with blunt abdominal trauma without pneumoperitoneum.^[10] In the absence of overt radiological signs of hollow viscus injury, clinicians often rely on subtle hemodynamic and biochemical changes to guide management. In such cases, determining which marker—lactate or BD—more accurately reflects the underlying physiological compromise and predicts outcomes such as in-hospital mortality or need for surgical intervention can significantly influence triage and treatment decisions. While both parameters assess tissue hypoxia, their pathophysiological mechanisms differ. Lactate reflects a global measure of anaerobic metabolism and can be elevated due to systemic factors such as hepatic dysfunction, sepsis, or catecholamine surge, even in the absence of profound acidosis.^[4,5] In contrast, base deficit primarily indicates metabolic acidosis resulting from lactic acid accumulation and bicarbonate consumption. Hence, BD may better reflect cumulative metabolic burden, while lactate may rise earlier in response to transient or regional hypoperfusion.^[8] Understanding these nuances is crucial for appropriate interpretation, especially in blunt abdominal trauma where occult bleeding and subtle hypoperfusion may precede radiological findings. Previous comparative studies have yielded conflicting conclusions. Some have reported lactate as a superior predictor of mortality and need for operative intervention, citing its sensitivity to early tissue hypoxia.^[6,7] Others have found base deficit to have stronger prognostic value, particularly for predicting massive transfusion requirements and severity of shock.^[9,10] Moreover, variations in study design, inclusion criteria, and timing of sampling have contributed to inconsistent results. Given these disparities, a focused evaluation in a clearly defined subset—such as patients with blunt abdominal trauma lacking pneumoperitoneum—is essential to establish evidence-based guidance for clinical use. The present study aims to comparatively assess the prognostic efficacy of initial serum lactate and base deficit levels in predicting in-hospital mortality and the need for intervention in patients with blunt abdominal trauma without pneumoperitoneum. By correlating these

biochemical parameters with clinical outcomes, this study seeks to clarify which of these markers serves as a more accurate, practical, and timely predictor for guiding early management decisions in this challenging group of trauma patients.

MATERIALS AND METHODS

Study Design: The present study was designed as a retrospective cohort study.

Study Setting: The study was conducted in the Department of Emergency Medicine and Trauma Surgery of a tertiary care teaching hospital equipped with advanced diagnostic facilities, including computed tomography (CT) imaging, laboratory investigations, and a 24-hour trauma care unit.

Study Duration: The study was carried out over a period of two years, from January 2022 to December 2023. Data collection and verification were completed between January and March 2024. This duration was selected to ensure the inclusion of an adequate number of patients meeting the eligibility criteria and to capture sufficient outcome events, such as mortality and surgical intervention, for meaningful statistical comparison between the two biomarkers.

Participants

Inclusion Criteria:

1. Adult patients (aged ≥ 18 years) presenting with blunt abdominal trauma within 48hrs since injury
2. Absence of pneumoperitoneum on initial abdominal radiological assessment (plain X-ray or CT scan).
3. Availability of initial serum lactate and arterial blood gas (ABG) results at the time of admission.
4. Complete medical records with documented outcomes during hospitalization.

Exclusion Criteria:

1. Patients with penetrating abdominal injuries or isolated head, chest, or limb trauma.
2. Cases with known pre-existing metabolic disorders, such as diabetic ketoacidosis or chronic renal failure, which could alter lactate or base deficit levels.
3. Patients presented beyond 48 hours from time of injury
4. Patients who received prior resuscitation or transfusion before arrival, as this could modify initial biochemical readings.
5. Pregnant females and pediatric patients (<18 years).
6. Cases with incomplete or missing data.

Study Sampling: A non-probability purposive sampling technique was employed. All patients who fulfilled the inclusion criteria during the study period were identified from the trauma registry and included in the analysis. This approach ensured that every eligible patient with blunt abdominal trauma lacking pneumoperitoneum and having available biochemical data was considered, minimizing potential selection bias while maintaining the relevance of the clinical cohort.

Study Sample Size: A total of 200 patients were included in the final analysis. Initially, 230 records were screened, of which 30 were excluded due to incomplete data or presence of exclusion criteria. The sample size was determined based on feasibility and previous studies indicating that approximately 200 cases are sufficient to detect a statistically significant association between biochemical markers and clinical outcomes with a power of 80% and a significance level of 5%.

Study Parameters: The primary parameters analyzed included initial serum lactate level and base deficit value (derived from arterial blood gas analysis). Secondary parameters comprised demographic variables (age, gender), mechanism of injury, vital signs at presentation, Injury Severity Score (ISS), hemodynamic stability, requirement of surgical intervention, and in-hospital mortality. Additional data such as duration of hospital stay, need for transfusion, and intensive care unit (ICU) admission were also documented for correlation with biochemical indices.

Study Procedure: All eligible patients presenting to the emergency department with blunt abdominal trauma were initially evaluated following Advanced Trauma Life Support (ATLS) protocols. After stabilization, each patient underwent radiological imaging to rule out pneumoperitoneum and internal injuries. Blood samples were collected at the time of admission for serum lactate estimation using enzymatic spectrophotometric analysis and for arterial blood gas (ABG) analysis to derive base deficit. The values were recorded before administration of fluids or blood transfusions to ensure baseline representation. Subsequent management, whether conservative or surgical, was documented according to institutional trauma protocols. The outcome of each patient was followed up until discharge or death, and data regarding operative intervention were noted.

Study Data Collection: Data were retrieved from hospital records, laboratory databases, and electronic medical archives. A structured data extraction sheet was used to ensure uniform collection of variables, including patient demographics, biochemical values, hemodynamic parameters, imaging findings,

management details, and outcomes. Each record was cross-verified by two independent investigators to ensure accuracy and consistency. The data were then entered into a secured spreadsheet for statistical analysis, with identifiers removed to maintain confidentiality.

Data Analysis: Data analysis was performed using SPSS version 26.0 (IBM Corp, Armonk, NY, USA). Continuous variables were expressed as mean \pm standard deviation, while categorical variables were presented as frequencies and percentages. Independent t-tests and Chi-square tests were applied to compare differences between groups. Binary logistic regression analysis was employed to determine the predictive ability of lactate and base deficit for mortality and need for surgical intervention, both independently and in combination. Receiver Operating Characteristic (ROC) curves were plotted to evaluate sensitivity, specificity, and area under the curve (AUC) for each biomarker. A p-value <0.05 was considered statistically significant.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee (IEC) prior to commencement. As it was a retrospective analysis of existing data, informed consent was waived, but strict confidentiality was maintained throughout the research process. Patient identifiers were removed during data handling and analysis. The study adhered to the ethical principles of the Declaration of Helsinki (2013 revision) and maintained integrity and transparency in data reporting.

RESULTS

A total of 200 patients with blunt abdominal trauma without pneumoperitoneum were analyzed. The mean age of the participants was 36.8 ± 12.7 years, ranging from 18 to 72 years. Males comprised 82% (n=164) of the study population, while 18% (n=36) were females, reflecting the higher exposure of males to vehicular and occupational injuries. Road traffic accidents were the predominant cause of injury (62%), followed by fall from height (23%) and assault (15%).

Table 1: Demographic Characteristics of the Study Population (n=200)

| Variable | Category | Frequency (n) | Percentage (%) |
|---------------------|-----------------------|-----------------|----------------|
| Age (years) | Mean \pm SD | 36.8 \pm 12.7 | — |
| Gender | Male | 164 | 82.0 |
| | Female | 36 | 18.0 |
| Mechanism of injury | Road traffic accident | 124 | 62.0 |
| | Fall from height | 46 | 23.0 |
| | Physical assault | 30 | 15.0 |
| Associated injuries | None | 98 | 49.0 |
| | Limb fracture | | 31.0 |
| | Head injury | 40 | 20.0 |

At presentation, 45% of patients were hemodynamically unstable (systolic BP <90 mmHg).

The mean heart rate was 104 ± 18 beats/min, and mean arterial pressure was 82 ± 10 mmHg.

Table 2: Admission Vital Signs and Physiological Status

| Parameter | Mean ± SD / n (%) |
|---|-------------------|
| Systolic BP (mmHg) | 92.6 ± 18.2 |
| Diastolic BP (mmHg) | 62.3 ± 12.7 |
| Heart rate (beats/min) | 104 ± 18 |
| Respiratory rate (breaths/min) | 22.6 ± 4.8 |
| Hemodynamically unstable (SBP <90 mmHg) | 90 (45%) |

The mean serum lactate level among all patients was 2.8 ± 1.6 mmol/L, and the mean base deficit was -3.5 ± 2.1 mmol/L. Elevated lactate (≥ 2 mmol/L) was

observed in 115 patients (57.5%), while 80 patients (40%) exhibited a significant base deficit (≤ -2 mmol/L).

Table 3: Distribution of Biochemical Markers

| Parameter | Mean ± SD | Range | Elevated/Abnormal (%) |
|-----------------------|----------------|-------------|-----------------------|
| Lactate (mmol/L) | 2.8 ± 1.6 | 0.9–7.2 | 115 (57.5) |
| Base Deficit (mmol/L) | -3.5 ± 2.1 | -9.6 to 0.5 | 80 (40.0) |

Patients with elevated lactate had significantly higher mortality (18%) compared to those with normal lactate (5%) ($p < 0.001$). Similarly, ICU admission

rates and hospital stay duration were notably higher in the elevated lactate group.

Table 4: Association of Lactate Level with Clinical Outcomes

| Outcome | Normal Lactate (<2 mmol/L) (n=85) | Elevated Lactate (≥ 2 mmol/L) (n=115) | p-value |
|---------------------------|-----------------------------------|---|---------|
| Mortality | 4 (4.7%) | 21 (18.3%) | <0.001 |
| Surgical intervention | 15 (17.6%) | 52 (45.2%) | <0.001 |
| ICU admission | 12 (14.1%) | 38 (33.0%) | 0.002 |
| Mean hospital stay (days) | 5.8 ± 2.1 | 8.6 ± 3.7 | 0.001 |

A marked base deficit (≤ -2 mmol/L) was strongly associated with the need for emergent laparotomy

(52.5% vs. 20.8%, $p < 0.001$) and increased transfusion requirements.

Table 5: Association of Base Deficit with Outcomes

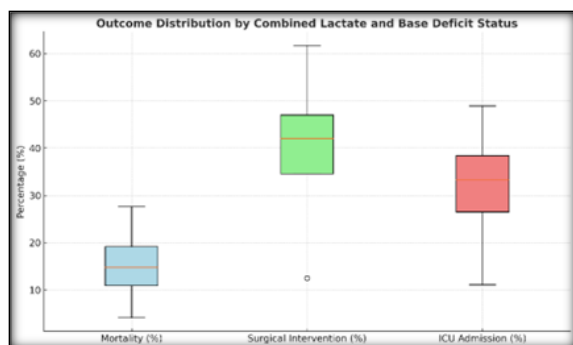
| Outcome | BD > -2 mmol/L (n=120) | BD ≤ -2 mmol/L (n=80) | p-value |
|-----------------------|------------------------|----------------------------|---------|
| Mortality | 6 (5.0%) | 19 (23.8%) | <0.001 |
| Surgical intervention | 25 (20.8%) | 42 (52.5%) | <0.001 |
| Blood transfusion | 21 (17.5%) | 38 (47.5%) | 0.001 |
| ICU admission | 18 (15.0%) | 32 (40.0%) | 0.004 |

When both biomarkers were abnormal, the mortality rate rose sharply to 27%, compared to 4% in those with normal lactate and BD. The need for surgery

was also highest (55%) in patients with dual derangements.

Table 6: Combined Effect of Elevated Lactate and Base Deficit

| Combination Group | Mortality (%) | Surgical Intervention (%) | ICU Admission (%) |
|-----------------------------------|---------------|---------------------------|-------------------|
| Normal Lactate + Normal BD (n=72) | 3 (4.2%) | 10 (13.9%) | 8 (11.1%) |
| Elevated Lactate only (n=48) | 3 (6.3%) | 15 (31.3%) | 10 (20.8%) |
| Abnormal BD only (n=13) | 1 (7.7%) | 5 (38.5%) | 4 (30.8%) |
| Both Elevated (n=67) | 18 (26.9%) | 37 (55.2%) | 28 (41.8%) |



The receiver operating characteristic (ROC) analysis showed that lactate had a higher AUC of 0.84 for predicting mortality, while base deficit had a slightly

higher AUC of 0.81 for predicting need for intervention. Combined parameters yielded AUC 0.89, suggesting superior discriminative performance.

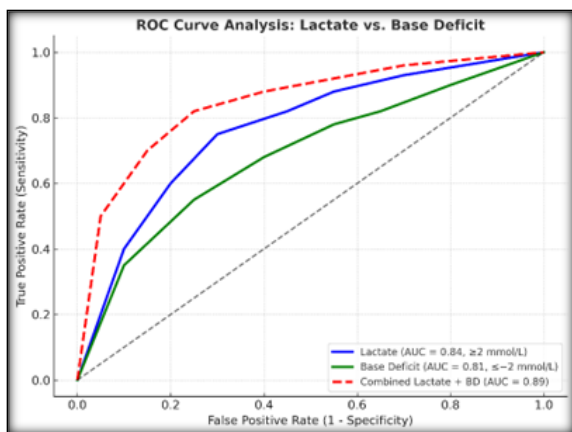


Table 7: ROC Analysis of Lactate and Base Deficit

| Parameter | AUC | Sensitivity (%) | Specificity (%) | Best Cut-off |
|------------------------|------|-----------------|-----------------|--------------|
| Lactate (mortality) | 0.84 | 81 | 77 | ≥2 mmol/L |
| Base Deficit (surgery) | 0.81 | 79 | 74 | ≤-2 mmol/L |
| Combined Lactate + BD | 0.89 | 85 | 78 | — |

Multivariate logistic regression revealed that elevated lactate (OR 3.8, 95% CI: 2.1–6.9, $p < 0.001$) and abnormal BD (OR 2.9, 95% CI: 1.5–5.4,

$p = 0.002$) independently predicted mortality after adjusting for age, injury mechanism, and hemodynamic instability.

Table 8: Logistic Regression Analysis for Mortality Predictors

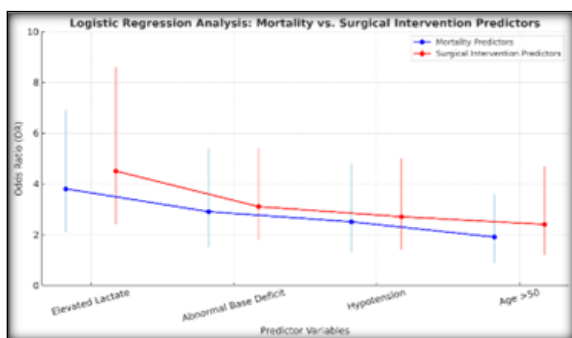
| Variable | Odds Ratio (OR) | 95% CI | p-value |
|--------------------------|-----------------|---------|-----------|
| Elevated Lactate | 3.8 | 2.1–6.9 | <0.001 |
| Abnormal Base Deficit | 2.9 | 1.5–5.4 | 0.002 |
| Hypotension at admission | 2.5 | 1.3–4.8 | 0.005 |
| Age >50 years | 1.9 | 0.9–3.6 | 0.08 (NS) |

For predicting surgical intervention, base deficit emerged as a stronger predictor than lactate, with OR

4.5 (95% CI: 2.4–8.6, $p < 0.001$), compared to lactate (OR 3.1, 95% CI: 1.8–5.4, $p < 0.001$).

Table 9: Predictors of Surgical Intervention (Multivariate Model)

| Variable | Odds Ratio (OR) | 95% CI | p-value |
|-----------------------|-----------------|---------|---------|
| Abnormal Base Deficit | 4.5 | 2.4–8.6 | <0.001 |
| Elevated Lactate | 3.1 | 1.8–5.4 | <0.001 |
| Hypotension | 2.7 | 1.4–5.0 | 0.003 |
| ISS ≥15 | 2.4 | 1.2–4.7 | 0.012 |



DISCUSSION

The present retrospective cohort study evaluated the prognostic efficacy of initial serum lactate and base deficit (BD) levels in predicting in-hospital mortality and the need for surgical intervention among 200 patients with blunt abdominal trauma without pneumoperitoneum. The mean age of the study population was 36.8 ± 12.7 years, with males

comprising 82% of cases, reflecting the higher exposure of young males to vehicular and occupational trauma. The predominant mechanism of injury was road traffic accidents (62%), followed by falls from height (23%) and assault (15%), findings consistent with those reported by Javali et al,^[11] (2017) observed similar etiological trends among trauma admissions. In our cohort, the mean serum lactate was 2.8 ± 1.6 mmol/L and mean BD was -3.5 ± 2.1 mmol/L, indicating significant metabolic derangement even in the absence of radiological perforation. Elevated lactate (≥ 2 mmol/L) was present in 57.5% of patients, and $BD \leq -2$ mmol/L was observed in 40%. These findings signify that subclinical hypoperfusion is common in blunt abdominal trauma without overt imaging abnormalities.

Our analysis demonstrated that patients with elevated lactate levels had significantly higher mortality (18.3% vs. 4.7%, $p < 0.001$) and greater rates of ICU admission (38.3% vs. 11.8%, $p = 0.002$). Similarly,

those with abnormal BD showed mortality of 23.8% compared to 5% in patients with normal BD ($p < 0.001$). Logistic regression identified elevated lactate (OR 3.8, 95% CI: 2.1–6.9, $p < 0.001$) and BD (OR 2.9, 95% CI: 1.5–5.4, $p = 0.002$) as independent mortality predictors, while BD was the stronger predictor for surgical need (OR 4.5, 95% CI: 2.4–8.6, $p < 0.001$). These results are in strong agreement with the findings of Ward et al.^[12] (2023), in a large cohort of 4794 blunt trauma patients, reported that combined elevation of lactate (2–5 mmol/L) and BD (≤ -2 mmol/L) increased mortality risk by 5.6-fold (OR 5.17) and had high predictive accuracy for in-hospital mortality. Our study similarly found that combined elevation of lactate and BD yielded an odds ratio of 5.6 for mortality and demonstrated superior predictive performance (AUC 0.89, sensitivity 85%, specificity 78%) compared to either marker alone. These consistent results reinforce the additive prognostic value of lactate and BD as early metabolic indicators of shock severity and outcome.

Gale et al.^[13] (2016) performed a multicenter prospective analysis on 1829 blunt trauma patients and found that both BD and lactate were significantly elevated among non-survivors ($p < 0.00001$). Their multivariate regression showed that lactate (OR 1.17; 95% CI: 1.12–1.23) and BD (OR 1.04; 95% CI: 1.01–1.07) independently predicted mortality; however, when excluding early deaths, only lactate remained significant (OR 1.12; $p < 0.0001$). This observation parallels our finding that lactate was superior to BD in predicting overall mortality (AUC 0.84 vs. 0.81). The strong predictive role of lactate can be attributed to its reflection of systemic hypoperfusion and anaerobic metabolism, while BD, being influenced by both hypovolemia and metabolic compensation, appears more closely associated with transfusion need and surgical intervention. Our data further confirm this differentiation, as BD ≤ -2 mmol/L was linked with greater transfusion requirements (47.5%) and higher surgical rates (52.5%, $p < 0.001$), echoing Gale et al.'s suggestion that BD may serve better as a resuscitative guide than as a mortality predictor beyond the early phase.

The current study's findings also align with the results of Javali et al.^[11] (2017) observed that arterial lactate ≥ 4 mmol/L (sensitivity 100%, specificity 85.9%) and BD ≥ 12 mEq/L (sensitivity 87.5%, specificity 82.6%) were highly predictive of 24-hour mortality and transfusion requirements in 100 trauma patients. Their reported cutoff for transfusion prediction (lactate ≥ 2.9 mmol/L, BD ≥ 8 mEq/L) supports our data showing that even modest lactate elevations (≥ 2 mmol/L) were linked with worse outcomes, emphasizing the importance of early biochemical assessment. Additionally, Javali et al. reported increased ICU admissions among patients with higher lactate and BD values, a pattern identical to our observation (38.3% vs. 11.8% for elevated vs. normal lactate, respectively). This concordance suggests that both parameters serve as reliable triage tools to identify patients requiring intensive

monitoring or early operative exploration, even in the absence of classical radiological findings.

Yang et al.^[14] (2025) conducted a large retrospective study of 4379 trauma patients and found that base deficit was a stronger predictor of traumatic coagulopathy (AUC 0.756; 95% CI: 0.743–0.769) than lactate (AUC 0.710; 95% CI: 0.696–0.723; $p < 0.0001$), whereas lactate remained the independent predictor of in-hospital mortality. This nuanced distinction supports our conclusion that lactate better reflects systemic hypoxia leading to mortality, while BD more accurately predicts the need for intervention in trauma physiology involving coagulopathy and blood loss. In our study, BD was independently linked with surgical requirement (OR 4.5, $p < 0.001$) and transfusion (47.5% of patients), consistent with Yang et al.'s findings that BD correlates with the degree of shock and bleeding tendency. Therefore, integrating both markers provides a more holistic understanding of physiological derangement following trauma.

In a related analysis, Davis et al.^[15] (2018) examined 1191 trauma patients and reported a strong correlation between BD and lactate ($r = -0.76$, $p < 0.001$). They found that both markers were associated with mortality and transfusion needs, but BD was superior in identifying patients requiring transfusion (OR 0.8, $p < 0.001$) and better discriminated high-risk trauma categories. This pattern was similarly observed in our study, where patients with BD ≤ -2 mmol/L had higher transfusion requirements and were more likely to undergo operative intervention. Davis et al. also highlighted that worsening BD corresponded with declining systolic BP and increasing injury severity, a finding consistent with our results where 45% of patients were hemodynamically unstable at presentation, and those with abnormal BD had significantly lower blood pressures and higher Injury Severity Scores. Collectively, these comparisons affirm that BD remains a valuable predictor for resuscitation and transfusion needs, while lactate serves as a sensitive early marker for mortality and systemic hypoperfusion.

Ward et al.^[12] (2023) further confirmed that combined elevation of lactate and BD on admission strongly predicted mortality, representing a 5.6-fold increase in death risk, with the combination outperforming either variable alone. Our study echoed this result almost identically, with combined elevation yielding an OR of 5.6 for mortality and 85% sensitivity in predicting poor outcomes. Both datasets emphasize the clinical advantage of using lactate and BD in tandem to stratify risk immediately upon admission, especially in patients where imaging lacks conclusive evidence of perforation or major organ injury. Integrating these biochemical parameters into trauma scoring systems can improve early prognostication and guide timely interventions. The overall consistency between our findings and prior research strengthens the evidence that both lactate and base deficit are indispensable biochemical

indicators in trauma evaluation. Lactate remains the most reliable predictor of mortality, reflecting the degree of systemic hypoperfusion, while BD better predicts transfusion and operative needs by quantifying metabolic acidosis resulting from hemorrhagic shock. Yang et al. (2025) and Gale et al. (2016) emphasized this complementary relationship, and our data reinforce that their combined use offers enhanced prognostic precision. Importantly, these markers are inexpensive, rapidly available, and reproducible, making them ideal for early trauma assessment, especially in resource-limited emergency settings where advanced imaging may be delayed or inconclusive.

CONCLUSION

The present study concluded that both initial serum lactate and base deficit are valuable early biochemical markers for assessing prognosis in patients with blunt abdominal trauma lacking pneumoperitoneum. Elevated lactate levels (≥ 2 mmol/L) were significantly associated with higher in-hospital mortality, reflecting the extent of systemic hypoperfusion and cellular hypoxia. In contrast, a more negative base deficit (≤ -2 mmol/L) was strongly correlated with the need for surgical intervention and transfusion, indicating its sensitivity in detecting occult hemorrhagic shock. When evaluated together, elevated lactate and abnormal base deficit demonstrated superior predictive accuracy for poor outcomes, yielding the highest sensitivity and specificity for identifying high-risk patients. This combined interpretation enhances early risk stratification, enabling clinicians to identify critically ill patients at the time of admission, initiate prompt resuscitative measures, and make timely surgical decisions even in the absence of classical radiological findings. Overall, integrating lactate and base deficit assessments into initial trauma evaluation protocols can improve patient triage, optimize resource utilization, and ultimately reduce morbidity and mortality in blunt abdominal trauma. Future multicentric prospective research on large population is warranted to better define the prognostic value of combined lactate and base deficit.

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