

Research Article

Utility Of Serum Lactate and Base Deficit as Predictors of Early Outcomes Aftercardiopulmonary Bypass

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ABSTRACT

Background: Cardiopulmonary bypass (CPB) is associated with complex physiological changes, including altered oxygen delivery, systemic inflammatory response, and metabolic disturbances that may contribute to postoperative morbidity. Serum lactate and base deficit are easily measurable indicators of tissue hypoperfusion and metabolic stress; however, their combined predictive utility for early outcomes after CPB requires further evaluation. This study aimed to assess the role of serum lactate and base deficit as predictors of early postoperative complications following CPB-assisted cardiac surgery.

Methods: Serial arterial blood gas analyses were performed at predefined perioperative time points, including before CPB, during CPB, at termination of CPB, ICU admission, and 6 hours postoperatively. Serum lactate levels and base deficit values were recorded. Patients were followed during the early postoperative period for major complications, including acute kidney injury, prolonged mechanical ventilation, low cardiac output syndrome, arrhythmias, prolonged ICU stay, and in-hospital mortality. The predictive performance of lactate and base deficit was evaluated using receiver operating characteristic (ROC) curve analysis.

Results: The mean age of study participants was 58.4 ± 10.6 years, with male predominance (67.8%). Serum lactate levels increased progressively from baseline (1.2 ± 0.4 mmol/L) to peak values at ICU admission (3.6 ± 1.5 mmol/L) before declining during early postoperative recovery. Early postoperative complications occurred in 32 patients (35.6%).

Conclusion: Serum lactate and base deficit are effective early predictors of adverse postoperative outcomes following cardiopulmonary bypass-assisted cardiac surgery.

Keywords: Cardiopulmonary Bypass, Serum Lactate, Base Deficit, Cardiac Surgery, Postoperative Complications, Metabolic Acidosis, Prognostic Biomarkers.

INTRODUCTION

Cardiopulmonary bypass (CPB) is an essential component of modern cardiac surgery, providing temporary cardiac and respiratory support by maintaining systemic circulation and oxygenation during procedures requiring cardiac arrest or manipulation of the heart. Although advances in surgical techniques, perfusion strategies, and perioperative care have substantially improved outcomes, the period immediately following CPB remains associated with complex physiological alterations that may contribute to postoperative morbidity and mortality. The use of CPB induces systemic inflammatory activation, haemodilution, alterations in microcirculatory function, coagulation disturbances, and changes in oxygen delivery and utilization. These physiological responses may result in tissue hypoperfusion, metabolic

stress, and organ dysfunction, making early identification of patients at increased risk a major focus in cardiac surgical care [1].

One of the most important consequences of inadequate tissue oxygenation during and after CPB is the development of metabolic acidosis, which can be assessed through biochemical markers such as serum lactate and base deficit. Serum lactate is widely recognized as a surrogate marker of anaerobic metabolism and impaired tissue oxygen balance. Under normal conditions, lactate is continuously produced through glycolysis and metabolized primarily by the liver and kidneys. However, when oxygen delivery becomes insufficient to meet cellular metabolic requirements, pyruvate metabolism shifts toward lactate production, resulting in increased circulating lactate concentrations. In patients undergoing cardiac surgery, elevated

lactate levels may indicate inadequate oxygen delivery, impaired perfusion, excessive inflammatory response, or reduced metabolic clearance [2].

Hyperlactatemia during CPB is a frequently observed phenomenon and may occur due to both hypoxic and non-hypoxic mechanisms. Type A lactic acidosis develops as a result of inadequate tissue oxygenation caused by reduced cardiac output, low pump flow, anaemia, or impaired microcirculation. Conversely, Type B hyperlactatemia may occur due to catecholamine administration, hypothermia, glucose metabolism alterations, and pharmacological effects during surgery. Regardless of the underlying mechanism, increased lactate levels during the perioperative period have been consistently associated with adverse outcomes, including prolonged mechanical ventilation, increased intensive care unit (ICU) stay, renal dysfunction, and increased mortality after cardiac surgery [3].

Several studies have demonstrated that lactate concentration during CPB provides valuable prognostic information regarding early postoperative outcomes. Demers et al. reported that elevated lactate levels during cardiac surgery were associated with increased postoperative complications and mortality, emphasizing the importance of lactate monitoring as an early indicator of inadequate perfusion [4]. Similarly, Ranucci et al. demonstrated that hyperlactatemia during CPB was associated with increased risk of morbidity and mortality, with factors such as prolonged bypass duration, reduced oxygen delivery, and complex surgical procedures contributing to lactate accumulation [5]. More recent studies have also suggested that intraoperative changes in lactate levels are independently associated with short-term outcomes, supporting its role as an accessible and clinically useful biomarker for risk assessment [6].

However, serum lactate alone may not provide a complete assessment of metabolic disturbances following CPB. Base deficit, obtained from arterial blood gas analysis, represents another important indicator of metabolic imbalance and cumulative oxygen debt. Base deficit reflects the amount of acid required to restore blood pH to normal under standardized conditions and provides information regarding metabolic acidosis caused by lactate accumulation, bicarbonate depletion, and other metabolic abnormalities.

Unlike lactate, which primarily reflects one component of metabolic stress, base deficit provides an integrated assessment of the overall acid–base status of the patient [7].

The development of significant base deficit after cardiac surgery has been associated with poor clinical outcomes. A persistent negative base deficit indicates ongoing metabolic derangement and may reflect inadequate tissue perfusion, systemic inflammatory response, or impaired organ function. Zante et al. evaluated the predictive value of lactate and base excess after cardiac surgery and reported that severely reduced base excess was independently associated with ICU mortality, suggesting that acid–base parameters may provide important prognostic information during early postoperative management [8]. Therefore, assessment of base deficit may complement lactate measurement and improve early recognition of patients requiring intensified monitoring and intervention.

The pathophysiological changes occurring during CPB are influenced by multiple factors, including duration of bypass, aortic cross-clamp time, haemoglobin concentration, temperature management, surgical complexity, and perioperative haemodynamic stability. Prolonged CPB duration may increase inflammatory activation and oxygen consumption imbalance, resulting in greater metabolic stress and accumulation of lactate and hydrogen ions. Furthermore, inadequate oxygen delivery during bypass can create an oxygen debt that persists even after restoration of spontaneous circulation, increasing the risk of postoperative organ dysfunction [9].

Early prediction of adverse outcomes after CPB remains challenging because conventional clinical parameters may not detect subtle metabolic abnormalities before the onset of organ failure. Biomarkers such as lactate and base deficit are attractive because they are routinely available, rapidly measurable, and can provide real-time information regarding the physiological condition of the patient. Identifying threshold values associated with poor outcomes may allow clinicians to optimize perfusion management, initiate corrective interventions earlier, and improve postoperative recovery.

Although previous studies have investigated lactate and acid–base abnormalities individually, the combined utility of serum lactate and base deficit as predictors of early

outcomes after CPB requires further evaluation. Understanding the relationship between these metabolic markers and postoperative complications may improve risk stratification and support individualized perioperative management strategies. Therefore, the present study aims to evaluate the utility of serum lactate and base deficit as predictors of early outcomes after cardiopulmonary bypass, with the objective of determining their association with postoperative morbidity and short-term clinical outcomes.

MATERIALS AND METHODS

Study Design and Study Setting

This prospective observational study was conducted in the Department of Cardiothoracic Surgery and Cardiac Anaesthesia at _____

Hospital to evaluate the utility of serum lactate and base deficit as predictors of early outcomes following cardiopulmonary bypass (CPB)-assisted cardiac surgery. The study was designed to assess perioperative metabolic changes and determine the association between intraoperative biochemical markers and short-term postoperative outcomes. The study was conducted over a predefined study period following approval from the Institutional Ethics Committee, and all procedures were performed in accordance with the principles outlined in the Declaration of Helsinki.

Study Population and Sample Size

A total of 90 adult patients undergoing elective cardiac surgery requiring cardiopulmonary bypass were prospectively enrolled in the study. Patients were recruited according to predefined inclusion and exclusion criteria. The sample size was selected based on previous studies evaluating the prognostic significance of lactate and acid–base parameters in cardiac surgical patients, considering the feasibility of recruitment and the requirement to identify clinically meaningful associations between metabolic biomarkers and postoperative outcomes.

Inclusion Criteria

Patients fulfilling the following criteria were included:

- Adult patients aged ≥ 18 years.
- Patients undergoing elective cardiac surgical procedures requiring cardiopulmonary bypass.
- Patients undergoing procedures such as coronary artery bypass grafting (CABG),

valve replacement/repair, or combined cardiac procedures.

- Patients with complete perioperative arterial blood gas and biochemical monitoring data.
- Patients or legally authorized representatives providing written informed consent.

Exclusion Criteria

Patients were excluded if they had:

- Emergency cardiac surgery.
- Pre-existing severe renal dysfunction requiring dialysis.
- Chronic liver disease affecting lactate metabolism.
- Active sepsis or systemic infection before surgery.
- Preoperative metabolic acidosis.
- Severe peripheral vascular disease affecting tissue perfusion.
- Incomplete perioperative laboratory or clinical data.
- Patients refusing participation in the study.

Preoperative Assessment

All enrolled patients underwent standardized preoperative evaluation, including detailed clinical history, physical examination, assessment of cardiac function, routine biochemical investigations, complete blood count, renal and liver function tests, coagulation profile, and echocardiographic evaluation. Baseline demographic characteristics including age, sex, body mass index, comorbid conditions (diabetes mellitus, hypertension, chronic kidney disease), left ventricular ejection fraction, and type of planned cardiac procedure were recorded.

Anaesthetic and Cardiopulmonary Bypass Management

All patients received standardized general anaesthesia according to institutional protocols. Invasive arterial monitoring was established before induction of anaesthesia for continuous haemodynamic monitoring and serial arterial blood sampling. Anaesthetic management, mechanical ventilation, temperature regulation, and vasoactive medication administration were performed according to standard cardiac anaesthesia practices.

Cardiopulmonary bypass was performed using a membrane oxygenator with non-pulsatile flow. Pump flow rates were adjusted according to patient body surface area and

haemodynamic requirements. Systemic anticoagulation was achieved using unfractionated heparin, and activated clotting time was maintained according to institutional CPB protocols. Core temperature management, myocardial protection strategies, aortic cross-clamp duration, and total CPB duration were recorded for each patient.

Measurement of Serum Lactate and Base Deficit

Serial arterial blood gas analyses were performed at predefined perioperative time points:

1. Before initiation of cardiopulmonary bypass (baseline).
2. During cardiopulmonary bypass.
3. At termination of CPB.
4. Immediately after admission to the intensive care unit (ICU).
5. Six hours postoperatively.

Serum lactate concentration (mmol/L) was measured using a validated blood gas analyser. Base deficit (mmol/L) was calculated automatically from arterial blood gas parameters, including pH, partial pressure of carbon dioxide, bicarbonate concentration, and haemoglobin values. The highest postoperative lactate value and maximum base deficit within the early postoperative period were considered for outcome analysis.

Assessment of Early Postoperative Outcomes

Patients were followed during the immediate postoperative period to evaluate early clinical outcomes. The primary outcome was the occurrence of major postoperative complications within the first seven postoperative days.

The secondary outcomes assessed included:

- Duration of mechanical ventilation.
- Length of intensive care unit stay.
- Requirement and duration of vasoactive support.
- Development of acute kidney injury according to Kidney Disease Improving Global Outcomes (KDIGO) criteria.
- Postoperative arrhythmias.
- Re-exploration for bleeding.

- In-hospital mortality.

Statistical Analysis

Data analysis was performed using appropriate statistical software. Continuous variables were tested for normality using the Shapiro–Wilk test and were expressed as mean $\pm$ standard deviation or median with interquartile range, depending on distribution. Categorical variables were presented as frequencies and percentages.

Patients were categorized into groups based on postoperative outcomes and compared according to lactate levels and base deficit values. Independent sample *t*-test or Mann–Whitney U test was used for comparison of continuous variables, while Chi-square test or Fisher’s exact test was applied for categorical variables.

Correlation between serum lactate, base deficit, and postoperative outcomes was evaluated using Pearson or Spearman correlation analysis. Receiver operating characteristic (ROC) curve analysis was performed to determine the predictive ability of lactate and base deficit for adverse postoperative outcomes. The area under the curve (AUC), sensitivity, specificity, and optimal cut-off values were calculated.

Multivariable logistic regression analysis was performed to identify independent predictors of early postoperative complications after adjusting for potential confounding variables, including age, comorbidities, CPB duration, aortic cross-clamp time, and baseline cardiac function. Odds ratios (OR) with 95% confidence intervals (CI) were reported. A two-tailed *p* value <0.05 was considered statistically significant.

RESULTS

A total of 90 patients undergoing cardiopulmonary bypass (CPB)-assisted cardiac surgery were included in the final analysis. All patients completed perioperative metabolic assessment with serial serum lactate and arterial blood gas measurements. The relationship between lactate, base deficit, and early postoperative outcomes was evaluated.

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants (N = 90)

Variables	Findings
Age (years), mean $\pm$ SD	58.4 $\pm$ 10.6
Age ≥ 60 years, n (%)	46 (51.1)

Male gender, n (%)	61 (67.8)
Female gender, n (%)	29 (32.2)
Body mass index (kg/m ²), mean ± SD	26.1 ± 3.8
Diabetes mellitus, n (%)	31 (34.4)
Hypertension, n (%)	55 (61.1)
Chronic kidney disease, n (%)	8 (8.9)
Left ventricular ejection fraction (%), mean ± SD	54.2 ± 7.9
CABG procedure, n (%)	49 (54.4)
Valve surgery, n (%)	29 (32.2)
Combined procedures, n (%)	12 (13.4)

The mean age of the study population was, with a predominance of male patients (67.8%). Hypertension was the most common comorbidity (61.1%), followed by diabetes

mellitus (34.4%). Coronary artery bypass grafting was the most frequently performed procedure (54.4%), followed by valve surgery.

Table 2. Intraoperative Characteristics and Cardiopulmonary Bypass Parameters (N = 90)

Variables	Mean ± SD / n (%)
CPB duration (minutes)	112.6 ± 28.4
CPB duration >120 minutes, n (%)	38 (42.2)
Aortic cross-clamp time (minutes)	74.5 ± 21.7
Intraoperative blood transfusion, n (%)	27 (30.0)
Use of vasoactive drugs during surgery, n (%)	41 (45.6)
Lowest haemoglobin during CPB (g/dL)	8.9 ± 1.1
Lowest temperature during CPB (°C)	32.1 ± 1.8

The mean duration of CPB was 112.6 ± 28.4 minutes, while the mean aortic cross-clamp time was 74.5 ± 21.7 minutes. Nearly half of the patients required intraoperative vasoactive

support (45.6%), reflecting the haemodynamic challenges associated with CPB-assisted procedures.

Table 3. Perioperative Serum Lactate and Base Deficit Changes Among Study Participants

Time point	Serum lactate (mmol/L) Mean ± SD	Base deficit (mmol/L) Mean ± SD
Pre-CPB baseline	1.2 ± 0.4	-1.8 ± 1.2
During CPB	2.4 ± 0.9	-4.1 ± 1.8
End of CPB	3.1 ± 1.2	-5.6 ± 2.3
ICU admission	3.6 ± 1.5	-6.2 ± 2.6
6 hours postoperative	2.1 ± 0.8	-3.4 ± 1.7

Serum lactate levels demonstrated a progressive increase during CPB, reaching the highest value at ICU admission (3.6 ± 1.5 mmol/L). Similarly, base deficit worsened during the intraoperative period, with

maximum metabolic derangement observed immediately after surgery (−6.2 ± 2.6 mmol/L), followed by gradual correction during the postoperative period.

Table 4. Comparison Of Metabolic Markers Between Patients with and Without Postoperative Complications

Parameters	Complication group (n=32)	No complication group (n=58)	p value
Peak lactate (mmol/L)	5.1 ± 1.4	2.8 ± 0.9	<0.001

Maximum base deficit (mmol/L)	-8.1 ± 2.5	-5.1 ± 1.9	<0.001
CPB duration (minutes)	132.5 ± 31.6	101.7 ± 22.9	<0.001
ICU stay (days)	5.8 ± 2.1	3.2 ± 1.4	<0.001
Mechanical ventilation duration (hours)	28.4 ± 13.6	14.7 ± 7.5	<0.001

Patients who developed postoperative complications had significantly higher peak lactate concentrations and greater base deficit compared with patients without complications.

Longer CPB duration, prolonged ICU stay, and extended mechanical ventilation were also significantly associated with adverse outcomes.

Table 5. Postoperative Complications Observed During Early Follow-Up (N = 90)

Postoperative outcomes	n (%)
Any major complication	32 (35.6)
Acute kidney injury (KDIGO criteria)	15 (16.7)
Prolonged mechanical ventilation (>24 h)	18 (20.0)
Low cardiac output syndrome	12 (13.3)
Postoperative arrhythmia	14 (15.6)
Re-exploration for bleeding	5 (5.6)
ICU stay >5 days	21 (23.3)
In-hospital mortality	4 (4.4)

Early postoperative complications occurred in 35.6% of patients. Acute kidney injury was observed in 16.7%, while prolonged

mechanical ventilation was required in 20% of patients. Overall, in-hospital mortality was 4.4%.

Table 6. Predictive Performance of Serum Lactate and Base Deficit for Early Postoperative Complications

Biomarker	Cut-off value	AUC (95% CI)	Sensitivity (%)	Specificity (%)	p value
Peak lactate	>4.0 mmol/L	0.84 (0.75–0.92)	81.3	79.3	<0.001
Maximum base deficit	<-7 mmol/L	0.79 (0.69–0.88)	75.0	77.6	<0.001
Combined lactate + base deficit	—	0.89 (0.82–0.95)	87.5	82.8	<0.001

ROC curve analysis demonstrated that both peak lactate and maximum base deficit were significant predictors of early postoperative complications. The combined assessment of

lactate and base deficit showed superior predictive ability with an AUC of 0.89, indicating excellent discrimination.

Figure 1. Serial Changes in Serum Lactate Concentration Before and After Cardiopulmonary Bypass

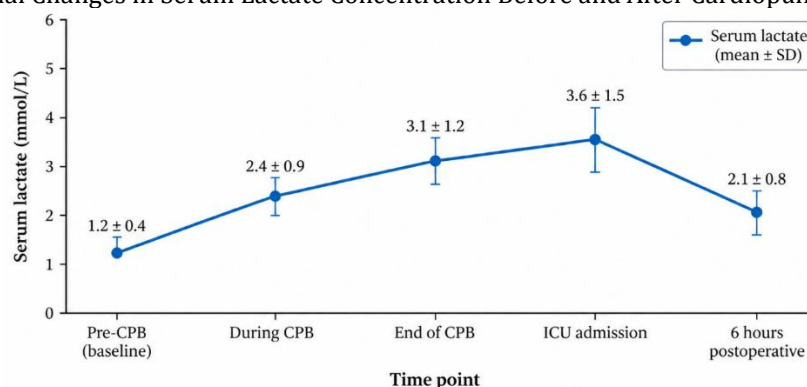


Figure 1 illustrates the temporal variation in serum lactate concentrations at different perioperative time points among patients undergoing cardiopulmonary bypass-assisted cardiac surgery. The mean baseline serum lactate level before initiation of CPB was 1.2 ± 0.4 mmol/L. Lactate levels progressively increased during CPB (2.4 ± 0.9 mmol/L) and further increased at the end of CPB (3.1 ± 1.2 mmol/L), indicating the development of metabolic stress associated with

extracorporeal circulation. The highest serum lactate concentration was observed at ICU admission (3.6 ± 1.5 mmol/L), reflecting the immediate postoperative impact of CPB-related alterations in oxygen delivery and tissue perfusion. Subsequently, lactate levels demonstrated a declining trend at 6 hours postoperatively (2.1 ± 0.8 mmol/L), suggesting gradual recovery of metabolic homeostasis after restoration of effective circulation.

Figure 2. Comparison Of Peak Serum Lactate Levels Between Patients with and Without Postoperative Complications

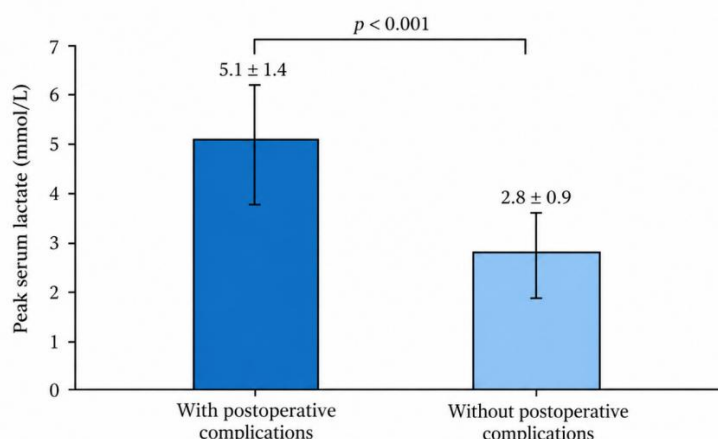


Figure 2 compares the peak serum lactate levels between patients who developed postoperative complications and those who did not following cardiopulmonary bypass-assisted cardiac surgery. Patients with postoperative complications showed a markedly higher mean

peak serum lactate concentration (5.1 ± 1.4 mmol/L) compared to patients without complications (2.8 ± 0.9 mmol/L). The difference between the two groups was statistically highly significant ($p < 0.001$).

Figure 3. ROC Curve Analysis of Serum Lactate and Base Deficit for Prediction of Early Postoperative Complications After CPB

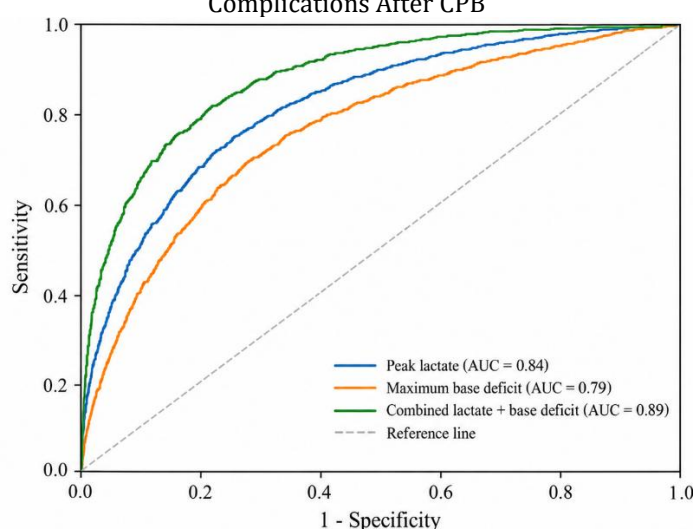


Figure 3 illustrates the receiver operating characteristic (ROC) curve analysis evaluating

the predictive performance of peak serum lactate, maximum base deficit, and their

combined assessment for early postoperative complications following cardiopulmonary bypass. Peak serum lactate showed good predictive ability with an area under the curve (AUC) of 0.84, while maximum base deficit also demonstrated significant predictive accuracy with an AUC of 0.79. The combined evaluation of serum lactate and base deficit provided the best discriminatory performance, with an AUC of 0.89, indicating superior ability to identify patients at risk of adverse early postoperative outcomes.

DISCUSSION

Cardiopulmonary bypass (CPB) remains an essential component of contemporary cardiac surgery; however, it is associated with significant metabolic disturbances resulting from alterations in oxygen delivery, systemic inflammatory activation, haemodilution, and temporary impairment of tissue perfusion. Early identification of patients at risk of postoperative complications is crucial for improving outcomes, and metabolic biomarkers such as serum lactate and base deficit have gained importance as readily available indicators of physiological stress. In the present study involving 90 patients undergoing CPB-assisted cardiac surgery, elevated peak serum lactate and worsening base deficit were significantly associated with early postoperative complications. Furthermore, combined assessment of lactate and base deficit demonstrated superior predictive performance for adverse outcomes, highlighting their potential role in perioperative risk stratification.

In this study, serum lactate levels demonstrated a progressive increase from baseline values before CPB (1.2 ± 0.4 mmol/L) to peak levels at ICU admission (3.6 ± 1.5 mmol/L), followed by a decline during the early postoperative period. This pattern reflects the metabolic stress induced by extracorporeal circulation and subsequent recovery after restoration of adequate systemic perfusion. Similar observations were reported by Ranucci et al., who demonstrated that hyperlactatemia during CPB was associated with increased postoperative morbidity and mortality, particularly when lactate elevation persisted beyond the intraoperative period [10]. Elevated lactate during cardiac surgery may indicate inadequate oxygen delivery relative to metabolic demand, and its persistence after

CPB may represent ongoing tissue hypoperfusion or impaired lactate clearance.

The present study demonstrated that patients who developed postoperative complications had significantly higher peak lactate concentrations compared with those without complications (5.1 ± 1.4 vs 2.8 ± 0.9 mmol/L, $p < 0.001$). These findings support previous evidence that perioperative hyperlactatemia is an important predictor of adverse outcomes after cardiac surgery. Hajjar et al. reported that elevated lactate levels following cardiac surgery were independently associated with increased mortality and prolonged ICU stay, suggesting that lactate monitoring provides valuable prognostic information beyond conventional clinical parameters [11]. Similarly, Minton and Sidebotham emphasized that hyperlactatemia after cardiac surgery is multifactorial but consistently correlates with increased risk of complications, including prolonged mechanical ventilation, renal dysfunction, and haemodynamic instability [12].

The association between increased lactate levels and adverse outcomes observed in our study may be explained by several mechanisms. CPB can impair microcirculatory flow and alter oxygen utilization despite apparently adequate systemic haemodynamics. Factors such as prolonged bypass duration, haemodilution, reduced haemoglobin concentration, and inflammatory mediator release may contribute to oxygen debt and increased anaerobic metabolism. In the current cohort, patients with complications also demonstrated significantly longer CPB duration (132.5 ± 31.6 minutes) compared with those without complications (101.7 ± 22.9 minutes), supporting the relationship between prolonged extracorporeal circulation and metabolic disturbance. Ranucci et al. previously demonstrated that reduced oxygen delivery during CPB and prolonged bypass exposure were important determinants of lactate production and postoperative outcomes [13].

In addition to lactate, this study evaluated base deficit as another marker of metabolic derangement. Maximum base deficit was significantly greater among patients with postoperative complications (-8.1 ± 2.5 mmol/L) compared with uncomplicated patients (-5.1 ± 1.9 mmol/L, $p < 0.001$). Base deficit reflects the magnitude of metabolic acidosis and provides an integrated assessment of cumulative oxygen debt, lactate

accumulation, and buffering capacity. Unlike lactate alone, base deficit incorporates multiple components of metabolic imbalance and may therefore provide additional prognostic information. Zante et al. demonstrated that base excess abnormalities were strongly associated with ICU mortality after cardiac surgery and suggested that acid–base parameters may complement conventional monitoring approaches [14].

The prognostic value of combining lactate and base deficit was an important finding of the present study. ROC curve analysis demonstrated that peak lactate alone had an AUC of 0.84, while maximum base deficit showed an AUC of 0.79. However, combined assessment improved predictive accuracy, achieving an AUC of 0.89 with higher sensitivity and specificity. This suggests that lactate and base deficit provide complementary information regarding metabolic stress after CPB. Lactate primarily reflects increased anaerobic metabolism, whereas base deficit reflects the overall severity of metabolic acidosis. Therefore, integrating both parameters may improve early identification of patients requiring intensified postoperative surveillance.

The occurrence of postoperative complications in the present study was 35.6%, including acute kidney injury (16.7%), prolonged mechanical ventilation (20%), low cardiac output syndrome (13.3%), and postoperative arrhythmias (15.6%). These complications are consistent with the known consequences of inadequate oxygen delivery and systemic inflammatory responses following CPB. Previous studies have highlighted that early metabolic abnormalities frequently precede clinically apparent organ dysfunction, supporting the importance of continuous biochemical monitoring during the perioperative period [15]. Early recognition of abnormal lactate and base deficit trends may allow clinicians to optimize haemodynamics, improve oxygen delivery, and prevent progression to irreversible organ injury.

The clinical applicability of lactate and base deficit measurement is strengthened by their rapid availability through routine arterial blood gas analysis. Unlike more complex biomarkers, these parameters can be assessed repeatedly during and after surgery, allowing dynamic monitoring of patient status. However, interpretation should consider potential confounding factors, including catecholamine administration, liver dysfunction, renal

impairment, and differences in surgical complexity. Lactate elevation does not always represent tissue hypoxia alone, and clinical correlation remains essential.

The present study has several limitations. The relatively small sample size and single-centre observational design may limit generalizability of the findings. Additionally, long-term outcomes beyond the early postoperative period were not evaluated. Future multicentre studies with larger populations are required to validate optimal lactate and base deficit thresholds and determine whether biomarker-guided interventions can improve postoperative outcomes.

CONCLUSION

The present study demonstrates that serum lactate and base deficit are valuable early predictors of postoperative outcomes in patients undergoing cardiopulmonary bypass-assisted cardiac surgery. A progressive increase in serum lactate levels and worsening base deficit during the perioperative period were significantly associated with the development of early postoperative complications, including prolonged mechanical ventilation, acute kidney injury, prolonged ICU stay, and haemodynamic instability. Peak serum lactate showed good predictive ability for adverse outcomes, while maximum base deficit provided additional information regarding the severity of metabolic disturbance and tissue oxygen imbalance. Importantly, the combined assessment of lactate and base deficit demonstrated superior prognostic performance compared with either parameter alone, suggesting that their integration may improve early risk stratification after CPB.

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