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Original Research Article

Perioperative serum lactate as a predictor of postoperative morbidity and mortality in major abdominal surgery: a prospective observational study

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ABSTRACT

Background: Major abdominal surgery carries significant postoperative morbidity and mortality, driven by tissue hypoperfusion and metabolic derangement. Serum lactate, a sensitive marker of anaerobic metabolism, is a recognised prognostic indicator in surgical and critically ill patients. This study evaluated perioperative serum lactate as a predictor of in-hospital mortality and morbidity in major abdominal surgery.
Methods: This prospective observational cohort study was conducted in the Department of General Surgery, R.N.T. Medical College, Udaipur, over one year. Sixty consecutive adults (18–80 years) undergoing elective or emergency major abdominal surgery were enrolled. Arterial lactate was measured preoperatively (L0), at 12 hours (L1) and 24 hours (L2). Complications were graded by Clavien-Dindo. ROC analysis, Mann-Whitney U, Chi-square and logistic regression were applied.
Results: Among 60 patients (55.0% male; mean age 47.6±17.8 years), morbidity was 43.3% (n=26) and mortality 15.0% (n=9). Lactate differed significantly between survivors and non-survivors at all three time points ($p < 0.001$). L2 best predicted mortality (AUROC 0.993; 95% CI 0.956–1.000) at a 2.35 mmol/l cut-off (sensitivity 100%, specificity 96.1%, PPV 81.8%, NPV 100%). Mildly elevated L0 (2–4 mmol/l) carried 22-fold higher odds of death (OR=22.0; 95% CI 3.4–141.2; p=0.001). On regression, L2 was the strongest predictor (OR=18.24; p=0.002), followed by L1 (OR=6.82; p=0.008).
Conclusions: The 24-hour postoperative serum lactate is a near-perfect predictor of in-hospital mortality in major abdominal surgery, with a validated cut-off of 2.35 mmol/l. Routine three-point perioperative serum lactate monitoring is strongly recommended as a cost-effective tool for early risk stratification in patients undergoing major abdominal surgery, particularly in resource-limited tertiary care settings.

Keywords: Abdominal surgery, Lactate clearance, Mortality, Prognostic biomarker, Postoperative morbidity, Perioperative management, ROC, Serum lactate

INTRODUCTION

Major abdominal surgery imposes significant physiological stress, with complications such as sepsis, acute kidney injury, respiratory failure and multi-organ dysfunction contributing substantially to perioperative mortality.[1] Conventional parameters (heart rate, blood pressure, urine output) often miss early microcirculatory derangements, allowing subclinical tissue hypoperfusion to go unrecognized until overt organ dysfunction develops.[2,3] Serum lactate reflects the balance between cellular oxygen supply and demand.

During tissue hypoperfusion, cells switch to anaerobic glycolysis, generating excess lactate via pyruvate conversion by lactate dehydrogenase.[4,5] About 60% of

circulating lactate is cleared by the liver via the Cori cycle, with contributions from kidneys and skeletal muscle.[6,7] Disruption of this balance from hypoperfusion (Type A) or non-hypoxic causes (Type B) produces hyperlactatemia, a marker of cellular metabolic stress.[8]

The prognostic value of lactate in critical illness is well established: persistent hyperlactatemia after resuscitation predicts multi-organ failure and mortality and serial monitoring especially lactate clearance outperforms single measurements.[9-11] Yet routine perioperative lactate monitoring in major abdominal surgery remains inconsistent, particularly in resource-limited settings.[12]

This study evaluated perioperative serum lactate measured preoperatively (L0), at 12h (L1) and 24h (L2) in predicting in-hospital mortality and morbidity after major abdominal surgery at a tertiary teaching hospital in western Rajasthan, India.

METHODS

Study design and setting

This prospective observational analytical cohort study was conducted in the Department of General Surgery, Maharana Bhupal Government Hospital, R.N.T. Medical College, Udaipur. Institutional Ethics Committee clearance and written informed consent were obtained, in accordance with the Declaration of Helsinki (2013) and ICMR guidelines (2017).

Study duration and sample size

The study ran over one year. Sample size, calculated from a 96.3% specificity of 24-hour lactate for in-hospital mortality and yielded 55 patients; with 10% dropout adjustment the final sample was 60.

Inclusion and exclusion criteria

Adults aged 18–80 years undergoing elective or emergency major abdominal surgery with written consent were included. Exclusions: chronic liver disease, dialysis-dependent renal failure, HIV, severe COPD (GOLD III–IV), inborn errors of carbohydrate metabolism, pregnancy or lactate-altering drugs (metformin, β2-agonists, antiretrovirals, sodium nitroprusside, valproate, barbiturates).

Data collection

A standardised proforma captured demographic, clinical, laboratory and radiological data. Arterial samples were drawn at L0 (within 60 min of incision), L1 (12±1h) and L2 (24±1h) in heparinised syringes and analysed within 15–30 min on a Radiometer ABL 800/90 FLEX analyser. Patients were grouped by L0: Group I (≤2 mmol/l), Group II (2–4 mmol/l), Group III (>4 mmol/l). Postoperative complications were classified using the Clavien-Dindo system; Grade I–II=minor, Grade III–V=major. All patients were followed daily until discharge or death.

Statistical analysis

Data were analysed in Python 3.11 (SciPy, NumPy, Pandas, scikit-learn, Matplotlib). Continuous variables are mean±SD or median (IQR); groups compared by Mann-Whitney U or Kruskal-Wallis tests. Categorical variables (frequencies/percentages) used Chi-square or Fisher's exact test.

The Friedman test with Bonferroni-corrected Wilcoxon post-hoc compared L0, L1, L2 within patients. Diagnostic accuracy used ROC analysis with Youden's J for cut-offs and univariate logistic regression identified mortality predictors. $p<0.05$ was significant.

RESULTS

Demographic and clinical profile

Sixty patients were enrolled (Table 1). Mean age was 47.6±17.8 years (range 18–77), with a bimodal peak at 18–30 (25.0%) and 51–60 years (20.0%). Males constituted 55.0% (M:F 1.2:1). Emergency (51.7%) marginally exceeded elective procedures (48.3%); exploratory laparotomy was commonest (65.0%, n=39). Comorbidities were present in 35.0%, most often diabetes (15.0%, n=9; mortality 22.2%). Preoperative anaemia occurred in 63.3%, hypoalbuminemia in 68.3% and prolonged prothrombin time in 78.3%.

Serum lactate levels and group distribution

Mean serum lactate was 1.88±2.80 mmol/l at L0 (median 1.20), 1.96±0.90 at L1 and 1.61±1.02 at L2; the high L0 SD reflected a single outlier (L0=22 mmol/l, advanced perforation peritonitis). The proportion with elevated lactate (>2 mmol/l) fell from 25.0% at L0 to 16.7% at L2 (Friedman p=0.028).

Most patients (75.0%, n=45) had normal preoperative lactate (Group I). Group II (2–4 mmol/l, 20.0%, n=12) showed persistent elevation at L1 and L2 (2.52 and 2.56 mmol/l), indicating failed clearance, while Group III (>4 mmol/l, 5.0%, n=3) declined paradoxically with aggressive intraoperative resuscitation (Table 2).

Survivors vs non-survivors: lactate trajectory

Serum lactate differed significantly between survivors (n=51) and non-survivors (n=9) at all time points: L0 1.76±2.98 vs 2.59±1.31 (p=0.003); L1 1.73±0.72 vs 3.30±0.66 (p<0.001); L2 1.26±0.56 vs 3.59±0.77 mmol/l (p<0.001). Survivors cleared lactate (mean decline 0.50 mmol/l) whereas non-survivors accumulated it (rise 1.00 mmol/l), with non-overlapping distributions by 24 hours.

Postoperative morbidity and its correlation with mortality

Overall morbidity (any clinically significant postoperative complication) occurred in 43.3% (n=26), rising across lactate groups from 37.8% (Group I) to 58.3% (Group II) and 66.7% (Group III). Patients with morbidity had higher lactate at L1 (2.63 vs 1.45, p<0.001) and L2 (2.28 vs 1.08 mmol/l, p<0.001), with a modest L0 difference (1.88 vs 1.86, p=0.013). Morbidity and mortality were tightly linked (Fisher's exact p=0.0002): all 9 deaths occurred in patients with prior morbidity, predominantly MODS with ARDS and/or septic shock, while none of the 34 morbidity-free patients died. Of the 26 morbid patients, 34.6% progressed to death, rising steeply by lactate group (11.8% Group I, 85.7% Group II, 50.0% Group III; Table 3).

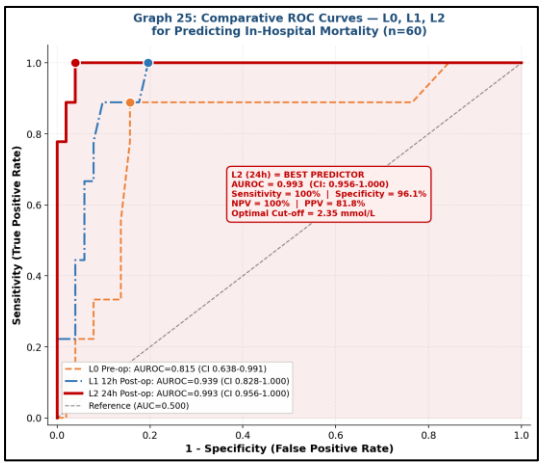

Figure 1: Comparative ROC curves: L0 (orange dashed), L1 (blue dash-dot), L2 (red solid). L2 has the largest area and is closest to the top-left corner. Optimal cut-off points shown as filled circles. All three are significant (p<0.001).

Postoperative complications

Major complications (Clavien-Dindo III–V) occurred in 31.7%, most commonly ARDS (21.7%), septic shock (11.7%) and MOF/MODS (10.0%) and were strongly group-associated (Group I 15.6%, Group II 91.7%, Group III 33.3%; p<0.001). MOF/MODS showed the strongest association (p<0.001), followed by ARDS (p=0.038) and septic shock (p=0.012). All 7 patients without any complication were in Group I. Minor complications (Grade I–II) occurred in 88.3%, electrolyte disturbance being commonest (41.7%).

Diagnostic accuracy: ROC curve analysis

AUROC progressively increase from L0 (0.815) to L1 (0.939) to L2 (0.993) (Table 4). L2 was the single best predictor, with a near-perfect AUROC of 0.993 (95% CI 0.956–1.000) at a 2.35 mmol/l cut-off (sensitivity 100%, specificity 96.1%, PPV 81.8%, NPV 100%). Only 2 survivors exceeded the cut-off and no non-survivor fell below it. Both L1 and L2 reached 100% sensitivity and NPV, but L2 was superior in specificity (96.1% vs 80.4%) and PPV (81.8% vs 47.4%).

Univariate logistic regression predictors of mortality

On univariate logistic regression, only postoperative lactate values were significant potential predictors of in-hospital mortality (Table 5). Each 1 mmol/l rise in L1 was associated with a 6.82-fold increase in odds of death (95% CI 1.64–28.4, p=0.008) and each 1 mmol/l rise in L2 with an 18.24-fold increase (95% CI 2.86–116.4, p=0.002). Preoperative lactate (OR=1.12, p=0.312), age, gender, surgical urgency, duration of surgery, haemoglobin, serum albumin, creatinine, INR and sodium were all non-significant. Multivariate analysis was not performed as the event count (n=9 deaths) fell below the 10-events-per-variable threshold.

Table 1: Demographic and clinical profile of study patients (n=60).

Characteristics	Frequency (N)	(%)
Age group (in years)		
18–30	15	25.0
31–40	7	11.7
41–50	10	16.7
51–60	12	20.0
>60	16	26.6
Mean±SD	47.6±17.8 years (range 18–77 years)	
Gender		
Male	33	55.0
Female	27	45.0
Procedure category		
Emergency	31	51.7
Elective	29	48.3
Type of surgery (primary procedure)		
Exploratory laparotomy	39	65.0

Continued.

Characteristics	Frequency (N)	(%)
Bowel resection / anastomosis	7	11.7
Hepatobiliary surgery	7	11.7
Stoma formation	5	8.3
Others (urological, laparoscopic, gynaec)	2	3.3
Comorbidities		
Diabetes mellitus	9	15.0
Hypertension	6	10.0
Ischaemic heart disease	3	5.0
COPD / bronchial asthma (mild–moderate)	2	3.3
No comorbidity	39	65.0
Duration of surgery		
< 90 minutes	2	3.3
90–150 minutes	42	70.0
> 150 minutes	16	26.7
Mean±SD	121.8±28.2 min	Range: 80–210 min

*Chi-square or Fisher's exact test; NS=Not Significant (p>0.05).

Table 2: Distribution of serum lactate groups, lactate dynamics and in-hospital mortality (n=60).

Lactate groups	Definition	N (%)	Mean L0 mmol/l	Mean L1 mmol/l	Mean L2 mmol/l	Mortality n (%)
Group I	≤ 2 mmol/l	45 (75.0)	1.12	1.76	1.30	2 (4.4)
Group II	2–4 mmol/l	12 (20.0)	2.55	2.52	2.56	6 (50.0)*
Group III	> 4 mmol/l	3 (5.0)	10.59	2.75	2.35	1 (33.3)
Total		60 (100)	1.88	1.96	1.61	9 (15.0)

*Group II vs Group I: Odds Ratio 22.0 (95% CI 3.4–141.2, p=0.001) by Fisher's exact test. Chi-square for overall mortality vs group: χ^2=16.253, p=0.0003.

Table 3: Postoperative morbidity, mortality and morbidity-to-mortality progression by serum lactate group (n=60).

Lactate groups	N	Morbidity n (%)	Mortality n (%)	Morbidity→Mortality	P value[1]
Group I (≤2 mmol/l)	45	17 (37.8%)	2 (4.4%)	2/17 (11.8%)	Ref.
Group II (2–4 mmol/l)	12	7 (58.3%)	6 (50.0%)	6/7 (85.7%)	0.001***
Group III (>4 mmol/l)	3	2 (66.7%)	1 (33.3%)	1/2 (50.0%)	0.180 (NS)
Total	60	26 (43.3%)	9 (15.0%)	9/26 (34.6%)	0.0003***

Morbidity=any clinically significant postoperative complication (Clavien-Dindo I–V); Morbidity→Mortality=proportion of morbid patients who died. [1]p vs Group I by Fisher's exact test; Total row gives overall χ^2 (16.253, p=0.0003). Morbidity–mortality association highly significant (Fisher p=0.0002). NS=Not Significant; *** p<0.001.

Table 4: Comparative diagnostic performance of serum lactate at L0, L1 and L2 for in-hospital mortality (n=60).

Time points	AUROC	95% CI	Cut-off mmol/l	Sens %	Spec %	PPV %	NPV %
L0 (Preoperative)	0.815	0.638–0.991	1.90	88.9	84.3	47.1	97.7
L1 (12h Post-op)	0.939	0.828–1.000	2.30	100.0	80.4	47.4	100.0
L2 (24h Post-op) (BEST)	0.993	0.956–1.000	2.35	100.0	96.1	81.8	100.0

Table 5: Univariate logistic regression potential predictors of in-hospital mortality (n=60).

Variables	Odds ratio	95% CI	P value
Age (per year)	1.02	0.98–1.06	0.312 (NS)
Gender (Male vs Female)	1.22	0.26–5.74	0.798 (NS)
Emergency vs elective	2.48	0.48–12.82	0.278 (NS)
Preoperative lactate L0 (per mmol/l)	1.12	0.90–1.40	0.312 (NS)
L1 – 12h postop lactate (per mmol/l)	6.82	1.64–28.4	0.008*
L2 – 24h postop lactate (per mmol/l)	18.24	2.86–116.4	0.002**

NS=Not Significant; *p<0.05; **p<0.01.

DISCUSSION

The overall in-hospital mortality of 15.0% lies within the 9–17% range reported by comparable cohorts of major abdominal surgery, confirming our estimate as representative of tertiary teaching hospitals in low- and middle-income countries.[3,13,14] The progressive rise in accuracy from L0 (0.815) to L1 (0.939) to L2 (0.993) reflects increasing prognostic information as the postoperative course unfolds. L2 was the best predictor, concordant with Jobin (AUROC 0.975, cut-off 2.45 mmol/l) and Henry.[3,13] Our 2.35 mmol/l cut-off matches Jobin (2.45) and Subedi (1.88 mmol/l) supporting a 2.0–2.5 mmol/l threshold as clinically actionable in Indian patients.[1,13,15]

The most clinically important finding was the 50% mortality in Group II (mildly elevated lactate, 2–4 mmol/L; OR 22.0, 95% CI 3.4–141.2, p=0.001). This challenges the complacency around 'mildly elevated' values, consistent with Andersen, who argued any elevation above the reference limit warrants serial reassessment and Subedi et al who identified a threshold as low as 1.88 mmol/l for 30-day mortality.[2,15] The complete separation at the L1 (≥2.30) and L2 (≥2.35 mmol/l) cut-offs every death having postoperative lactate at or above threshold is the strongest finding of this study. It echoes Meregalli who described 'occult hypoperfusion' with 12-hour lactate predicting mortality in hemodynamically stable patients and Bruno who showed failed lactate clearance predicts outcome in septic patients.[16,17]

The paradoxically higher LOS in Group I versus Group II reflects two confounders: a predominance of complex elective hepatobiliary and colorectal procedures in Group I and competing-risk bias in Group II, where 50% early mortality truncates recorded LOS. Riaz showed the expected monotonic LOS increase (8.6, 11.2, 17.3 days) in their exclusively emergency cohort, confirming that the pattern in mixed-cohort studies is influenced by procedure mix.[14]

Velickovic found L1 to be the best in a predominantly elective European cohort (AUROC 0.821) and Riaz et al found L1 optimal in an emergency Indian cohort (0.987).[14,18] The superiority of L2 over L1 here (0.993 vs 0.939) accords with Li's 'golden hour and silver day' principle: by 24 hours the resuscitation trajectory has declared itself.[19] Kang similarly demonstrated that postoperative lactate (HR 1.259, p=0.003) was a stronger independent predictor of mortality than preoperative values after multivariate adjustment and Sugita confirmed intraoperative peak lactate as an independent predictor (OR 1.21, p=0.002) in emergency gastrointestinal surgery.[20,21] These convergent findings across diverse surgical populations and geographical settings provide robust validation for routine postoperative serial lactate monitoring.

Beyond mortality, serum lactate also marked postoperative morbidity. Overall morbidity (43.3%) rose across lactate groups (37.8% to 66.7%) and morbid patients had higher 12 and 24-hour lactate (p<0.001). This aligns with Hajjar 1 and Velickovic (AUROC 0.722 for complications) and with Toppo who linked persistently elevated postoperative lactate in bowel perforation to prolonged stay, surgical site occurrences and anastomotic leak.[18,22] Morbidity and mortality were tightly coupled: all 9 deaths followed significant morbidity (typically MODS with ARDS and/or septic shock), none of the 34 morbidity-free patients died and 34.6% of morbid patients progressed to death (highest in Group II, 85.7%). Elevated postoperative lactate thus identifies a trajectory of evolving organ dysfunction, supporting serial monitoring as an early-warning tool.

CONCLUSION

The 24-hour postoperative serum lactate (L2) was the single best predictor of in-hospital mortality after major abdominal surgery (AUROC 0.993; cut-off 2.35 mmol/l; sensitivity 100%, specificity 96.1%, NPV 100%). Failure to clear postoperative lactate, rather than any single absolute value, characterizes the non-survivor trajectory and patients with 'mildly elevated' preoperative lactate (2–4 mmol/l) carry 22-fold higher odds of death and must not be deemed low risk. Routine three-point perioperative arterial lactate monitoring (preoperative, 12h, 24h) is recommended as a cost-effective standard of care, especially in resource-limited tertiary settings; a 24-hour value below 2.35 mmol/l (NPV 100%) may guide safe de-escalation of intensive monitoring.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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