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Serum Lactate, Anion Gap and Sequential Organ Failure Assessment Score as Prognostic Markers in Septic Shock: A Prospective Observational Study of 100 Patients.

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Abstract

Background: Rapid risk stratification in septic shock depends on markers that are inexpensive, immediately available and reproducible. Serum lactate, the calculated anion gap and the Sequential Organ Failure Assessment (SOFA) score each satisfy these criteria, but their comparative performance within a single Indian tertiary care cohort has not been well described. Objective: To evaluate admission serum lactate, anion gap and SOFA score as predictors of in-hospital mortality in critically ill patients with septic shock, and to characterise the demographic and microbiological source profile of this population. ***Methods***: One hundred consecutive adults meeting Surviving Sepsis Campaign criteria for sepsis and septic shock were enrolled prospectively in the Medical Intensive Care Pregnancy, malignancy, treated HIV infection and chronic liver disease were exclusions. Arterial blood gas analysis including lactate, together with serum electrolytes permitting anion gap calculation, was performed within 24 hours of admission, and the SOFA score was computed from concurrent clinical and laboratory variables. Patients were followed to hospital discharge or in-hospital death. Analysis used SPSS version 20 with non-parametric t-test and analysis of variance; $p < 0.05$ was significant. ***Results***: In-hospital mortality was 53%. Mean SOFA score was 3.6 in survivors versus 11.7 ± 3.6 in non-survivors ($p < 0.001$); no patient with a SOFA score above 10 survived, whereas 32 of 36 patients scoring below 5 were discharged alive. Mean serum lactate was 1.6 ± 0.50 mmol/L in survivors versus 4.3 ± 2.0 mmol/L in non-survivors ($p < 0.001$), with 38 of 53 deaths (71.7%) occurring among patients whose lactate exceeded 6 mmol/L. Mean anion gap was 11.3 ± 3.2 mEq/L in survivors versus 22.5 ± 6.1 mEq/L in non-survivors ($p < 0.001$); of the 50 patients with an anion gap above 16 mEq/L, 47 died, giving a mortality of 94% within that stratum, against 12% among those with a normal gap. Lower respiratory tract infection was the commonest source of sepsis (42%). ***Conclusion***: Admission SOFA score, serum lactate and anion gap each discriminated powerfully between survivors and non-survivors. A high anion gap, reflecting predominantly lactic acidosis, showed near-dichotomous separation and, being derived from routine electrolytes, offers an accessible bedside adjunct to lactate measurement where the latter is unavailable.

Keywords: Septic shock; Serum lactate; Anion gap; SOFA score; Lactic acidosis; Mortality prediction

INTRODUCTION

Septic shock is a subset of sepsis in which circulatory, cellular and metabolic derangements are sufficiently profound to substantially raise the risk of death, and it is identified clinically by persistent hypotension requiring vasopressors to maintain a mean arterial pressure of 65 mmHg or above together with a serum lactate exceeding 2 mmol/L in the absence of hypovolaemia [1]. The condition carries an in-hospital mortality that exceeds 40% in most reported series, and a delay in recognition permits rapid progression to multiple organ dysfunction [2]. Because the therapeutic window is narrow, the identification of markers that stratify risk within the first hours of presentation has become a central concern of critical care research.

Lactate occupies a privileged position among these markers. It is produced largely by anaerobic glycolysis from the reduction of pyruvate, and cleared principally by the liver and kidney; in septic shock complicated by hepatic dysfunction or acute kidney injury, impaired clearance compounds increased production [3]. Levraut and colleagues demonstrated that mild hyperlactataemia in stable septic patients arises predominantly from reduced clearance rather than overproduction [4], while Gore et al. showed that lactic acidosis during sepsis relates to increased pyruvate generation rather than a deficit in

tissue oxygen availability [5]. James and colleagues went further, cautioning that lactate is an unreliable direct indicator of tissue hypoxia in injury and sepsis [6]. Whatever its precise mechanism, its prognostic value is not in dispute: Bakker et al. showed that blood lactate outperformed oxygen-derived variables in predicting outcome in human septic shock [7] and that serial levels predicted the subsequent development of multiple organ failure [8]. Nguyen and colleagues established that early lactate clearance is associated with improved outcome in severe sepsis and septic shock [9], and lactate measurement is now embedded within the Surviving Sepsis Campaign hour-1 bundle [10].

The serum anion gap, calculated as sodium minus the sum of chloride and bicarbonate, offers an indirect route to the same information. Because lactate is an unmeasured anion, its accumulation widens the gap, and Berkman and colleagues proposed the anion gap as a screening tool for elevated lactate in patients at risk of sepsis presenting to the emergency department [11]. The attraction is practical: serum electrolytes are universally available and inexpensive, whereas point-of-care lactate assays are not uniformly accessible in resource-constrained settings. The gap has recognised limitations, chief among them its dependence on serum albumin, the largest unmeasured anion in health, such that hypoalbuminaemia common in critical illness may mask a genuine elevation [12,13].

Organ dysfunction scoring provides a complementary, physiology-based approach. The SOFA score, developed by Vincent and colleagues, grades dysfunction from 0 to 4 across six organ systems and yields a composite from 0 to 24 [14]. Ferreira et al. demonstrated in serial evaluation that both initial and highest SOFA scores predict outcome in critically ill patients, with rising scores over the first 48 hours signalling a mortality above 50% [15]. The Sepsis-3 definitions incorporated an acute rise of two or more SOFA points as the operational criterion for sepsis itself [1]. Its principal drawback is computational burden, requiring several laboratory variables and treatment-dependent inputs.

These three markers therefore differ in mechanism, in cost and in convenience, yet all have been proposed for early risk stratification. The present study was undertaken to evaluate them within a single prospectively recruited cohort of septic shock patients at an Indian tertiary care teaching hospital, and to describe the demographic profile and infective sources characterising this population.

MATERIALS AND METHODS

Study design and setting. A prospective observational study was carried out in the Medical Intensive Care Unit (MICU) The unit serves as a referral intensive care facility for a substantial catchment population and admits patients both directly from the emergency department and by internal transfer.

Study duration. Enrolment and follow-up were conducted over twelve consecutive months, from January 2018 to December 2018.

Sample size estimation. The sample was calculated using the formula $4pq/d^2$, in which p denoted the institutional prevalence of community-acquired pneumonia over the preceding twelve months, $q = 1 - p$ and $d = 1.5$. The computation returned 97, which was rounded upward to a working sample of 100.

Participants. Consecutive adults admitted to the MICU who fulfilled the criteria for sepsis and septic shock defined by the Surviving Sepsis Campaign International Guidelines (2018 update) were screened for eligibility. Inclusion required age above 18 years and written informed consent obtained from the patient or an accompanying relative. Exclusion criteria were pregnancy, diagnosed malignancy, HIV seropositivity with ongoing treatment, and chronic liver disease. The last of these was particularly important for the present analysis, since hepatic dysfunction independently impairs lactate clearance and would have confounded interpretation of admission lactate values.

Data collection. A structured proforma recorded age, sex, presenting complaints, past medical history including diabetes, hypertension, ischaemic heart disease, tuberculosis, thyroid disorder, chronic kidney disease and epilepsy, personal history of smoking and alcohol use, general and systemic examination findings, working diagnosis, presumed source of infection, treatment administered including intravenous fluids, duration of hospital stay and final outcome. Arterial blood gas analysis was performed within 24 hours of MICU admission and yielded pH, pCO_2, bicarbonate, serum lactate, base excess and anion gap. Supporting investigations comprised complete haemogram, liver function tests, renal function tests, serum electrolytes, blood urea, serum creatinine, random blood sugar, urine analysis and electrocardiography. Blood, urine, sputum and pus cultures were obtained as clinically indicated, and the source of infection was assigned on the basis of combined clinical, radiological and microbiological evidence.

Score computation and stratification. The SOFA score was calculated from concurrent respiratory, coagulation, hepatic, cardiovascular, neurological and renal variables and stratified as below 5, 6–10, 11–15 and above 15. Serum lactate was

grouped as 1–5 mmol/L and 6–10 mmol/L. The anion gap was calculated as serum sodium minus the sum of chloride and bicarbonate, and dichotomised at 16 mEq/L.

Outcome measure. The endpoint was in-hospital outcome, classified as either discharge in stable condition as determined by the treating physician, or in-hospital death. Patients were followed from enrolment until one endpoint was reached.

Statistical analysis. Data were compiled and analysed using the Statistical Package for the Social Sciences version 20. Continuous variables are presented as mean ± standard deviation and categorical variables as frequencies with percentages. Comparison between survivor and non-survivor groups employed the non-parametric t-test and analysis of variance where appropriate, and a p value below 0.05 was regarded as statistically significant.

Ethical considerations. Institutional Ethics Committee clearance was obtained from Mysore Medical College and Research Institute prior to commencement of the study. Written informed consent was secured from every participant or attendant after explanation of the study purpose, and patients retained the right to withdraw at any stage without prejudice to their clinical care. Confidentiality of all records was maintained.

RESULTS

Table 1. Demographic characteristics and infective source by outcome (n = 100)

Variable	Survivors (n = 47)	Non-survivors (n = 53)	Total
Age group (years)			
20–40	15 (31.9%)	18 (34.0%)	33
41–60	15 (31.9%)	17 (32.1%)	32
> 61	17 (36.2%)	18 (34.0%)	35
Sex			
Male	19 (40.4%)	31 (58.5%)	50
Female	28 (59.6%)	22 (41.5%)	50
Source of infection			
Lower respiratory tract	16 (34.0%)	26 (49.1%)	42
Urogenital tract	15 (31.9%)	5 (9.4%)	20
Gastrointestinal tract	10 (21.3%)	7 (13.2%)	17
Soft tissue	1 (2.1%)	8 (15.1%)	9
Liver	2 (4.3%)	2 (3.8%)	4
Central nervous system	3 (6.4%)	1 (1.9%)	4
Dengue	0	1 (1.9%)	1
Pancreas	0	1 (1.9%)	1
Others	0	2 (3.8%)	2

Overall mean age 51.7 ± 17.2 years; males 49.7 ± 2.5 years, females 53.6 ± 2.5 years.

Fifty-three of 100 patients died, an in-hospital mortality of 53%. Age was distributed almost uniformly across the three bands with no gradient in outcome, indicating that in this cohort chronological age alone offered no discrimination. Sex distribution was exactly balanced at enrolment yet diverged sharply at outcome: 62% of males died compared with 44% of females. Lower respiratory tract infection was both the leading source overall and over-represented among deaths, contributing 49.1% of non-survivors against 34.0% of survivors. Urogenital sepsis showed the opposite pattern, accounting for nearly a third of survivors but under a tenth of deaths, consistent with the generally better prognosis of urinary source sepsis. Soft tissue infection, though numerically small at nine patients, was associated with eight deaths.

Table 2. Sequential Organ Failure Assessment score by outcome

SOFA score	Survivors (n = 47)	Non-survivors (n = 53)	Total	Mortality within stratum
< 5	32 (68.1%)	4 (7.5%)	36	11.1%
6–10	15 (31.9%)	13 (24.5%)	28	46.4%
11–15	0	26 (49.1%)	26	100%
> 15	0	10 (18.9%)	10	100%
Mean ± SD	**3.6**	**11.7 ± 3.6**		p < 0.001

The SOFA score produced an almost perfect ordinal gradient in mortality, rising from 11.1% in the lowest stratum through 46.4% in the intermediate band to 100% in both upper strata. Not a single patient with a score above 10 survived to discharge, and conversely more than two-thirds of survivors scored below 5. The mean difference of approximately eight points between groups is substantial in a scale that ranges from 0 to 24. The intermediate 6–10 band is the only genuinely equivocal stratum, splitting 15 to 13, which identifies it as the range in which clinical judgement and response to early resuscitation, rather than the admission score alone, will determine outcome.

Table 3. Serum lactate by outcome

Serum lactate (mmol/L)	Survivors (n = 47)	Non-survivors (n = 53)	Total
1–5	44 (93.6%)	15 (28.3%)	59
6–10	3 (6.4%)	38 (71.7%)	41
Mean ± SD	**1.6 ± 0.50**	**4.3 ± 2.0**	$p < 0.001$

Stratum counts reconstructed from the reported proportions so that group totals reconcile to 47 and 53 respectively.

The dichotomy at 6 mmol/L separated the cohort decisively. Of the 41 patients with a lactate in the 6–10 mmol/L range, 38 died, a stratum mortality of 92.7%; of the 59 with a lactate of 5 mmol/L or below, 44 survived. Expressed from the outcome perspective, 71.7% of all deaths occurred among the minority of patients whose lactate exceeded 6 mmol/L. The survivor mean of 1.6 mmol/L lies within the normal range, which is notable given that all patients met septic shock criteria and indicates that a substantial proportion of these patients had achieved adequate tissue perfusion by the time of sampling. It should be noted that the reported non-survivor mean of 4.3 mmol/L is difficult to reconcile with 71.7% of that group falling in the 6–10 mmol/L band, and the two figures cannot both be correct.

Table 4. Anion gap by outcome

Anion gap (mEq/L)	Survivors (n = 47)	Non-survivors (n = 53)	Total	Mortality within stratum
< 16	44 (93.6%)	6 (11.3%)	50	12.0%
> 16	3 (6.4%)	47 (88.7%)	50	94.0%
Mean ± SD	**11.3 ± 3.2**	**22.5 ± 6.1**		$p < 0.001$

The anion gap divided the cohort into two exactly equal halves of 50 patients with radically divergent fates: 94% mortality above the threshold against 12% below it. A single cut-point at 16 mEq/L correctly classified 91 of 100 patients, the highest classification accuracy of any variable examined in this study. The mean anion gap of 22.5 mEq/L among non-survivors is roughly double the survivor value and approximately twice the upper limit of the conventional reference range. Lactic acidosis was identified as the predominant contributor to the elevated gap. The practical significance is considerable: the gap is derived from a routine electrolyte panel available in virtually every hospital laboratory, and in this cohort it performed at least as well as directly measured lactate.

Table 5. Comparison of prognostic markers with published series

Parameter	Present study (2019)	Kellum et al. (2001)	Ganesh et al. (2016)	Smith et al. (2001)	Ferreira et al. (2001)
Mean age (years)	51.7	42.7	56.2	—	—
SOFA, survivors	5	—	—	5	5
SOFA, non-survivors	11	—	—	13	11
Lactate, survivors (mmol/L)	1.6	2.1	1.9	—	—
Lactate, non-survivors (mmol/L)	6.2	9.0	7.5	—	—
Anion gap, survivors (mEq/L)	11.3	15.6	10.4	—	—
Anion gap, non-survivors (mEq/L)	22.5	28.0	17.0	—	—
Respiratory source (%)	42	—	—	47	—

The non-survivor lactate figure of 6.2 mmol/L is that used in the original comparative analysis and differs from the value of 4.3 mmol/L reported in Table 3; the discrepancy is unresolved.

Direction of effect was reproduced across every comparator series. SOFA scores align closely, with survivors clustering around 5 and non-survivors around 11 to 13 in all three cohorts reporting the score. Lactate values in the present study sit at the lower end of the published range in both outcome groups, while anion gap values fall between those of Kellum and Ganesh. Respiratory tract infection was the dominant source at 42% here against 47% in the Smith series, confirming the consistency of this epidemiological pattern.

DISCUSSION

This cohort demonstrates that three markers of very different character — a composite organ dysfunction score, a directly measured metabolite and a calculated electrolyte derivative — each stratified septic shock mortality powerfully, and that the simplest and cheapest of the three performed at least as well as the others.

The SOFA findings reproduce the established literature closely. The absence of any survivor scoring above 10, and the 100% mortality in both upper strata, are consistent with the original validation work of Vincent and colleagues [14] and with the serial evaluation of Ferreira et al., who showed that both initial and maximum scores predict outcome and that rising values over 48 hours carry a mortality exceeding 50% [15]. The mean values of approximately 5 in survivors and 11

in non-survivors correspond almost exactly to those reported in comparator series. The reservation attaching to SOFA is operational rather than statistical: computation requires arterial oxygenation, platelet count, bilirubin, creatinine, Glasgow Coma Scale and vasopressor dosing, which limits its utility as an immediate triage instrument. The qSOFA construct was introduced partly to address this constraint [1].

Serum lactate behaved as expected. Its prognostic standing rests on a long literature: Bakker and colleagues showed it to be superior to oxygen-derived variables in predicting outcome in septic shock [7] and demonstrated that serial values predict subsequent multiple organ failure [8]. Nguyen et al. established that early lactate clearance, rather than any single value, associates most strongly with survival [9], which is precisely the dimension the present single-sample design could not capture. The mechanistic literature counsels interpretative caution — Levraut and colleagues attributed hyperlactataemia in stable septic patients principally to impaired clearance [4], Gore et al. to increased pyruvate production rather than oxygen deficit [5], and James et al. argued that lactate is an unreliable direct index of tissue hypoxia in sepsis [6]. Lactate is therefore best understood as a robust severity marker whose mechanism is heterogeneous.

The anion gap result is the most practically consequential finding. A cut-point of 16 mEq/L classified 91 of 100 patients correctly, with 94% mortality above the threshold and 12% below it. This supports the proposal of Berkman and colleagues that the anion gap can serve as a screening surrogate for elevated lactate in patients at risk of sepsis [11], and it matters most where point-of-care lactate assay is unavailable, as remains common in district-level Indian practice. Two caveats apply. Serum albumin, the principal unmeasured anion in health, was not measured, and hypoalbuminaemia frequent in critical illness can mask an otherwise elevated gap [12,13]; correction for albumin, as advocated by Hatherill et al., would probably have improved discrimination further [13]. Second, Cusack and colleagues found that the related strong ion gap lacked prognostic value in a mixed intensive care population [16], indicating that derived acid–base indices do not perform uniformly across case mixes.

The limitations of this study are material. It was observational with no intervention, and analysis rested on a single blood gas sample obtained within 24 hours of admission with no follow-up measurement, so that lactate clearance — the variable most strongly linked to survival in the literature [9] — could not be assessed. Chloride and albumin were not analysed separately, precluding partition of the acidosis into lactic, hyperchloraemic and unmeasured-anion components as Noritomi et al. achieved [17]. The single-centre design and sample of 100 limit precision, and no multivariable model was constructed, so the independence of these three markers from one another cannot be established from these data.

CONCLUSION

Among 100 prospectively enrolled patients with septic shock, in-hospital mortality was 53%, and admission SOFA score, serum lactate and calculated anion gap each discriminated strongly between survivors and non-survivors at $p < 0.001$. No patient with a SOFA score above 10 survived, whereas mortality was 11.1% among those scoring below 5. Serum lactate exceeding 6 mmol/L accounted for 71.7% of all deaths. The anion gap divided the cohort into two equal halves with mortality of 94% above 16 mEq/L and 12% below it, correctly classifying 91 of 100 patients and thereby achieving the highest single-variable accuracy in the study; lactic acidosis was the principal contributor to the elevated gap. Because the anion gap is derived from a routine serum electrolyte panel available in essentially every hospital laboratory, it represents a valuable adjunct for early risk stratification in settings where point-of-care lactate measurement is not accessible. Lower respiratory tract infection was the commonest source of sepsis at 42% and was over-represented among deaths. Prospective work incorporating albumin-corrected anion gap and serial lactate clearance is required to establish whether these markers contribute independently of one another.

REFERENCES

1. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-10.
2. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock: 2016. Crit Care Med. 2017;45(3):486-552.
3. Gladden LB. Lactate metabolism: a new paradigm for the third millennium. J Physiol. 2004;558(Pt 1):5-30.
4. Levraut J, Ciebiera JP, Chave S, Rabary O, Jambou P, Carles M, et al. Mild hyperlactatemia in stable septic patients is due to impaired lactate clearance rather than overproduction. Am J Respir Crit Care Med. 1998;157(4 Pt 1):1021-6.
5. Gore DC, Jahoor F, Hibbert JM, DeMaria EJ. Lactic acidosis during sepsis is related to increased pyruvate production, not deficits in tissue oxygen availability. Ann Surg. 1996;224(1):97-102.
6. James JH, Luchette FA, McCarter FD, Fischer JE. Lactate is an unreliable indicator of tissue hypoxia in injury or sepsis. Lancet. 1999;354(9177):505-8.
7. Bakker J, Coffernils M, Leon M, Gris P, Vincent JL. Blood lactate levels are superior to oxygen-derived variables in predicting outcome in human septic shock. Chest. 1991;99(4):956-62.

8. Bakker J, Gris P, Coffernils M, Kahn RJ, Vincent JL. Serial blood lactate levels can predict the development of multiple organ failure following septic shock. Am J Surg. 1996;171(2):221-6.
9. Nguyen HB, Rivers EP, Knoblich BP, Jacobsen G, Muzzin A, Ressler JA, et al. Early lactate clearance is associated with improved outcome in severe sepsis and septic shock. Crit Care Med. 2004;32(8):1637-42.
10. Levy MM, Evans LE, Rhodes A. The Surviving Sepsis Campaign Bundle: 2018 update. Crit Care Med. 2018;46(6):997-1000.
11. Berkman M, Ufberg J, Nathanson LA, Shapiro NI. Anion gap as a screening tool for elevated lactate in patients with an increased risk of developing sepsis in the emergency department. J Emerg Med. 2009;36(4):391-4.
12. Figge J, Jabor A, Kazda A, Fencl V. Anion gap and hypoalbuminemia. Crit Care Med. 1998;26(11):1807-10.
13. Hatherill M, Waggie Z, Purves L, Reynolds L, Argent A. Correction of the anion gap for albumin in order to detect occult tissue anions in shock. Arch Dis Child. 2002;87(6):526-9.
14. Vincent JL, Moreno R, Takala J, Willatts S, De Mendonça A, Bruining H, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. Intensive Care Med. 1996;22(7):707-10.
15. Ferreira FL, Bota DP, Bross A, Mélot C, Vincent JL. Serial evaluation of the SOFA score to predict outcome in critically ill patients. JAMA. 2001;286(14):1754-8.
16. Cusack RJ, Rhodes A, Lochhead P, Jordan B, Perry S, Ball JA, et al. The strong ion gap does not have prognostic value in critically ill patients in a mixed medical/surgical adult ICU. Intensive Care Med. 2002;28(7):864-9.
17. Noritomi DT, Soriano FG, Kellum JA, Cappi SB, Biselli PJ, Libório AB, et al. Metabolic acidosis in patients with severe sepsis and septic shock: a longitudinal quantitative study. Crit Care Med. 2009;37(10):2733-9.
18. Smith I, Kumar P, Molloy S, Rhodes A, Newman PJ, Grounds RM, et al. Base excess and lactate as prognostic indicators for patients admitted to intensive care. Intensive Care Med. 2001;27(1):74-83.
19. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345(19):1368-77.
20. Kumar A, Roberts D, Wood KE, Light B, Parrillo JE, Parsa S, et al. Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock. Crit Care Med. 2006;34(6):1589-96.
21. Rocha LL, Pessoa CM, Colombo G, Correa TD, de Assunção MS. Lactate as a prognostic marker in patients with severe sepsis or septic shock admitted to the ICU. Crit Care. 2013;17(Suppl 3):P51.
22. Kiran HS, Anil GD, Sudharshana Murthy KA, Basavana Gowdappa H. Severe metabolic acidosis in critically ill patients and its impact on the outcome: a prospective observational study. Int J Sci Study. 2015;3(6):39-44.