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Original Research Article

Vasoactive-inotropic score as a predictor of mortality in neonatal shock: a prospective validation study in a sepsis dominant resource-limited setting

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ABSTRACT

Background: Shock is an important clinical entity in critically ill neonates. Vasopressors and inotropes are mainstay of therapy. There is paucity of good quality evidence regarding the outcome of vasopressors/inotropes in neonatal shock management from Indian setting. The objective was to evaluate the maximum vasoactive-inotropic score (VIS_{max}) an independent predictor of mortality and identify a high-risk threshold in a sepsis-dominant neonatal cohort.

Methods: This prospective study enrolled 107 neonates requiring vasoactive support in a tertiary level neonatal intensive care unit. VIS_{max} was calculated during the first 72 hours. Predictive accuracy was assessed via ROC analysis and multivariate logistic regression adjusting for birth weight and sepsis.

Results: Sepsis was the primary aetiology in 70.6% of non-survivors. Non-survivors required significantly higher support (median VIS_{max} 20 versus 10; p=0.002). A VIS_{max} ≥20 yielded a PPV of 84.8% and specificity of 78.1% (AUC=0.690). Multivariate analysis identified Sepsis (aOR=3.965) and VIS_{max} (aOR=1.080) as independent predictors of death (p<0.01).

Conclusions: VIS_{max} ≥20 is a critical bedside "red flag" for septic neonates, marking an 85% case fatality rate and providing a robust framework for prognostic counselling.

Keywords: Vasoactive-inotropic-score, Neonatal shock, Mortality predictor, Neonates, Sepsis, VIS_{max}

INTRODUCTION

Circulatory failure, or shock, is a critical syndrome in the neonatal intensive care unit (NICU) characterized by imbalance between oxygen delivery and cellular demand. In India, neonatal shock remains a significant mortality driver, with 12.0% incidence among NICU admissions.^{1,2} Untreated, this progresses to multi-organ dysfunction and death. The pathophysiology differs in low-to-middle-income countries compared to high-income settings. While high-resource centres manage shock from cardiac surgery or transitional physiology, sepsis remains the primary cause in South Asian NICUs.²⁻⁴ Management

prioritizes fluid resuscitation and vasoactive agent.⁵⁻⁸ Due to septic shock's distinct pathophysiology, traditional mortality predictors like CRIB-II may not accurately capture vasoactive support needed for survival.⁹

The vasoactive-inotropic score (VIS), developed for paediatric cardiac surgery outcomes, and later refined to include vasopressors like epinephrine and milrinone is a well-established prognostic tool in high-income countries.¹⁰⁻¹³ However, prospective validation is needed for sepsis-dominant cohorts in resource-limited settings. Unlike myocardial-centric shock in cardiac surgery, the systemic inflammatory response and multi-organ

dysfunction of neonatal sepsis necessitates population-specific benchmarks that reflect unique hemodynamic constraints and higher baseline mortality.^{11,12,14,15}

Previous studies have illustrated the score's utility in neonatal septic shock, but these findings are often limited by small sample sizes and narrow inclusion criteria.^{16,17} There is a significant gap in understanding how VIS performs as a surrogate of cumulative ionotropic burden in identifying a definitive, bedside threshold that correlates with survival among neonates across the wider, heterogeneous spectrum of all-cause neonatal shock in resource-limited settings.

Identifying a definitive, population-specific bedside threshold is therefore essential for accurate risk stratification and timely clinical intervention in these high-stakes environments.

The primary objective of the study was to determine the relationship between VIS_{max} and mortality in neonates requiring cardiovascular support.

The secondary objectives were: to identify the optimal prognostic threshold of VIS_{max} for predicting mortality in a sepsis-heavy cohort, to compare the predictive accuracy of VIS_{max} for mortality against traditional markers (birth weight, gestational age), and to document the clinical aetiologies and pharmacotherapeutic patterns of neonatal shock in a tertiary NICU in Eastern India.

METHODS

Study design and setting

This was a prospective, longitudinal observational study conducted in the level III NICU of a tertiary care teaching hospital. The study was conducted over a 12-month period from 01 July 2023 to 30 June 2024 in accordance with the declaration of Helsinki. The institutional ethics committee provided formal approval and written informed consent was obtained from the parents or legal guardians of all participants prior to enrolment.

Participants and eligibility

All neonates (aged 0-28 days) admitted to the NICU were screened for inclusion.

Inclusion criteria

Neonates of any gestational age and birth weight presenting with clinical signs of shock requiring initiation of vasoactive agents (inotropes/vasopressors).

Shock was defined by presence of any one of the following: capillary refill time (CRT) >3 seconds, poor peripheral pulses, tachycardia or bradycardia, mottled skin, and cold extremities, which persisted despite initial fluid resuscitation (10-20 ml/kg of isotonic crystalloids)

with or without documented hypotension [systolic (SBP) or diastolic blood pressure (DBP) <5th percentile of gestational age (in weeks) and postnatal age (in hours)].^{2,5,7,18}

Exclusion criteria

Neonates with lethal congenital malformations and those who were moribund at the time of admission (expected survival <6 hours) were excluded.

Variables

Baseline demographic and clinical data were recorded upon enrolment, including gestational age (GA), birth weight (BW), sex, mode of delivery, and Apgar scores.

Live born infants who were delivered before 37 weeks of gestational age were classified as preterm.¹⁹ The primary aetiology of shock was categorized based on clinical and laboratory findings.

Septic shock

Based on international consensus definitions - the presence of shock during a clinically probable or culture-proven sepsis episode or clinical suspicion with positive biomarkers and systemic inflammatory response.^{3,20,21}

Cardiogenic shock

Shock in asphyxiated neonates within first 72 hours of life with a negative sepsis workup.^{5,7,22-24}

Other aetiologies

Congenital heart disease (CHD), patent ductus arteriosus (PDA), transitional circulation (shock due to immaturity of myocardium in a very low birth weight baby within the first 12 hours of birth, not explained by other aetiologies like asphyxia, PDA or sepsis).

Clinical management and monitoring

Blood pressure was monitored using non-invasive blood pressure (NIBP) oscillometric devices, adhering to the standardized techniques recommended by the international neonatal consortium.^{20,25,26}

Hemodynamic management followed a standardized protocol by the NICU team.^{5,7} Initial stabilization included airway management and volume expansion. Vasoactive support was initiated per NICU guidelines using standard vasopressors/inotropes.⁵

Vital signs, including heart rate, NIBP, and SpO₂ were continuously monitored. Other management followed standard NICU protocol guidelines.

Vasoactive-inotropic score (VIS)

The intensity of pharmacological support was quantified using the vasoactive-inotropic score (VIS).¹¹ It was calculated as a weighted sum of all administered vasopressors/inotropes.

Vasoactive inotropic score

$$\begin{aligned} &= \text{Dopamine dose } (\mu\text{g/kg/min}) \\ &+ \text{Dobutamine dose } (\mu\text{g/kg/min}) \\ &+ 100 \times \text{Epinephrine dose } (\mu\text{g/kg/min}) \\ &+ 10 \times \text{Milrinone dose } (\mu\text{g/kg/min}) \\ &+ 10 \times \text{Vasopressin dose (mU/kg/min)} \\ &+ 100 \times \text{Norepinephrine dose } (\mu\text{g/kg/min}) \end{aligned}$$

The VIS_{max} was defined as the highest score recorded for a neonate during the first 72 hours of shock management or until death/discharge.

Outcome measures

Primary outcome was short-term mortality, defined as death prior to discharge from the NICU.

Secondary outcome was identification of a specific VIS_{max} threshold that best predicts mortality.

Statistical analysis

Data were analysed using statistical package for the social sciences (SPSS) version 26.0 (IBM corp, Armonk, NY).

Descriptive statistics

Continuous variables were presented as mean $\pm$ standard deviation (SD) or median with interquartile range (IQR), and categorical variables as frequencies and percentages.

Inferential statistics

For continuous variables, differences between survivors and non-survivors were assessed using the student's t-test for parametric data and Mann-Whitney U test and Hodges-Lehmann test for non-parametric data continuous data.

Chi-square or Fisher's exact test for used to analyse categorical data.

Prognostic accuracy

A receiver operating characteristic (ROC) curve was generated for VIS_{max} against mortality to determine the area under the curve (AUROC). The optimal prognostic cut-off was determined using the Youden index

$$J = \text{Sensitivity} + \text{Specificity} - 1$$

Multivariate analysis

Variables with $p < 0.05$ in univariate analysis were entered into a multivariate logistic regression model to identify independent predictors of mortality, reporting adjusted Odds Ratios (aOR) and 95% confidence intervals (CI).

A p value of <0.05 was considered statistically significant.

RESULTS

A total of 107 neonates, involving 123 distinct episodes of shock, were enrolled in the study. The longitudinal progression from initial assessment to outcome is detailed in the study flow diagram (Figure 1).

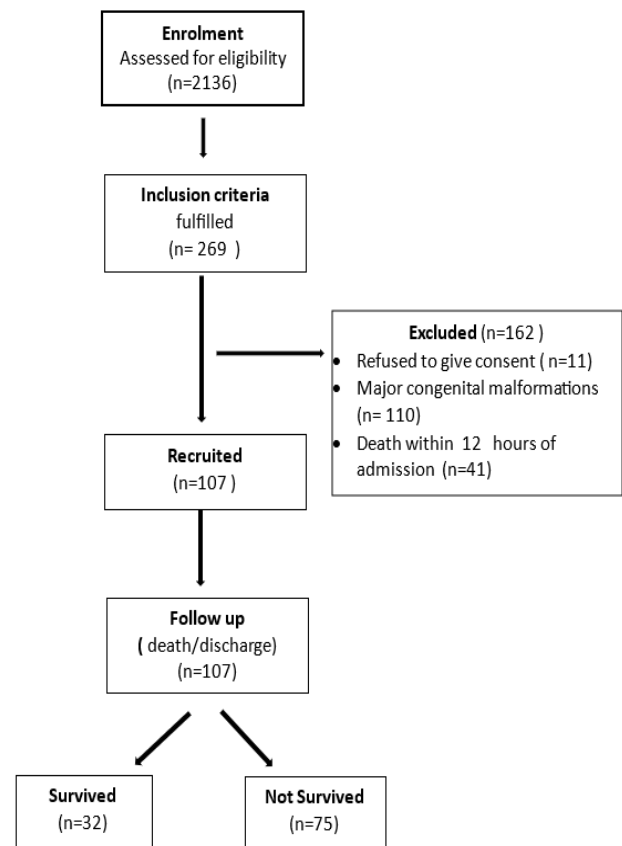


Figure 1: STROBE diagram.

The median gestational age was 32.03 $\pm$ 5.1 weeks, with a median birth weight of 1580 $\pm$ 840 grams. Very low birth weight (VLBW) infants comprised 53.27% of the participants. As summarized in the consolidated baseline and clinical characteristics (Table 1), significant differences were observed between survivors and non-survivors regarding birth weight ($p=0.02$) and the presence of sepsis ($p<0.001$). Sepsis was identified as the primary aetiology in 70.6% of the non-survivor group, compared to 34.3% in survivors.

Prognostic accuracy and threshold identification of maximum vasoactive ionotropic score (VIS_{max}).

The intensity of cardiovascular support, quantified by VIS_{max} , served as a primary differentiator of clinical outcome. As illustrated in the box plot (Figure 2a), non-survivors required significantly higher pharmacological

support, with a median VIS_{max} of 20 (IQR 10-30), compared to a median of 10 (IQR 7.5-17.63) in the survivor group ($p=0.002$).

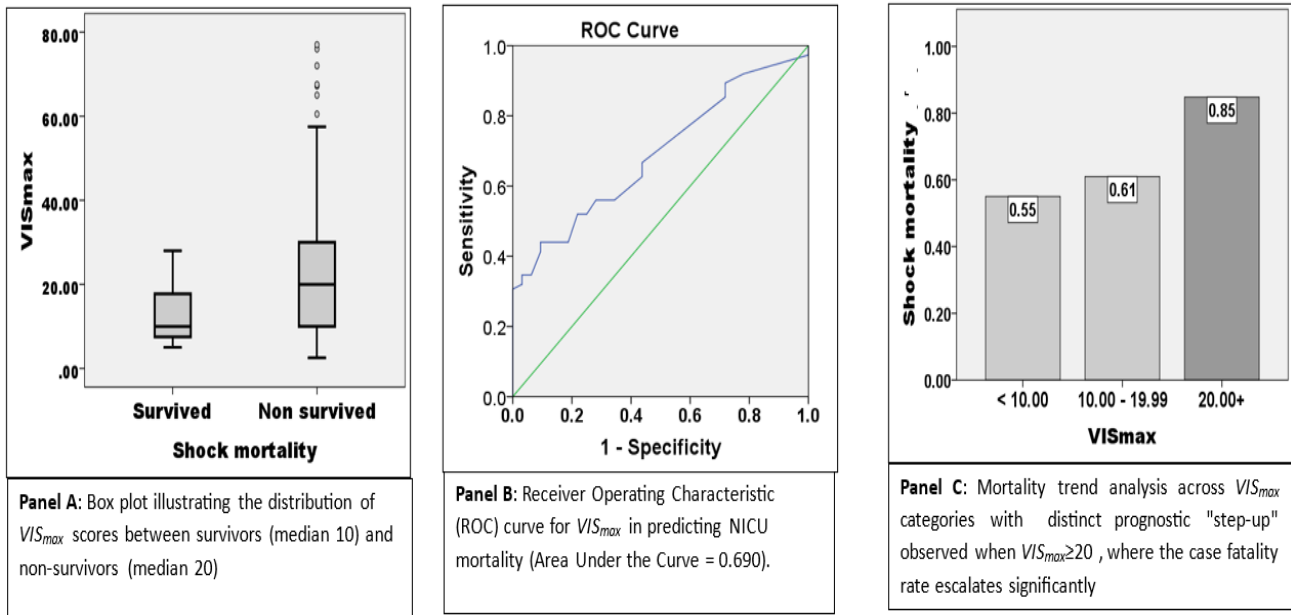


Figure 2: Prognostic performance of the maximum vasoactive-inotropic score (VIS_{max}).

The prognostic performance of VIS_{max} in predicting mortality was evaluated using receiver operating characteristic (ROC) curve analysis (Figure 2b) which yielded an area under the curve (AUC) of 0.690 (95% CI: 0.590-0.790; $p=0.002$), indicating fair prognostic accuracy. The 95% confidence interval ranged from 0.590 to 0.790 ($p=0.002$), indicating that the score remains a statistically significant marker of outcome despite the complex nature of the septic cohort.

To identify the optimal clinical threshold, the Youden index was calculated across the coordinates of the curve. The maximum value for J (0.301) was observed at a $VIS_{max} \geq 20$, yielding a sensitivity of 52% and a specificity of 78.1%.

The score further demonstrated a positive predictive value (PPV) of 84.8% and a negative predictive value (NPV) of 41.0%. The high PPV reinforces the clinical utility of this threshold as a definitive marker for high-risk stratification in septic neonatal shock. For enhanced bedside utility, a threshold of $VIS_{max} \geq 20$ was adopted as the primary "red flag" for high mortality risk, as higher scores corresponded with a rapid increase in specificity.

Categorical trend analysis across consolidated clinical tiers revealed a clear prognostic 'step-up' as cardiovascular support intensity increased (Figure 2c). Mortality rates rose from 55% in the low-support group ($VIS_{max} < 10$) to 61% in the moderate-support group ($VIS_{max} = 10.00-19.99$). A sharp escalation to 85% mortality was observed once the

$VIS_{max} \geq 20$ threshold was reached, reinforcing this level as a critical physiological inflection point for neonates in septic shock.

Independent risk factors for mortality

To isolate the independent predictors of mortality, a multivariate logistic regression model was constructed including sepsis, VIS_{max} and birth weight. The model demonstrated good fit as evidenced by the Hosmer and Lemeshow test ($p=0.537$) and an Omnibus test of model coefficients ($p<0.001$). A non-significant Hosmer and Lemeshow test p value indicates that the model's predicted probabilities match the observed mortality. The model successfully classified 70.1% of the cases.

As detailed in Table 3, sepsis remained the strongest independent predictor of mortality, with an adjusted odds ratio (aOR) of 3.965 (95% CI: 1.436-10.954; $p=0.008$). This indicates that neonates with sepsis had nearly a four-fold increased risk of death compared to those with other aetiologies of shock.

The VIS_{max} also maintained independent statistical significance, with an aOR of 1.080 (95% CI: 1.026-1.137; $p=0.003$). For every one-unit increase in the VIS_{max} , the odds of mortality increased by 8%. Conversely, Birth Weight did not show a statistically significant independent association with mortality in the adjusted model (aOR=1.000; $p=0.22$).

Table 1: Baseline neonatal, maternal, and clinical characteristics of participants.

Variables	Total (n=107)	Survivor (n=32)	Non-survivor (n=75)	Odd's ratio (95% CI)	Mean difference or median difference (95% CI)	P value
Gestational age (weeks), mean±SD	32.03±5.12	33.38±4.91	31.453±4.70	-	1.922 (-0.074 to 3.917) [†]	0.059
Birth weight (g), median (IQR)	1371 (890-2137)	1638 (1065-2400)	1210 (830-2070)	-	353.5 (-698 to -44) [‡]	0.027*
Male sex, N (%)	61 (57)	18 (56.3)	43 (57.3)	1.04 (0.45 to 2.40)	-	0.91
Maternal and birth factors, N (%)						
Inborn status	76 (71.0)	26 (81.3)	50 (66.7)	2.16 (0.77-5.89)	-	0.12
LSCS delivery	61 (57)	21 (65.6)	40 (53.3)	0.59 (0.25-1.41)	-	0.2
Prematurity	79 (73.8)	20 (62.5)	59 (78.6)	2.2 (0.89 to 5.46)	-	0.08
PROM >18 hours	24 (22.4)	5 (15.6)	19 (25.3)	1.83 (0.61 to 5.49)	-	0.27
ANS coverage	37	13 (40.6)	24 (32)	1.45 (0.61 to 3.4)	-	0.39
Primary etiology of shock, N (%)						
Sepsis	64 (59.8)	11 (34.3)	53 (70.6)	4.59 (1.9 to 11.11)	-	0.0007*
Perinatal asphyxia	53 (49.5)	18 (56.3)	35 (46.6)	0.68 (0.29 to 1.5)	-	0.36
Congenital heart disease	21 (19.6)	7 (21.8)	14 (18.6)	0.81 (0.29 to 2.2)	-	0.7
Patent ductus arteriosus	60 (56)	20 (62.5)	40 (53.3)	0.68 (0.29 to 1.60)	-	0.38
Vasoactive inotrope score (VIS), median (IQR)						
VIS _{start}	12.5 (10-17.75)	10 (7.5-13.5)	15 (10-20)	-	3.00 (0 to 5.0) [^]	0.012*
VIS _{max}	15 (10-27.5)	10 (7.5-17.63)	20 (10-30)	-	5.25 (2.5 to 12.5) [^]	0.002*

*Statistically significant; [†] mean difference with 95% CI; [‡] median difference with 95% CI

Table 2: Vasopressor/inotrope usage profile in the participants (singly or in combination).

Agent	No. of babies in whom administered n (%)	Mean±SD starting dose (mcg/kg/min)	Median (IQR) cumulative dose (mg)	Median (IQR) duration (hours)
Dopamine	70 (65.42)	8.11±2.69	15.56 (5.4-35.33)	30.5 (10-45)
Dobutamine	35 (32.71)	7.64±2.39	12.5 (5.53-34.36)	27 (12.25-60.5)
Adrenaline	52 (48.59)	0.08±0.1	0.14 (0.04-0.438)	23 (7.5-51.5)
Noradrenaline	22 (20.56)	0.1±0.1	0.16 (0.06-0.67)	27 (13-43.75)
Milrinone	34 (31.78)	0.39±0.14	1.37 (0.29-3.77)	26 (12.5-48)

SD=standard deviation; IQR=inter-quartile range

Table 3: Multivariate logistic regression analysis of mortality predictors.

Variable	Odd's ratio	95% CI of odd's ratio	P value
Sepsis	3.965	1.436 to 10.954	0.008
VIS_{max}	1.080	1.026 to 1.137	0.003
Birth weight	1	0.999 to 1.000	0.22

VIS_{max} =Vasoactive inotrope score at maximum dose

DISCUSSION

The results of this prospective validation study identify VIS_{max} as a robust, independent predictor of mortality in neonates with shock in a resource-limited setting.

A central finding is that in a sepsis-dominant cohort, the "physiological ceiling" for survival appears significantly lower than in the cardiac or transitional shock populations of high-income settings. Our identified threshold of VIS_{max}

≥ 20 served as a definitive clinical inflection point, where the case fatality rate escalated sharply to 85%. This suggests that the systemic inflammatory burden and multi-organ dysfunction characteristic of neonatal sepsis leave infants with a diminished cardiovascular reserve, causing them to reach a point of physiological futility at lower inotropic loads than previously reported in myocardial-centric cohorts.

There are few studies across the globe that reported the profile of neonatal shock and the role of VIS together.^{9,11-14,16,17,27-34} The overall case fatality rate (CFR) of 70.1% observed in our cohort is significantly higher than the 21% reported in high-resource US centres.¹³ This disparity is fundamentally driven by the aetiology of shock. In our study, sepsis was the primary aetiology in 70.6% of non-survivors and was associated with a nearly four-fold increase in the risk of death (aOR=3.971; $p=0.008$). This mirrors the findings of Saini et al.²

While global literature consistently identifies VIS as a reliable mortality predictor, the specific thresholds for clinical futility vary significantly by population. In high-income settings like the cohort of ELBW infants in US, near-universal mortality was only observed when VIS_{max} scores exceeded 30.¹³ In contrast, our data shows 85% case fatality rate with a lower ceiling of VIS_{max} ≥ 20 . While Demirhan et al previously demonstrated that VIS_{max} ≥ 20 is a reliable predictor of mortality in septic shock, our study builds upon this by identifying a definitive "physiological inflection point".¹⁶

Although some neonates survived with scores up to 28, the concentration of non-survivor data points above the 20 mark suggests that in the context of sepsis, escalating inotropic support beyond this level often yields diminishing returns, compared to a relatively stable mortality trend at lower support levels. Sepsis was thus proved to be an independent driver of mortality in our multivariate model (aOR=3.965; $p=0.008$).

This lower threshold is likely explained by the systemic inflammatory burden of sepsis. Furthermore, unlike surgical cohorts where VIS_{max} often reflects transitional hemodynamic adjustment, our results reinforce that in septic neonates, VIS_{max} acts as a dynamic surrogate for disease severity that outperforms baseline markers like birth weight ($p=0.22$).

The variability in identified thresholds-ranging from Desai et al's threshold of 32 to our optimal threshold of 20-underscores the need for population-specific validation.¹⁷ While higher thresholds are often associated with culture-positive sepsis or specific morbidities like oliguria, our study demonstrates that a lower threshold of VIS_{max} ≥ 20 captures a critical prognostic 'step-up' to 85% mortality in a broader, real-world NICU cohort.

VIS thresholds and prognostic power

A critical finding of our study is the AUC of 0.690, found in ROC analysis of VIS_{max}, which reflects a "fair" prognostic accuracy. This is notably lower than the excellent ranges of AUCs (0.86-0.90) reported by Aziz et al or Gopal et al.¹³⁻³¹

This discrepancy highlights a fundamental truth about neonatal septic shock in LMICs: unlike the relatively predictable hemodynamic recovery following cardiac surgery, septic shock is a heterogeneous, multi-systemic insult. The lower AUC suggests that while the intensity of inotropic support (VIS_{max}) is a significant independent predictor, other factors - such as the timing of antibiotic administration, the specific pathogen virulence, and the degree of metabolic acidosis, myocardial depression, capillary leak, inflammatory dysregulation etc play a substantial role in determining survival in preterm septic infant which cannot be fully captured by VIS_{max} alone.

Nevertheless, the VIS_{max} ≥ 20 threshold remains a valuable bedside indicator. The identification of an 8% increase in death risk per unit-increase in VIS provides clinicians with an objective, quantified measure of "physiological cost". It identifies a critical juncture where escalating pharmacological support may yield diminishing returns, prompting a more comprehensive multi-modal assessment.

The prognostic performance of VIS_{max} in our cohort reflects the unique challenges of managing neonatal shock in a resource-limited, sepsis-dominant setting.

Clinical interpretation of diagnostic metrics

Our high PPV of 84.8% and specificity of 78.1% are significant. The clinical utility of a prognostic marker is defined by the balance between its sensitivity and specificity, which in turn drives its predictive accuracy in a specific population. At VIS_{max} ≥ 20 , this specificity drives the robust 84.8% PPV. While Demirhan et al noted the score's predictive power, the clear "step-up" in our cohort provides a direct bedside "red flag".¹⁶ This high specificity effectively "rules in" mortality risk, minimizing false positives and helping clinicians identify critically ill infants for care escalation or prognostic counselling.

However, the 52% sensitivity indicates limitations, as nearly half of non-survivors had VIS_{max} below 19. This lower sensitivity and 41% NPV highlight septic shock's "silent" severity. In our cohort, sepsis was a strong predictor of death (aOR=3.965), with many infants experiencing organ failure before reaching extreme inotropic requirements. While VIS_{max} ≥ 20 serves as a definitive "red flag," a lower score cannot rule out mortality risk. This reinforces our conclusion that VIS_{max} must be integrated into a multi-modal assessment that includes clinical biomarkers and aetiology-specific risks rather than being used as a standalone prognosticator.

Clinical utility and the adrenaline paradigm

Our study reveals a distinct pharmacological profile compared to high-resource settings. While dopamine was the most frequent primary agent (65.42%), nearly half the cohort (48.59%) required adrenaline (mean starting dose 0.08 ± 0.1 ug/kg/min). In Western cohorts, adrenaline is often reserved as a rescue therapy for cardiac failure or post-surgical support.^{13,33} In our context, the early and frequent escalation to adrenaline underscores the severity of myocardial depression and systemic vasodilation characteristic of neonatal septic shock. Our findings validate that even in an adrenaline-heavy treatment protocol, the VIS remains a dynamic surrogate for illness severity.

Strengths

A key strength of this study is its prospective, longitudinal design, enabling hourly VIS calculation during the first 72 hours of neonatal shock management. Our multivariate model's robustness, confirmed by the Hosmer and Lemeshow test, shows good calibration and data fit.

Limitations

The exclusion of multi-collinearity (all VIFs <1.3) confirms that VIS_{max} and sepsis effects are distinct. This validates that vasoactive support intensity better predicts survival than baseline demographics. However, limitations exist. The single-centre study may limit the $VIS_{max} \geq 20$ threshold's generalizability to other NICUs. The moderate AUC (0.690) and low NPV (41%) reflect neonatal sepsis heterogeneity, where mortality often occurs before high inotropic requirements. The reliance on non-invasive oscillometry is a constraint, as it may overestimate blood pressure during severe hypotension and high-dose vasopressor use, leading to delayed identification of refractory shock. Finally, we lacked functional echocardiography and continuous cardiac output monitoring, which would have better shown myocardial response to inotropic loads.

CONCLUSION

This study validates VIS_{max} as a robust, independent predictor of mortality in a sepsis-dominant neonatal population. While traditional risk assessment relied on static markers like birth weight, our results demonstrate that the physiological intensity of cardiovascular support—quantified by the VIS_{max} —is a more potent driver of outcome. Each one-unit increase in pharmacological support intensity adds 8% to the odds of mortality, highlighting VIS_{max} as a dynamic surrogate for disease severity.

A pivotal finding is the identification of $VIS_{max} \geq 20$ as a high-risk physiological threshold, characterized by a PPV of 84.8% and a specificity of 78.1%. Neonates reaching this level of support exhibited a distinct prognostic "step-

up," with the case fatality rate escalating to 85%. Although the moderate AUC (0.690) and NPV (41%) reflect the multi-systemic complexity and heterogeneity of neonatal sepsis, the high specificity confirms that a $VIS_{max} \geq 20$ serves as a definitive clinical "red flag."

We conclude that the VIS_{max} is an invaluable bedside tool for early risk stratification in resource-limited NICUs. Integrating this score into routine protocols provide clinicians with an objective, data-driven framework to guide care escalation and facilitate timely, standardized prognostic counselling for families during the critical "golden hours" of neonatal shock.

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

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