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Original Article

Paediatrics Section

Anthropometric and Neurodevelopmental Trajectories of High-risk SNCU Graduates in the First Year of Life: A Longitudinal Cohort Study

NUPUR MISHRA[1], MANGLA SOOD[2], ANUPAM SINGH[3]

ABSTRACT

Introduction: While neonatal intensive care expansion has improved survival, high-risk neonates face significant Extrauterine Growth Restriction (EUGR) and neurodevelopmental morbidity. Longitudinal data on both these trajectories remains scarce in resource constrained hilly areas in North India.

Aim: To evaluate the longitudinal anthropometric and neurodevelopmental catch-up growth of high-risk Sick Newborn Care Unit (SNCU) graduates over their first year and also to identify independent perinatal predictors of developmental delay.

Materials and Methods: A longitudinal cohort study evaluated 170 high-risk neonates discharged from SNCU at Indira Gandhi Medical College Shimla, Himachal Pradesh, India, from July 2024 till December 2025. Anthropometric parameters {World Health Organisation (WHO)/Intergrowth-21st Z-scores} were assessed at three, six, nine, and 12 months of corrected age. Neurodevelopment was evaluated at six and 12 months using the Developmental Assessment Scale for Indian Infants (DASII). Multivariate logistic regression was utilised to determine independent risk factors for motor and mental delay with statistical significance set at $p<0.05$.

Results: The cohort (64.7% preterm; mean birth weight 2191±734 g) experienced profound early EUGR. By 12 months, somatic weight completely normalised (mean Z-score -0.30±0.86), but severe linear stunting persisted in 41.23% of infants. Multivariate analysis at 12 months identified prematurity (Motor aOR 19.86; Mental aOR 8.31), birth asphyxia (Motor aOR 6.15; Mental aOR 5.27), and neonatal sepsis (Mental aOR 4.05) as independent predictors of delay.

Conclusion: Despite robust weight catch-up, linear stunting and neurodevelopmental delay persist. Individualised early intervention must aggressively target infants surviving sepsis and asphyxia alongside extreme preterm and prolonged SNCU stay as trigger for early enrollment.

Keywords: Asphyxia neonatorum, Extrauterine growth restriction, Infant, Neurodevelopmental disorders, Premature, Sick newborn care unit

INTRODUCTION

The establishment and expansion of Sick New born Care Units (SNCU) across India have led to a commendable decline in neonatal mortality, significantly improving the survival rates of preterm and critically ill infants [1]. However, this increased survival has precipitated a corresponding rise in neonatal morbidity [2]. High-risk SNCU graduates, particularly those with a history of prematurity, neonatal sepsis, and birth asphyxia, remain exquisitely vulnerable to EUGR and subsequent neurodevelopmental impairment [2,3]. While intensive care protocols have successfully mitigated acute mortality, the long-term somatic and neurological trajectories of these infants during the critical first year of life remain a pressing public health concern in resource-limited settings [4].

Current global literature extensively documents neurodevelopmental outcomes using western assessment models [5,6]; however, there is a paucity of prospective, longitudinal data utilising local culturally and socioeconomically validated tools, such as the DASII, from resource-constrained state-level SNCU cohorts in Northern India. Furthermore, while the isolated impact of prematurity or asphyxia are well-recognised, the independent neurotoxic burden of systemic inflammatory insults, such as culture-positive sepsis and prolonged mechanical ventilation, requires precise multivariable quantification [7,8].

Post-discharge tracking is frequently hindered by challenging geographic terrain leaving the true burden of long-term impairment poorly quantified. Furthermore, relying solely on crude post-discharge survival data or isolated cross-sectional growth can mask a highly discordant recovery trajectory, where somatic weight gain co-exists with hidden linear stunting, microcephaly, and progressive neurological deficits. Understanding the timeline of somatic catch-up growth, and its correlation with motor and mental milestones is essential for optimising District Early Intervention Centre (DEIC) protocols.

Although clinical outcomes of high-risk neonates are documented in metropolitan tertiary centres [2,3,7], these findings cannot be generalised to regions with significant geographic barriers to healthcare access. Therefore, this prospective cohort study was conducted to bridge this literature gap, and evaluate the longitudinal anthropometric and neurodevelopmental trajectories of high-risk SNCU graduates over their first year of life. The secondary objective was to identify the independent perinatal and clinical predictors of severe motor and mental delay at 12 months of corrected age to better stratify infants requiring targeted early intervention.

MATERIALS AND METHODS

This longitudinal cohort study was conducted at the SNCU of Indira Gandhi Medical College, Shimla, Himachal Pradesh, India, from July 2024 till December 2025. The study protocol was approved by the Institutional Ethics Committee (HFW/MC/IEC/2025-61), and written informed consent was obtained from the parents or legal guardians of all enrolled infants prior to inclusion.

An a priori sample size calculation was not executed; instead, a time-bound consecutive sampling strategy was utilised.

Inclusion criteria: The study population comprised high-risk neonates discharged alive from the SNCU over a consecutive

six-month period and prospectively followed-up to one year of corrected gestational age to capture a full annual cohort of survivors. Neonates were enrolled if they met one or more of the following high-risk criteria [9]: prematurity (gestational age <37 weeks), low birth weight (<1500 grams), severe birth asphyxia {5-minute Appearance, Pulse, Grimace, Activity, and Respiration (APGAR) score <3 or requiring advanced resuscitation}, culture-positive or clinical neonatal sepsis, neonatal encephalopathy/seizures, severe hyperbilirubinaemia requiring exchange transfusion, or requirement of respiratory support {Continuous Positive Airway Pressure (CPAP) or mechanical ventilation >24 hours}.

Exclusion criteria: Infants with major congenital malformations, recognised chromosomal anomalies or genetic syndromes, and those whose parents refused consent for long-term follow-up were excluded.

Study Procedure

Follow-up and anthropometric assessment: Enrolled infants were prospectively followed at the high-risk clinic at 3, 6, 9, and 12 months of age. For infants born prematurely, corrected age (post-menstrual age) was calculated and utilised for all anthropometric and developmental assessments. At each visit, growth parameters were recorded using standardised techniques. Weight was measured using an electronic infant weighing scale (accuracy ±10 g). Length was measured using a standard infantometer to the nearest 0.1 cm. Occipitofrontal Circumference (OFC) was measured using a non-stretchable fibreglass measuring tape over the most prominent part of the occiput and just above the supraorbital ridges, to the nearest 0.1 cm. Anthropometric Z-scores (Weight-for-age, Length-for-age, and OFC-for-age) were computed to determine the incidence of EUGR, stunting, and microcephaly. The INTERGROWTH-21st standards were utilised for premature infants until 50 weeks post-menstrual age [10]. The WHO Child Growth Standards were strictly applied for term infants and for preterms once they reached term-equivalent corrected age [11].

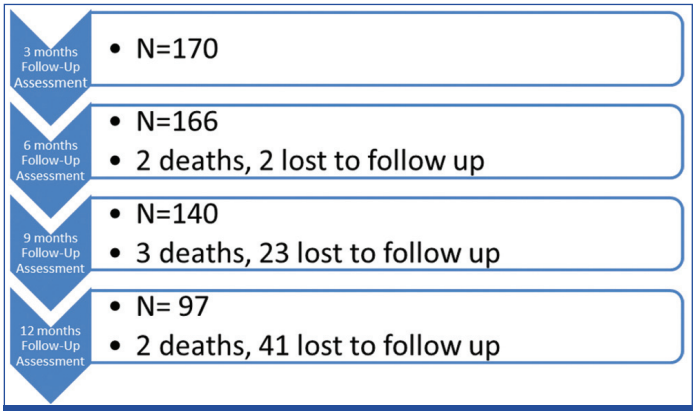
Neurodevelopmental assessment: Neurodevelopmental outcomes were evaluated at 6 and 12 months of corrected age using the DASII [12,13], a culturally validated Indian adaptation of the Bayley Scales of Infant Development (BSID) [5]. The scale comprises a total of 230 items divided across two distinct developmental domains: the Motor Scale (67 items) evaluating gross and fine motor capabilities, and the Mental Scale (163 items) assessing sensory-perceptual tracking, cognition, memory, and language. Individual items were evaluated through direct clinical observation, scored on a binary basis as passed (1 point) or failed/not performed (0 points). Cumulative raw scores for each domain were converted into a Developmental Age (DA) based on normative tables. The assessment yielded two primary indices: Motor Development Quotient (MoDQ) and Mental Development Quotient (MeDQ). Quotients were calculated as (DA/Corrected chronological age)×100. Outcomes were categorised as normal (≥85), and delay (<85).

STATISTICAL ANALYSIS

Data was entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS) version 18 statistical software. Continuous variables were expressed as mean±Standard Deviation (SD) or median with Interquartile Range (IQR). Categorical variables were summarised as frequencies and percentages. The primary outcome was developmental delay (Quotient <85) at 12 months. Univariable analysis was performed using the Chi-square test or Fisher's exact test for categorical risk factors. Variables demonstrating statistical significance (p<0.025) were subsequently entered into a multivariable logistic regression model. Adjusted Odds Ratios (aOR) with 95% Confidence Interval (CI) was calculated to identify independent predictors of motor and mental delay. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 170 high-risk neonates were enrolled after discharge from SNCU. There were total seven deaths during this period. All of these were out-of-hospital deaths that occurred at home, and the specific underlying causes remained undetermined [Table/Fig-1].



[Table/Fig-1]: Study flowchart.

The cohort was predominantly male (n=97, 57.1%) and preterm (n=110, 64.7%), with a mean gestational age of 34.84±3.06 weeks. Most infants (n=143, 84.1%) were appropriate for gestational age. Reflecting significant critical illness, the mean SNCU admission duration was 14.15±11.40 days (median stay of 9 days). The most prevalent primary morbidities during hospitalisation included neonatal jaundice (n=126, 74.1%), clinical or culture-positive sepsis (n=66, 38.8%), severe birth asphyxia (n=44, 25.9%), and hypoglycaemia (n=29, 17.1%) [Table/Fig-2].

Variables	Frequency (%) or Mean±SD
Gender	
Male	97 (57.1%)
Female	73 (42.9%)
Gestation type	
Preterm (<37 weeks)	110 (64.7%)
Term (≥37 weeks)	60 (35.3%)
Gestational age (weeks)	34.84±3.06
Birth weight (grams)	2191.35±734.40
Birth weight category	
Appropriate for Gestational Age (AGA)	143 (84.1%)
Small for Gestational Age (SGA)	21 (12.4%)
Large for Gestational Age (LGA)	6 (3.5%)
SNCU length of stay (days)	14.15±11.40
Primary neonatal morbidities	
Neonatal Jaundice (NNJ)	126 (74.1%)
Neonatal sepsis	66 (38.8%)
Birth asphyxia	44 (25.9%)
Neonatal hypoglycaemia	29 (17.1%)
Neonatal seizures	8 (4.7%)

[Table/Fig-2]: Baseline demographic and clinical characteristics of high-risk neonates (N=170).

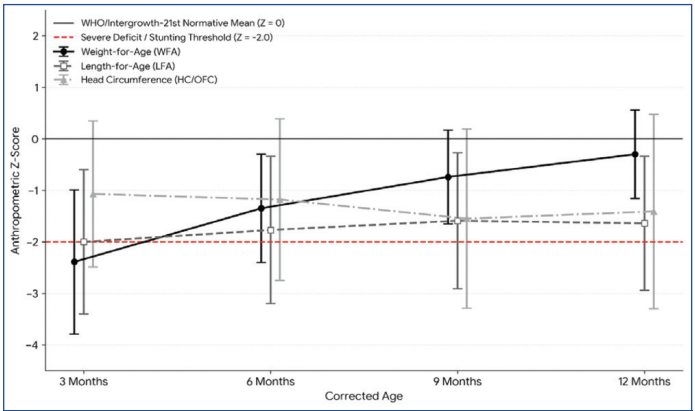
Longitudinal anthropometric analysis using WHO and INTERGROWTH-21st growth standards revealed a distinct dichotomy between weight and linear catch-up growth. At 3 months of chronological age, 100 infants (58.8%) in the cohort were severely underweight (Z-score <-2). However, a rapid catch-up in somatic mass occurred after six months. By 12 months, none of the followed-up cohort remained severely underweight, with the mean weight Z-score normalising to -0.30±0.86. In contrast, linear growth remained persistently suppressed, with the prevalence of stunting (Length Z-score <-2) persisting in 40 (41.23%) at 1 year. Corrected OFC Z-scores demonstrated initial brain-sparing at three months

(mean Z=-1.07), but the incidence of acquired microcephaly (Z <-2) increased to 41.23% (n=40) 12 months [Table/Fig-3].

[Table/Fig-4] showed longitudinal catch-up growth trajectories of high-risk SNCU graduates over the first year of life. It demonstrated the discordant somatic recovery: while weight normalises by 12 months, linear growth remains persistently stunted below the -1.5 Z-score threshold.

Time point	Mean weight Z-score±SD	Severe underweight N (%)	Mean length Z-score±SD	Severe stunting N (%)	Corrected OFC Z-score±SD	Microcephaly N (%)
3 months	-2.39±1.40	100 (58.8)	-2.00±1.40	86 (50.6)	-1.07±1.42	44 (25.8)
6 months	-1.35±1.05	45 (27.1)	-1.77±1.43	68 (40.9)	-1.18±1.57	51 (30.9)
9 months	-0.74±0.91	15 (10.8)	-1.59±1.32	54 (38.6)	-1.55±1.74	57 (40.7)
12 months	-0.30±0.86	0	-1.64±1.30	40 (41.23)	-1.41±1.89	40 (41.23)

[Table/Fig-3]: Longitudinal anthropometric Z-scores of high-risk neonates over the first year.



[Table/Fig-4]: Longitudinal catch-up growth trajectories.
Data points represent the mean Z-scores for Weight-for-Age, Length-for-Age and Head Circumference-for-Age at 3, 6, 9, and 12 months of corrected age, based on WHO and Intergrowth-21st standards. Error bars indicate ±1 SD. The solid line at Z=0 represents the normative population mean, while the dashed red line at Z=-2.0 denotes the threshold for severe Extra Uterine Growth Restriction (EUGR) and stunting

Neurodevelopmental Outcomes and Predictors of Delay

Neurodevelopmental assessment utilising the DASII at six and 12 months identified a significant burden of developmental impairment. At six months, 124 infants had motor delay and 112 demonstrated mental delay. Regression analysis identified factors associated with severe critical illness and immaturity as the predictors across both domains. Conversely, common neonatal morbidities including hypoglycaemia, seizures, neonatal jaundice, and birth asphyxia were not independent predictors of delay in the unadjusted 6-month analysis [Table/Fig-5].

At the 12-month follow-up, the overall prevalence of delay had decreased substantially due to neurodevelopmental catch-up (45 infants identified with motor and 37 with mental delay). Despite this overall improvement, the profile of significant risk factors remained consistent. Infants with normal 6-month DASII scores had a 47.1% dropout rate, whereas only 24.4% of delayed infants were lost to follow-up. Mild to Moderate Delay (DASII 70-84) experienced a mid-tier dropout rate, contributing to an overall delay attrition of 40.2%.

After adjusting for confounding variables, prematurity, birth asphyxia and neonatal sepsis emerged as significant independent predictors for both motor and mental delay at 12 months. Birth asphyxia independent effect was confounded by prematurity, and in multivariable model, once the premature babies were removed, a term baby who suffered birth asphyxia was six times more likely to have motor delay (aOR: 6.15; 95% CI: 1.22-30.92) and five times more likely to have mental delay (aOR: 5.27; 95% CI: 1.28-21.72) at one year [Table/Fig-6].

DISCUSSION

This longitudinal cohort study demonstrated that high-risk SNCU graduates experience profound EUGR at discharge, followed by a discordant somatic catch-up over the first year of life. While weight normalised completely by 12 months, linear growth remained persistently stunted. Concurrently, neurodevelopmental assessment utilising the DASII revealed prematurity, neonatal sepsis and birth

Motor and mental delay at 6 months						
Perinatal risk factor	Motor delay identified in 124	Motor OR (95% CI)	p-value	Mental delay identified in 112	Mental OR (95% CI)	p-value
Preterm (<37 weeks)	95 (76.6%)	7.31 (3.37-15.86)	<0.0001	88 (78.6%)	6.05 (2.96-12.38)	<0.0001
Very low birth weight (<1500 g)	30 (24.2%)	13.09 (1.73-99.22)	0.0010	30 (26.8%)	19.02 (2.52-143.77)	<0.0001
Prolonged SNCU stay (>14 days)	51 (41.1%)	59.56 (3.58-990.02)	<0.0001	51 (45.5%)	89.60 (5.40-1487.21)	<0.0001
Neonatal sepsis	57 (46.0%)	4.25 (1.76-10.31)	0.0008	55 (49.1%)	4.72 (2.10-10.57)	<0.0001
Prolonged ventilation (>72 hrs)	26 (21.0%)	10.88 (1.43-82.85)	0.0032	24 (21.4%)	4.55 (1.30-15.86)	0.0121
Bronchopulmonary Dysplasia (BPD)	9 (7.3%)	6.99 (0.40-122.76)	0.1135	9 (8.0%)	9.82 (0.56-172.00)	0.0589
Hypoglycaemia	23 (18.5%)	1.37 (0.51-3.63)	0.6419	22 (19.6%)	1.61 (0.64-4.04)	0.3843
Neonatal seizures	7 (5.6%)	2.45 (0.29-20.54)	0.6809	7 (6.2%)	3.47 (0.42-28.93)	0.4385
Neonatal Jaundice (NNJ)	92 (74.2%)	0.90 (0.40-2.03)	0.8407	85 (75.9%)	1.24 (0.59-2.60)	0.5709
Birth asphyxia	35 (28.2%)	1.44 (0.63-3.32)	0.4264	29 (25.9%)	0.89 (0.43-1.84)	0.8507
Motor and mental delay at 12 months						
Perinatal risk factor	Motor delay identified in 45	Motor OR (95% CI)	Motor p-value	Mental delay identified in 37	Mental OR (95% CI)	Mental p-value
Preterm (<37 weeks)	38 (84.4%)	7.40 (2.79-19.65)	<0.0001	30 (81.1%)	4.29 (1.63-11.26)	0.0026
Very low birth weight (<1500 g)	18 (40.0%)	10.89 (2.94-40.33)	<0.0001	18 (48.6%)	18.00 (4.77-67.92)	<0.0001
Prolonged SNCU stay (>14 days)	36 (80.0%)	403.42 (22.76-7151.28)	<0.0001	34 (91.9%)	328.67 (52.27-2066.63)	<0.0001
Neonatal sepsis	26 (57.8%)	4.11 (1.73-9.73)	0.0017	24 (64.9%)	5.54 (2.27-13.52)	0.0001
Prolonged ventilation (>72 hrs)	15 (33.3%)	3.83 (1.34-10.98)	0.0130	13 (35.1%)	3.52 (1.29-9.62)	0.0208
Bronchopulmonary Dysplasia (BPD)	3 (6.7%)	8.65 (0.43-172.08)	0.0962	3 (8.1%)	12.28 (0.62-244.74)	0.0527
Hypoglycaemia	11 (24.4%)	2.48 (0.83-7.37)	0.1138	10 (27.0%)	2.80 (0.96-8.19)	0.0611

Neonatal seizures	4 (8.9%)	2.44 (0.43-13.99)	0.4114	4 (10.8%)	3.52 (0.61-20.24)	0.1978
Neonatal Jaundice (NNJ)	33 (73.3%)	1.34 (0.55-3.22)	0.6571	28 (75.7%)	1.56 (0.62-3.92)	0.3723
Birth asphyxia	13 (28.9%)	1.00 (0.42-2.42)	1	12 (32.4%)	1.32 (0.54-3.23)	0.6457

[Table/Fig-5]: Univariable logistic regression analysis for developmental delay.

Risk factor	Motor delay aOR (95% CI)	p-value	Mental delay aOR (95% CI)	p-value
Prematurity (<37 weeks)	19.86 (3.99-98.79)	0.0003	8.31 (2.01-34.34)	0.0034
Birth asphyxia	6.15 (1.22-30.92)	0.0275	5.27 (1.28-21.72)	0.0216
Neonatal sepsis	2.42 (0.86-6.83)	0.0951	4.05 (1.45-11.26)	0.0075
Prolonged ventilation (>72 hrs)	3.55 (0.93-13.63)	0.0645	2.33 (0.70-7.75)	0.1674

[Table/Fig-6]: Multivariable logistic regression analysis for developmental delay at 12 months.
aOR: Adjusted odds ratio; CI: Confidence interval

asphyxia as independent predictors of motor and mental delay at one year. The high incidence of EUGR observed in this cohort aligned with global neonatal network data, reflecting the massive protein-energy deficit incurred during critical illness [14]. The rapid weight catch-up observed, improving from 58.8% severe under nutrition at three months to 0% at 12 months, highlights the immense physiological resilience of these infants once complementary feeding is established and the acute catabolic state of the SNCU resolves. However, the persistence of stunting (~42% at 12 months) underscores a well-documented physiological paradigm: linear growth recovery lags significantly behind weight gain [15,16]. Chronic early-life stressors, such as systemic infections and prolonged respiratory support, trigger endocrine and inflammatory cascades that specifically suppress chondrocyte proliferation at the epiphyseal growth plate [17]. This leaves a permanent skeletal "scar" even after somatic fat and muscle are restored.

This study confirmed that the neurodevelopmental trajectory of the high-risk neonate is shaped by multiple, independent insults. Prematurity, sepsis and severe intrapartum hypoxic events emerged as independent risk factors of adverse neurodevelopmental outcome at 12 months corrected age. The wide CI observed is reflection of severe neurological deficit within the preterm subgroup, due to known vulnerability of the immature oligodendrocyte lineage and subependymal germinal matrix to germinal matrix haemorrhages and Periventricular Leukomalacia (PVL) [18,19].

Similarly, birth asphyxia increased the odds of motor delay and mental delay highlighting the global nature of hypoxic-ischaemic encephalopathy as previously identified by Constantin ILB et al., and Wallander JL et al., [20,21]. It triggers an immediate primary energy failure followed by a delayed, secondary neurotoxic cascade that indiscriminately damages both the basal ganglia (disrupting motor pathways) and the cerebral cortex (disrupting cognitive and mental pathways) [22]. Birth asphyxia independently predicted 12-month mental delay, explained by early hypoxic insult to the cerebral cortex clinically "unmasked" later in infancy, as the brain attempts to execute higher-order cognitive and language tasks [22].

A notable finding in this study is the domain-specific impact of clinical or culture-positive neonatal sepsis. While sepsis was associated with a substantial four-fold increase in the risk of severe mental delay, it failed to reach independent statistical significance for motor delay, a finding that closely mirrors the results reported by Mehdi SF et al., [17]. The systemic inflammatory response syndrome and blood-brain barrier disruption associated with neonatal sepsis may preferentially injure cortical and hippocampal networks rather than the primary motor cortex or corticospinal tracts [17]. Circulating pro-inflammatory cytokines are known to trigger microglial activation and astrogliosis, which selectively interrupt early synaptogenesis and axonal pruning, clinically manifesting as cognitive, language, and behavioural (mental) deficits rather than gross motor impairments [23].

Prolonged mechanical ventilation (>72 hours) trended toward an association with motor delay in the unadjusted analyses, but it lost statistical significance as an isolated, independent driver for both motor and mental delay in this cohort. This aligns with Raffay TM and Martin RJ who emphasised that its negative impact is largely a marker of the underlying clinical severity, driven by the respiratory distress of extreme prematurity or the severe depression of birth asphyxia [24].

Notably, the present study identified an inverse relationship between study attrition and neurodevelopmental impairment severity. Loss to follow-up was highest (47.1%) among infants with normal six-month DASII scores, while only 24.4% of delayed infants failed to return for their one-year assessment. Although significant dropout rates were frequently reported in existing literature for similar longitudinal cohorts [2,3,13,25,26], our pattern of differential retention protects against 'healthy survivor' bias. Because the present study cohort demonstrated a significantly higher retention rate among infants with developmental delays rather than those with normal DASII scores, this ensures accurate representation of risk factors within our high-risk infants and reinforces the internal validity of our 12-month neurodevelopmental outcomes.

The primary strength of this study is its prospective, longitudinal design with multiple, closely spaced anthropometry and neurodevelopmental assessment intervals at discharge, 3, 6, 9, and 12 months. This allowed for the precise mapping of growth velocity and the timing of somatic catch-up. Furthermore, the rigorous application of corrected age for all infants across growth charts and developmental scoring prevented the artificial over-diagnosing of stunting and microcephaly. The DASII tool ensured that motor and mental quotients were not skewed by the biases associated with western scales.

Limitation(s)

The study has its limitations, being a single tertiary centre design, it skewed the cohort toward high-acuity illnesses limiting generalisability to community settings. The significant attrition at one year follow-up (n=170 to n=97) driven by the region's challenging mountainous terrain and severe winters introduced potential selection bias. Finally, a 12-month follow-up is insufficient to detect subtle higher-order cognitive deficits, language delays, or autism spectrum traits, which typically manifest at 24 to 36 months.

CONCLUSION(S)

In conclusion, while high-risk neonates demonstrate remarkable somatic weight catch-up, they remain highly vulnerable to stunting and neurodevelopmental delays. The identification of risk factors of delay mandates improved antenatal care to prevent prematurity, newborn resuscitation trainings to labour room staff and an uncompromising enforcement of SNCU infection-control bundles, along with pre-emptive enrollment of the high-risk infants into DEIC immediately upon discharge. Future research should focus on extending longitudinal follow-up to minimum five years of age to accurately capture higher-order cognitive, language, and behavioural deficits. Additionally, studies should focus on efficacy of early DEIC intervention in mitigating these domain specific delays.

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PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Paediatrics, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India.
2. Professor, Department of Paediatrics, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India.
3. Clinical Psychologist, Department of Paediatrics, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Mangla Sood,
Professor, Department of Paediatrics, IGMC, Shimla, Himachal Pradesh, India.
E-mail: drmanglasood@gmail.com

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