

ORIGINAL ARTICLE

Non-oliguric hyperkalemia in neonates less than 30 weeks of gestation: clinical characteristics and outcomes

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ABSTRACT

Hyperkalemia is a frequent electrolyte imbalance in very preterm infants, often occurring in the first 72 hours of life. A unique entity, non-oliguric hyperkalemia (NOHK), is characterized by elevated serum potassium despite adequate urine output. While frequently reported, its risk factors, biochemical profile, and outcomes remain inadequately understood, particularly in low- and middle-income countries. To determine the incidence, maternal and neonatal risk factors, biochemical profile, and outcomes associated with NOHK in preterm neonates between 26⁺⁰ and 30⁺⁰ weeks' gestation, we conducted a retrospective cohort study in a tertiary neonatal intensive care unit (NICU) in South India. Infants born between 26⁺⁰ and 30⁺⁰ weeks' gestation and surviving beyond 12 hours during the 2-year study period were eligible. Infants born at <26⁺⁰ weeks were excluded due to variability in care practices during the study period, including the provision of comfort care. Other exclusions included

congenital renal anomalies, acute kidney injury, hemolyzed samples, and incomplete records. NOHK was defined as serum potassium >6.5 mmol/l within 72 hours with urine output ≥ 1 ml/kg/h. Maternal, perinatal, and neonatal variables were extracted. Outcomes included mortality and major morbidities. Statistical comparisons used Mann–Whitney *U* and Chi-square/Fisher's exact tests; multivariate logistic regression adjusted for gestational age, antenatal steroids, Doppler abnormalities, and hypocalcemia. Of the total 159 eligible infants, 59 (37.1%) developed NOHK. Lower gestational age and hypocalcemia (18.6% vs. 2%, OR 11.2, $p < 0.001$) were significant risk factors. NOHK infants had higher blood urea nitrogen, though creatinine and sodium were comparable. Mortality was significantly higher in NOHK (11.9% vs. 2%, $p = 0.001$) and remained independently associated after adjustment. Morbidities such as bronchopulmonary dysplasia, retinopathy of prematurity, intraventricular hemorrhage, and patent ductus arteriosus did not differ.

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KEYWORDS

Infant; Premature; Cohort; Electrolyte imbalance; Hyperkalemia; Neonatal Mortality.

INTRODUCTION

Hyperkalemia is one of the earliest electrolyte disturbances in very preterm and extremely low birth weight (ELBW) infants, with onset typically in the first 48–72 hours of life [1–3]. Unlike hyperkalemia due to acute kidney injury (AKI), many preterm infants develop non-oliguric hyperkalemia (NOHK), where urine output remains preserved. Incidence estimates range from 10% to 60%, depending on the population, potassium threshold used, and NICU practices [3–6].

The underlying mechanisms are multifactorial. Immaturity of Na⁺-K⁺-ATPase pumps reduces potassium uptake into cells [7,8]; combined with relative aldosterone resistance and reduced distal tubular secretion [9], this leads to extracellular potassium accumulation. Transient shifts of potassium during catabolic stress, hypoxia, or acidosis may further contribute [10,11].

While some reports suggest NOHK resolves spontaneously, others have linked it to arrhythmias, intraventricular hemorrhage (IVH), periventricular leukomalacia, and sudden death [3,12,13]. Antenatal steroids may reduce incidence by promoting cellular maturation [14], while antenatal magnesium exposure and hypocalcemia have also been implicated [15]. However, results remain inconsistent, and there is a lack of data from India and other low- and middle-income countries where resource constraints and variability in perinatal care may alter risk profiles.

This study aimed to evaluate the incidence, maternal and neonatal risk factors, biochemical profile, and short-term outcomes of NOHK among neonates born between 26+0 and 30+0 weeks' gestation admitted to a tertiary NICU in India.

MATERIALS AND METHODS

Study setting

The study was conducted in a level III NICU of a tertiary referral center in South India, admitting approximately 800–900 neonates annually, including a large proportion of very preterm and ELBW infants.

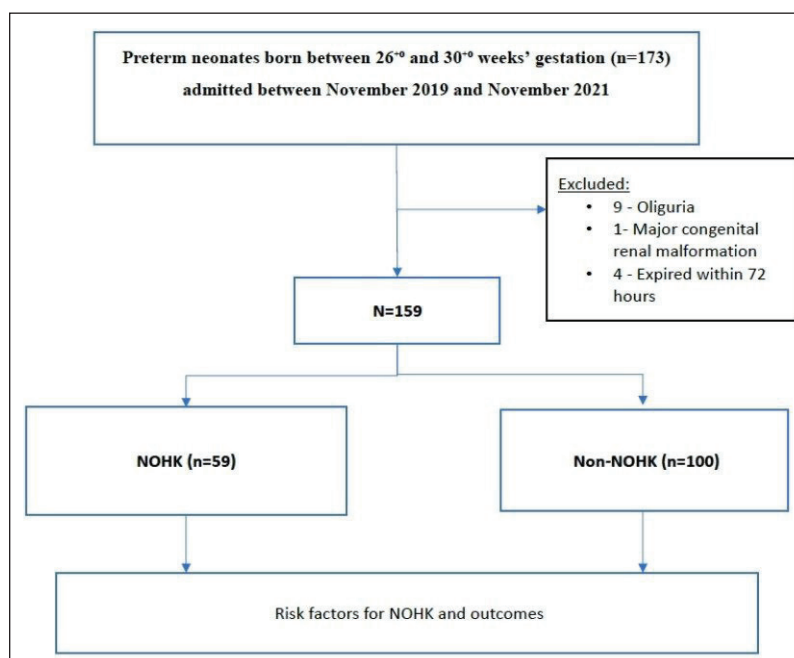


Figure 1. Flowchart of the study. NOHK, Non-oliguric hyperkalemia.

Study design and population

This was a retrospective analytical cohort study, and data were collected between April and June 2022 (Figure 1). We included neonates born between 26⁺⁰ and 30⁺⁰ weeks' gestation between November 2019 and November 2021 who were admitted to the NICU within 12 hours of birth and survived beyond 12 hours. Neonates born at <26⁺⁰ weeks were excluded due to variability in care practices during the study period, including the provision of comfort care. Additional exclusions included congenital anomalies of the kidney or urinary tract, evidence of acute kidney injury (AKI) as defined by the modified neonatal KDIGO criteria [16], hemolyzed serum potassium samples, and incomplete medical records. Hospital records of eligible infants were retrieved. Details regarding the blood sampling method (venous, arterial, or capillary; squeezed vs. free-flowing) were not consistently documented in the records and could not be systematically analyzed.

Definition of NOHK

- Serum potassium >6.5 mmol/l in two independent, non-hemolyzed blood samples drawn within a 4-hour interval.
- Urine output ≥ 1 ml/kg/h during the same period.

Controls were those with potassium within 3.5–6.0 mmol/l and no AKI.

Clinical management

As per NICU protocol, infants <30 weeks were started on glucose infusion with amino acids; potassium supplementation was added only after 48 hours if serum K⁺ <5 mmol/l and urine output was adequate. Hyperkalemia management included calcium gluconate (for cardiac membrane stabilization), insulin-dextrose infusion, sodium bicarbonate, beta-agonists, and dialysis if refractory [13,17,18].

Data collection

Clinicodemographic profile at birth was recorded, including period of gestation (GA), antenatal morbidities, perinatal exposure to antenatal steroids and magnesium sulfate, fetal Doppler,

mode of delivery, gender, BW, and any other significant antenatal event. The postnatal details included all recorded potassium levels, daily urine output, laboratory parameters such as urea, creatinine, sodium, bicarbonate, calcium levels, and hyperkalemia-induced electrocardiographic abnormalities. The management strategies for hyperkalemia were also documented. Other clinical comorbidities such as respiratory distress syndrome, patent ductus arteriosus (PDA), sepsis, IVH, necrotizing enterocolitis (NEC), and retinopathy of prematurity (ROP) were also documented. ROP screening was initiated at three weeks of life by an ophthalmologist and managed per clinical findings. Routine cranial ultrasonography was performed on admission, and if normal, repeated on days 3, 7, 14, 28, and at 36 weeks post-menstrual age or discharge. IVH was graded using the Papile classification, and follow-up imaging was based on IVH severity [19]. Bronchopulmonary dysplasia (BPD) was defined and graded according to the 2018 revised NICHD criteria [20]. NEC grade ≥ 2 was classified using modified Bell's criteria [21].

Statistical analysis

Continuous variables were expressed as medians with interquartile ranges and compared using the Mann–Whitney *U* test. Categorical variables were expressed as proportions and compared using the Chi-square test or Fisher's exact test as appropriate. Multivariate logistic regression was performed to identify independent predictors, including GA, antenatal steroids, abnormal Doppler, and hypocalcemia. Statistical significance was defined as $p < 0.05$. Analyses were performed using SPSS version 25.0

RESULTS

Cohort profile

Of 159 eligible neonates, 59 (37.1%) developed NOHK. Median GA was lower in NOHK infants (27.6 vs. 28.1 weeks, $p = 0.001$). BW (Table 1) did not differ significantly (1,018 vs. 1,000 g).

Maternal and antenatal factors

Antenatal steroid exposure was similar between groups (76% vs. 78%, $p = 0.74$). Antenatal

Table 1. Maternal and neonatal predictors of NOHK (N = 159).

Parameters		NOHK (K > 6.5) (N = 59)	Non NOHK (K < 6.5) (N = 100)	OR (95% CI)	p value
Maternal characteristics					
Primigravida		44 (74.6%)	61 (61%)	0.53 (0.26, 1.08)	0.08
Pregnancy-induced hypertension		4 (6.8%)	14 (14%)	0.44 (0.14,1.4)	0.20
Gestational diabetes mellitus		11 (18.6%)	14 (14 %)	1.4 (0.6,3.3)	0.5
Hypothyroid		7 (11.9%)	13 (13%)	0.9 (0.33,2.4)	1
Antenatal steroids	Complete	26 (44.1%)	49 (49%)	-	0.47
	Incomplete	28 (47.5%)	47 (47%)		
	Not given	5 (8.5 %)	4 (4%)		
Antenatal magnesium sulfate		24 (40.7%)	57 (57%)	0.51 (0.27,1)	0.051
Antenatal Doppler abnormality		6 (10.2%)	24 (24%)	0.35 (0.14,0.94)	0.04
Neonatal characteristics					
Gestational age Median (IQR)		27.6 (27.0, 28.6)	28.1 (26.6, 29)		0.44
BW Median (IQR)		1018 (860, 1160)	1000 (850.5, 1200)		0.85
Weight for gestational age	AGA	46 (78%)	79 (79%)	-	0.87
	SGA	12 (20.3 %)	20 (20%)		
	LGA	1 (1.7%)	1 (1%)		
Delayed cord clamping		33 (56 %)	60 (60 %)	0.85 (0.44, 1.6)	0.62
Required at least positive pressure ventilation in the delivery room		22 (37.3%)	36 (36%)	0.94 (0.49, 1.8)	0.87

AGA, appropriate for gestational age; CI, confidence interval; IQR, interquartile range; LGA, large for gestational age; NOHK, non-oliguric hyperkalemia; OR, odds ratio; SGA, small for gestational age. Bold values indicate statistically significant results ($p < 0.05$).

magnesium use showed no association. Doppler abnormalities were less frequent in NOHK (10.2% vs. 24%, OR 0.35, $p = 0.04$). Other maternal variables (pregnancy-induced hypertension, gestational diabetes mellitus, thyroid disease) showed no significant associations (Table 1).

Biochemical profile

NOHK infants had higher peak potassium levels (7.2 vs. 5.5 mmol/L). Hypocalcemia was more frequent in the NOHK group (18.6% vs. 2%; OR 11.2, $p < 0.001$). Blood urea nitrogen levels were higher (40 vs. 32 mg/dL, $p = 0.003$) in the NOHK group, while creatinine, sodium, and bicarbonate levels were comparable between groups (Table 2).

Outcomes

Mortality was significantly higher in NOHK (11.9% vs. 2%, $p = 0.001$), independent of GA and antenatal factors. NEC occurred exclusively in non-NOHK (8% vs. 0%, $p = 0.03$). Other morbidities (IVH, PDA, BPD, ROP) showed no significant difference. Median NICU stay and duration of ventilation were comparable (Table 3).

DISCUSSION

We observed NOHK in 37% of neonates born between 26⁺⁰ and <30⁺⁰ weeks' gestation, consistent with earlier reports from Western and Asian cohorts [3,5,22]. This high incidence

Table 2. Clinical and biochemical parameters of the study population (N=159).

Parameters	NOHK (K > 6.5) (N = 59)	Non NOHK (K < 6.5) (N = 100)	OR (95% CI)	p value
Delivery room CPAP	11 (18.6%)	17 (17%)	1.1 (0.48, 2.5)	0.83
Intubation in the delivery room	21 (35.6%)	32 (32%)	1.1 (0.6, 2.3)	0.73
Respiratory acidosis	22 (37.3%)	34 (34%)	1.1 (0.6, 2.2)	0.73
Duration of invasive ventilation (hours), Median (IQR)	12.0 (0.0, 24.0)	17.0 (4.0, 51.7)		0.55
Need for invasive ventilation	28 (47.5%)	58 (58%)	0.65 (0.34, 1.25)	0.25
Need for surfactant	40 (67.8%)	61 (61%)	1.35 (0.68, 2.65)	0.5
Need for >1 dose of surfactant	4 (6.8%)	10 (10%)	0.65 (0.19, 2.1)	0.57
BUN, Median (IQR)	40 (32, 49)	32.0 (23.0, 28.7)		0.003
Creatinine, Median (IQR)	0.8 (0.7, 0.9)	0.8 (0.7, 0.9)		0.7
Potassium, Median (IQR)	7.2 (7.0, 7.7)	5.5 (4.8, 6.1)		<0.001
Bicarbonate, Median (IQR)	17 (16, 18)	18 (16, 18)		0.88
Sodium, Median (IQR)	137(135, 142)	138(135, 141)		0.71
Calcium, Median (IQR)	8.2(7.3, 9.1)	8.6(7.5, 9.1)		0.15
Hypocalcemia	11 (18.6 %)	2 (2%)	11.2 (2.4, 52.6)	<0.001

BUN, Blood urea nitrogen; CI, confidence interval; CPAP, continuous positive airway pressure; IQR, interquartile range; NOHK, non-oliguric hyperkalemia; OR, odds ratio. Bold values indicate statistically significant results ($p < 0.05$).

Table 3. Outcomes in the study population (N = 159).

Parameters	NOHK (K > 6.5) (N = 59)	NON NOHK (K < 6.5) (N = 100)	OR (95% CI)	p-value
Expired	7 (11.9%)	2 (2%)		0.001
Duration of hospital stay (days), Median (IQR)	23.0 (12.0, 36.0)	23.5 (9.2, 37.5)		0.91
Duration of invasive ventilation (hours), Median (IQR)	10.0 (0.0, 24.0)	7.5 (0.0, 26.25)		0.24
Duration of any respiratory support, Median (IQR)	6.0 (4.0, 19.0)	8.0 (5, 20.8)		0.14
Apnea	38(64.4%)	65 (65%)	0.97 (0.5, 1.9)	1
PDA requiring treatment	5 (8.5%)	9 (9%)	0.94 (0.3, 2.9)	1
Necrotising enterocolitis	0 (0%)	8 (8%)		0.03
BPD	0 (0)	1 (1%)		1
Retinopathy of prematurity	11 (18.6%)	19 (19%)	0.97 (0.43, 2.2)	1
IVH	4 (6.8%)	2 (2%)	3.5 (0.63, 20)	0.2

CI, confidence interval; IQR, interquartile range; NOHK, non-oliguric hyperkalemia; OR, odds ratio.

[§]Adjusted for gestational age, antenatal steroids, antenatal magnesium sulfate, and hypocalcemia. Bold values indicate statistically significant results ($p < 0.05$).

underscores the vulnerability of very preterm infants to metabolic derangements in the immediate postnatal period.

Lower GA was a key risk factor, consistent with Yuan et al. [6]. Hypocalcemia emerged as a strong correlate, supporting previous work by Thayyil et al. [23]. The mechanism may involve impaired calcium–potassium regulation in immature tissues. Antenatal steroids, though protective in some studies [14,24], showed no benefit here, possibly due to incomplete or late dosing. Contrary to the prior report [22], antenatal magnesium exposure was not associated with NOHK. The observed lower frequency of antenatal Doppler abnormalities among infants with NOHK, despite statistical significance, is difficult to explain biologically. Given the absence of a plausible mechanistic link and inconsistent reporting in the literature, this finding is most likely attributable to chance or unmeasured confounders inherent to retrospective analyses.

Early provision of parenteral amino acids has been shown to reduce the risk of NOHK by reversing the immediate postnatal catabolic state, promoting intracellular protein synthesis and potassium uptake, and thereby lowering extracellular potassium levels [25]. Many neonatal nutrition guidelines recommend initiating amino acids early and advancing rapidly to target intakes of 2–3 g/kg/day in the first days to blunt protein catabolism and its metabolic consequences [26–28]. In our unit, amino acids were initiated at 1.5 g/kg/day and increased to 3 g/kg/day; nevertheless, NOHK was still observed. Several factors may explain this. First, the timing and speed of reaching an effective amino-acid dose are important. A lower starting dose (1.5 g/kg/day) delays the anabolic effect compared with protocols that start at ≥ 2 g/kg/day, so very immature infants may remain in a catabolic state long enough for transcellular potassium shifts to occur. Second, infants <28 weeks' gestation have profoundly immature cellular Na^+/K^+ -ATPase activity and renal tubular secretion, which may require a longer period or higher relative protein delivery to restore intracellular potassium uptake. Third, concomitant metabolic stressors, such as hypoxia, acidosis, hypocalcemia, and high catabolic load, can overwhelm the benefit of amino acids and precipitate NOHK despite nutritional measures

[29]. Finally, population- and protocol-specific factors, including the potassium/amino-acid ratio in parenteral fluids, delayed initiation of optimal TPN, limited phosphorus or sodium delivery, and intercurrent illness, may modify electrolyte handling and reduce the protective effect of early amino-acid supplementation [26,27]. The occurrence of NOHK despite early amino acid supplementation may indicate an underlying cellular immaturity and hypercatabolic state, reflected by higher BUN levels in affected infants.

Mortality in NOHK infants (11.9%) was striking. Prior studies have shown potassium levels >7 mmol/l predispose to arrhythmia, IVH, and sudden death [3,30]. Increased mortality in NOHK infants as in other studies might be due to rapid potassium shifts precipitating arrhythmias and circulatory collapse. Although IVH and BPD rates did not differ significantly, trends toward increased IVH have been observed. Interestingly, NEC was absent in NOHK, likely due to early deaths before gastrointestinal complications developed.

Our findings highlight that NOHK is not a benign biochemical phenomenon but an independent predictor of death. Close electrolyte monitoring is essential, particularly in infants <28 weeks. Aggressive but safe correction strategies should be standardized across NICUs. Preventive strategies, including optimizing antenatal steroid coverage and early initiation of optimal total parenteral nutrition, warrant emphasis.

Strengths include a strict case definition, exclusion of hemolyzed samples, and multivariate adjustment. Limitations of this study include its retrospective, single-center design, which restricted the availability of certain clinical details, such as whether blood samples were squeezed or obtained as free-flowing, and thereby may have influenced potassium measurements. Electrocardiographic correlation for arrhythmias was not consistently available. The relatively small sample size also limits generalizability. Although our cohort included extremely preterm infants (<28 weeks' gestation), their number was relatively small. As this subgroup is at the highest risk for NOHK due to profound immaturity of Na^+/K^+ -ATPase activity and renal potassium handling, the limited sample size may restrict

the precision of our estimates for this gestational bracket. Larger multicenter prospective studies with adequate representation of <28-week neonates are needed to confirm these findings and to better delineate incidence, risk factors, and outcomes in this most vulnerable group.

CONCLUSION

NOHK is common in neonates <30 weeks and independently predicts mortality despite preserved urine output. Vigilant biochemical surveillance and early interventions are critical to improving outcomes in this vulnerable population.

LIST OF ABBREVIATIONS

AKI	Acute kidney injury
BPD	Bronchopulmonary dysplasia
BW	Birth weight
BUN	Blood urea nitrogen
ELBW	Extremely low birth weight
GA	Gestational age
IVH	Intraventricular hemorrhage
KDIGO	Kidney Disease: Improving Global Outcomes
LMICs	Low- and middle-income countries
Na ⁺ /K ⁺ -ATPase	Sodium–potassium adenosine triphosphatase
NEC	Necrotizing enterocolitis
NICHHD	National Institute of Child Health and Human Development
NICU	Neonatal intensive care unit
NOHK	Non-oliguric hyperkalemia
OR	Odds ratio
PDA	Patent ductus arteriosus
ROP	Retinopathy of prematurity
TPN	Total parenteral nutrition

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

FUNDING

None.

CONSENT FOR PUBLICATION

Not applicable.

ETHICS APPROVAL

The study was approved by IHEC (IHEC-I/0839/22) and was registered in Clinical Trial Registry India (CTRI/2022/03/041680). The data analysis was conducted retrospectively, and confidentiality was maintained at all levels.

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