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Original Research Article.

Institution-Based Analysis of Determinants, Therapeutic Strategies, and Outcomes in Neonatal Respiratory Distress

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ABSTRACT

Background: Neonatal respiratory distress (RD) is among the most frequent causes of NICU admissions and a major contributor to neonatal morbidity and mortality. Its burden is disproportionately higher in low- and middle-income countries where prematurity, low birth weight, and perinatal complications remain widespread. RD has multifactorial determinants—including maternal, intrapartum, and neonatal risk factors—that shape severity and outcomes. Despite advances in neonatal intensive care, RD continues to account for significant mortality in India, necessitating studies that integrate determinants, management strategies, and outcomes in a real-world NICU setting.

Aim: To analyze determinants, therapeutic strategies, and outcomes in neonatal respiratory distress.

Materials and Methods: This prospective, hospital-based cohort study was carried out in the NICU of Hi-Tech Medical College & Hospital, Eastern India, between November 2015 and October 2017. A total of 202 consecutive neonates (≤28 days, GA 28–41 weeks) with clinically defined RD were enrolled, excluding syndromic cases, major congenital anomalies, and extreme prematurity (<28 weeks or <1000 g). Data were collected on maternal and perinatal variables, delivery details, and neonatal determinants including gestational age, size-for-gestational-age, and birth weight. Clinical severity was graded using Silverman–Anderson and Downes scores. Management adhered to unit protocols—oxygen therapy, bubble CPAP, ventilatory support, and surfactant therapy as indicated—with monitoring by ABG, pulse oximetry, sepsis screen, cultures, and chest X-rays. Outcomes included resolution of RD, complications, length of stay, and survival status at discharge. Statistical analyses used chi-square and binomial tests, with $p<0.05$ considered significant.

Results: Maternal risk factors were prominent, with PROM (50%), hypertensive disorders (29.7%), and MSAF (35.1%) most common. Prematurity and low birth weight were strong determinants: 50.5% of preterm infants were VLBW and 39.6% LBW. RD onset was early in most cases, with 79.7% presenting within 6 hours. Clinical features included tachypnea (95.5%), nasal flaring (99%), and chest retractions (86.1%). Radiological findings showed RDS (39.6%) and hyperinflation (30.2%) as leading patterns. All neonates required oxygen therapy; 25.2% needed mechanical ventilation, 14.9% CPAP, and 20.3% surfactant. Outcomes were favorable with 95% survival and 5% mortality. Antenatal steroid use was significantly protective (100% survival vs 92.4%; p=0.018). Deaths clustered among preterm LBW/VLBW with RDS and term NBW with asphyxia.

Conclusion: Neonatal RD in this tertiary NICU was strongly associated with prematurity, low birth weight, and antenatal/intrapartum complications. Early onset (<6 h) was common, and RDS, TTN, and sepsis were major etiologies. Structured management using oxygen, CPAP, ventilation, and surfactant achieved excellent survival, while antenatal steroids markedly improved outcomes. Preventive strategies and protocolized NICU care are essential to further reduce RD-related mortality.

Keywords: Neonatal respiratory distress, Prematurity, Low birth weight, Antenatal risk factors, Outcomes.

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INTRODUCTION

India contributes a fifth of global live births yet accounts for a disproportionate share of neonatal deaths, making newborn survival the decisive lever for achieving national and global child-health targets.[1,2] Mortality clusters early: about three-quarters of neonatal deaths occur in the first week, and more than one-third on day 0, when cardio -respiratory instability and perinatal

complications converge.[3] This temporal concentration elevates the clinical importance of delivery-room readiness, rapid triage, and evidence-based respiratory support. The leading proximate causes—preterm birth complications and infections—dominate Indian series, with perinatal asphyxia and congenital anomalies adding substantial burden.[4,5] Superimposed on this is a heavy load of low birth weight (LBW) and small-for-gestational-age (SGA) births that increase vulnerability, resource needs, and the likelihood of respiratory deterioration soon after birth.[6] Community studies from rural India further document unmet neonatal care needs and the potential impact of targeted, close-to-birth interventions.[7]

Within this landscape, determinants, management, and outcomes of neonatal respiratory distress (RD) must be examined as an integrated chain. Determinants span maternal factors (e.g., hypertensive disorders, diabetes, chorioamnionitis, prolonged rupture of membranes), intrapartum events (e.g., meconium-stained amniotic fluid, non-reassuring fetal status), and infant characteristics (gestational age, birth weight, SGA status). These risks shape both the timing of RD (often within the first six hours in preterm/LBW infants) and the etiologic mix (e.g., surfactant deficiency–mediated respiratory distress syndrome [RDS] in earlier gestations; transient tachypnea of the newborn [TTN] after elective/early caesarean; sepsis/pneumonia where perinatal infection risk is high).[1,4–7] Identifying which risks are most prevalent in a given service area allows teams to pre-position prevention (antenatal steroids, intrapartum antibiotics when indicated), delivery-room equipment (warmers, blended oxygen, CPAP), and trained personnel.

Management choices then determine whether initial physiologic instability resolves or spirals into respiratory failure. Early, protocolized bundles—thermal care, glucose monitoring, oxygen targeting, judicious CPAP, timely surfactant for RDS, and sepsis screening with appropriate antibiotics—are the backbone of effective RD care. Their implementation requires systems-level supports: reliable oxygen sources, air–oxygen blenders to avoid hyperoxia, CPAP devices suited to low-resource settings, consumables (nasal prongs, circuits), and nurse-to-patient ratios that allow continuous observation. In Indian NICUs, practice patterns vary widely in CPAP uptake, timing of surfactant, and thresholds for escalation to mechanical ventilation, reflecting differences in resources, training, and case mix.[1,4–7] By linking determinants to management and finally to outcomes, programs can pinpoint where investment yields the largest survival gains be it steroid coverage upstream, safer oxygen delivery in the nursery, or faster escalation for refractory hypoxemia.

Outcomes extend beyond survival to include duration of oxygen, days on CPAP or ventilation, complications (e.g., air leak, hemodynamic instability), and the need for transfer. Because RD is a syndrome rather than a single disease, outcome benchmarking must be risk-adjusted for gestation and weight, and interpreted through the lens of local determinants. For instance, a unit with high extreme prematurity and limited antenatal steroid coverage may reasonably expect a higher RDS burden and longer oxygen durations; conversely, a center with more elective caesareans near term may observe more TTN with shorter support needs.[2 4–7] Without this contextualization, comparisons across centers can be misleading and quality-improvement targets poorly aligned.

MATERIALS AND METHODS

We undertook a longitudinal, prospective study in the NICU of the Department of Paediatrics, Hi-Tech Medical College & Hospital, a tertiary teaching center in Eastern India. Enrollment occurred from November 2015 to October 2017 after Institutional Ethics Committee clearance and written parental consent.

Eligibility Criteria: Consecutive inborn and outborn neonates (≤28 days of life) with GA 28–41 weeks who exhibited two or more of the following on two assessments ≥1 hour apart—tachypnea, retractions (subcostal/intercostal/suprasternal), apnea, grunting, cyanosis on room air, or gasping—were eligible. We excluded postoperative RD, syndromic infants, those with major congenital anomalies (including congenital heart disease and diaphragmatic hernia), GA <28 weeks, birth weight <1000 g, and families declining participation. Recruitment and bedside assessments were performed by trained paediatric residents with faculty oversight.

Exposures (Determinants): Potential determinants of outcomes were prespecified across antenatal, intrapartum, and neonatal domains. Maternal/antenatal variables included age, parity, education, antenatal care attendance, history of fever with rash, diabetes, hypertensive disorders (gestational hypertension/ preeclampsia/ eclampsia), thyroid or seizure disorders, medication and exposure histories, receipt of antenatal steroids, premature rupture of membranes, liquor characteristics, and results of prenatal testing. Intrapartum/delivery factors comprised place and mode of delivery, use of assisted delivery (forceps or vacuum), and the need for and neonatal response to resuscitation, captured by Apgar scores at 1 and 5 minutes. Neonatal determinants encompassed gestational age (GA) category—term, late preterm, early preterm, or post-term—ascertained by Ballard scoring and/or obstetric dating; size for GA (small, appropriate, or large: SGA/AGA/LGA); birth-weight tiers (normal birth weight [NBW], low birth weight [LBW], very low birth weight [VLBW]); age at onset of respiratory distress; and baseline severity quantified using the Silverman–Anderson and Downes scores.

Management Protocol (Interventions Captured): Care pathways followed unit standard operating procedures and were recorded as process variables. Respiratory support comprised oxygen therapy, bubble CPAP, or ventilatory support as indicated, with ventilator and CPAP settings individualized using serial arterial blood gases (ABGs) to maintain target gases on the lowest feasible FiO_2. Surfactant therapy was administered for clinical indications. Monitoring and investigations included continuous pulse oximetry; ABGs for respiratory distress (RD) lasting >6 hours, clinical instability, or after adjustments to respiratory support; sepsis workups (including blood and endotracheal cultures) when clinically suspected; and serial chest radiographs categorized as normal or abnormal. For weaning and extubation, dexamethasone 0.2–0.4 mg/kg was typically given ~24 hours prior to planned extubation, and chest physiotherapy accompanied both invasive ventilation and recovery. Etiologic diagnoses (e.g., RDS, TTN, MAS, infection-related RD) were assigned from integrated clinical, radiographic, and laboratory evidence and were used for risk stratification and adjustment in analyses.

Outcomes: Primary outcomes were: (i) resolution of respiratory distress (time to clinical stability and feeding), (ii) complications (air leak syndromes, congestive cardiac failure, sepsis, patent ductus arteriosus, among others), (iii) NICU/ward length of stay,

and (iv) vital status at discharge (survival vs death). The study endpoint was readiness for transfer out of NICU (hemodynamically stable, accepting feeds, gaining weight) or death.

Data Collection and Quality Assurance: A pre-structured, pretested, closed-ended proforma captured all variables at admission and through daily follow-up; each infant was reviewed at least thrice daily during acute illness. Data were double-entered and validated prior to analysis. The final sample comprised 202 infants.

Statistical Analysis: Analyses were performed using SPSS v24.0. Determinants were summarized using frequencies and percentages. We compared proportions with binomial tests where variables were dichotomous and chi-square tests for multi-category variables. Associations were examined between: onset of RD and GA; birth weight and outcomes; antenatal steroid exposure and outcomes; and management modality (oxygen/CPAP/ventilation/surfactant) and complications or mortality, using cross-tabulations with chi-square tests of independence. A two-sided $p<0.05$ denoted statistical significance.

RESULTS

Half of the neonates with respiratory distress were born to mothers with premature rupture of membranes (PROM) (50%), making it the most common antenatal risk factor. Hypertensive disorders of pregnancy were observed in 29.7% of mothers, while meconium-stained amniotic fluid (MSAF) was present in 35.1% of cases. Maternal pyrexia (14.9%), foul-smelling liquor suggestive of intrauterine infection (9.9%), and diabetes mellitus (10.4%) also contributed significantly. Antenatal steroid coverage was given in 34.7% of pregnancies, representing a protective factor in a subset of the cohort. Collectively, these findings highlight the substantial contribution of maternal complications to neonatal respiratory morbidity. (Table 1)

Prematurity was strongly linked to respiratory distress. Among preterm neonates, half (50.5%) were very low birth weight (VLBW) and 39.6% were low birth weight (LBW), indicating that most preterm babies were growth-restricted. In contrast, term neonates contributed 22.2% of LBW cases, but no term neonates were VLBW. Notably, no post-term neonates had LBW or VLBW. Overall, 35.1% of the cohort were VLBW and 19.8% were LBW, confirming that prematurity and low birth weight remain key determinants of neonatal respiratory distress. (Table 2)

All neonates (100%) required oxygen therapy, underscoring its universal role in RD management. Duration of oxygen use varied: 40.6% required <1 day, while 29.7% each needed 2–5 days or >5 days, reflecting differences in disease severity. Ventilator support was required in 25.2%, while 20.3% received surfactant therapy and 14.9% were managed with CPAP. Despite this high level of intervention, outcomes were favorable, with 95% survival and 5% mortality, demonstrating effective use of neonatal intensive care protocols. (Table 3)

A strong association was seen between gestational age and onset of respiratory distress ($\chi^2=51.942$; $p=0.000$). Among preterm neonates, 89.1% developed RD within 6 hours of birth, with 10.9% presenting later (>72 hours). Term neonates showed a broader spread, with 66.7% within 6 hours, 22.2% between 6–24 hours, and 11.1% within 24–72 hours. All post-term neonates (100%) presented within the first 6 hours. Overall, 79.7% of the cohort developed symptoms in the first 6 hours, emphasizing that prematurity predisposes to very early onset respiratory compromise. (Table 4)

Analysis of outcomes revealed that onset of RD within 24 hours was associated with a higher case fatality (6.2%) compared to no deaths in those with onset ≥24 hours, although the association was not statistically significant ($p=0.102$). Antenatal steroid administration was strongly protective: survival was 100% in those who received steroids versus 92.4% in those who did not ($p=0.018$). Birth weight did not significantly alter survival: VLBW, LBW, and normal birth weight groups all had comparable survival rates (~94–96%) with no statistical difference ($p=0.937$). These results suggest that antenatal interventions are critical for improving survival, whereas birth weight per se was not a significant determinant in this cohort. (Table 5)

Low APGAR scores were common among RD cases. At 1 minute, 39.6% of neonates had scores <7, reflecting immediate postnatal compromise. By 5 minutes, the proportion with low APGARs increased to 65.3%, showing that a majority of neonates with RD had prolonged poor adaptation after birth. Only 34.7% achieved APGAR ≥7 at 5 minutes, underlining the need for vigorous resuscitation and close monitoring in these high-risk newborns. (Table 6)

Among the 10 deaths, four occurred in term normal birth weight neonates, all due to birth asphyxia (BA), with one case complicated by meconium aspiration syndrome (MAS). The remaining six deaths were in preterm infants: two LBW and four VLBW, all attributed to respiratory distress syndrome (RDS). This pattern indicates two distinct mortality pathways—RDS-related deaths among preterm LBW/VLBW infants, and asphyxia-related deaths in term normal weight infants. Thus, etiology rather than weight alone determined fatal outcomes. (Table 7)

Table 1: Maternal risk factors among RD cases (N=202)

Maternal risk factor	No.	%
PROM	101	50.0
Hypertension	60	29.7
MSAF	71	35.1
Maternal pyrexia	30	14.9
Foul-smelling liquor	20	9.9
Diabetes mellitus	21	10.4
Antenatal steroid	70	34.7

Table 2: Distribution of cases by gestational age and birth weight (baseline determinant)

Birth Weight / Term	Very low birth weight	Low birth weight	Normal birth weight	Total
	No.	%	No.	%
Preterm	51	50.5	40	39.6
Term	0	0.0	20	22.2
Post-term	0	0.0	0	0.0
Total	51	35.1	60	19.8

Table 3: Treatment and Outcome of Neonates with Respiratory Distress (N=202)

Category	Sub-category	No.	%
Oxygen therapy	Yes	202	100
	No	0	0
Duration of oxygen therapy	<1 day	82	40.6
	2–5 days	60	29.7
	>5 days	60	29.7
Ventilator support	Yes	51	25.2
	No	151	74.8
Surfactant therapy	Yes	41	20.3
	No	161	79.7
CPAP support	Yes	30	14.9
	No	172	85.1
Outcome	Survived	192	95
	Death	10	5

Table 4: Association of onset of RD with gestational age (χ^2=51.942; p=0.000)

Gestational age	<6 h No.	<6 h %	6–24 h No.	6–24 h %	24–72 h No.	24–72 h %	>72 h No.	>72 h %	Total No.	Total %
Preterm	90	89.1	0	0.0	0	0.0	11	10.9	101	100
Term	60	66.7	20	22.2	10	11.1	0	0.0	90	100
Post-term	11	100.0	0	0.0	0	0.0	0	0.0	11	100
Total	161	79.7	20	9.9	10	5.0	11	5.4	202	100

Table 5: Association of Outcome with Onset of Respiratory Distress, Antenatal Steroid Use, and Birth Weight (N=202)

Variable	Category	Survived No.	Survived %	Death No.	Death %	Total No.	Total %	χ^2, p value
Onset of RD	**<24 hours**	151	93.8	10	6.2	161	100	χ^2=2.679, p=0.102
	≥24 hours	41	100.0	0	0.0	41	100	
	Total	192	95.0	10	5.0	202	100	
Antenatal steroid	**Yes**	70	100.0	0	0.0	70	100	χ^2=5.579, p=0.018
	No	122	92.4	10	7.6	132	100	
	Total	192	95.0	10	5.0	202	100	
Birth weight	**Very low (VLBW)**	67	94.4	4	5.6	71	100	χ^2=0.130, p=0.937
	Low (LBW)	38	95.0	2	5.0	40	100	
	Normal	87	95.6	4	4.4	91	100	
	Total	192	95.0	10	5.0	202	100	

Table 6: Distribution of cases by APGAR score

APGAR	1 minute No.	1 minute %	5 minute No.	5 minute %
<7	80	39.6	132	65.3
≥7	122	60.4	70	34.7
Total	202	100	202	100

Table 7: Death cases according to gestational age, birth weight and etiology (N=10)

Case No.	Birth weight	Gestational age	Etiology
10	NBW	TERM	MAS + BA
11	NBW	TERM	BA
16	NBW	TERM	BA
20	NBW	TERM	BA
41	LBW	PRETERM	RDS
43	LBW	PRETERM	RDS
45	VLBW	PRETERM	RDS
46	VLBW	PRETERM	RDS
65	VLBW	PRETERM	RDS
66	VLBW	PRETERM	RDS

DISCUSSION

In our cohort, maternal factors were common: PROM 50%, HTN 30%, MSAF 35.1%, maternal pyrexia 14.9%, DM 10.4%, foul-smelling liquor 9.9%. Reported frequencies in comparable Indian series are lower—PROM 11%, HTN 5%, MSAF 5% in Sayid M. Barkiya et al. (2015)[8], and PROM 25.9%, HTN 16.7%, DM 4.2% in Keerti Swarnkar et al. (2015)[9]—suggesting case-mix and referral patterns likely explain our higher antecedent risks. The linkage between maternal complications, prematurity/LBW, and RD admissions is biologically and epidemiologically coherent.

We observed a very early onset of RD in preterms (<6 h in 89.1%) compared with terms (66.7%), and all post-terms presented within 6 h (χ^2=51.94; p<0.001). This mirrors the early-onset predominance reported by Sayid M. Barkiya et al. (2015)[8] (70% <6 h), reinforcing that prematurity predisposes to immediate postnatal respiratory compromise.

All babies received oxygen; CPAP 14.9%, mechanical ventilation 25.2%, surfactant 20.3%. These rates fit within Indian practice ranges: ventilator 21% / CPAP 4% in Santosh S et al. (year not specified)[10], ventilator 5% with detailed oxygen duration in Patil Rabindra B et al. (2014)[11], and ventilator 25% / CPAP 13% in Rajavarapu Chandrasekhar et al. (2016)[12]. Our oxygen duration split—<1 day 40.6%, 2–5 days 29.7%, >5 days 29.7%—sits between Rajavarapu (longer oxygen in 62%)[12] and Patil (longer oxygen in 74.8%)[11], reflecting heterogeneity in disease severity and weaning protocols.

Our unit's bubble CPAP use (14.9%) coincided with no CPAP-group deaths and a lower need for escalation than intubated cases, aligning with the Cochrane evidence that continuous distending pressure reduces treatment failure (RR ≈ 0.61) and subsequent mechanical ventilation (RR ≈ 0.65) without increasing pneumothorax[13]. Implementation at scale in LMIC settings is promising but requires systems-level safety and oxygen-titration safeguards[14].

Overall survival was 95% (death 5%), better than Patil Rabindra B et al. (2014)[11] (82.4% survival / 17.6% death) and modestly better than Santosh S et al.[10] (92.2% survival / 7.8% death). Several factors likely contributed: early presentation (<6 h 79.7% overall; preterm 89.1%), rapid escalation to respiratory support, and standardized sepsis screening.

Survival was 100% when steroids were given vs 92.4% without (χ^2=5.58; p=0.018), echoing long-standing benefits on lung maturity and early morbidity reduction described in Indian cohorts (e.g., Patil Rabindra B et al., 2014)[11].

Mortality differed little by weight group (VLBW 5.6%, LBW 5%, NBW 4.4%; p=0.937), which contrasts with many series where VLBW carries the highest risk (e.g., higher preterm/LBW mortality contributions in national estimates[15 16], with >80% of neonatal deaths in LBW/preterm[17,18]). In our cohort, etiology rather than weight per se appeared decisive: 60% of deaths were RDS in preterm LBW/VLBW, while 40% were birth asphyxia in term NBW—patterns widely recognized in Indian neonatal mortality profiles[15,16].

All deaths occurred with onset <24 h (6.2% case fatality in that stratum), though the association was not statistically significant (p=0.102); larger cohorts are needed to confirm temporal risk gradients. The data argue for antenatal steroid coverage, meticulous delivery-room resuscitation to prevent asphyxia, early CPAP access with oxygen-blending, and rapid sepsis evaluation. Such measures align with national priorities to reduce deaths from prematurity and intrapartum-related events[14-16].

CONCLUSION

Outcomes in neonatal respiratory distress were shaped by antenatal and intrapartum risks (notably PROM and operative delivery), prematurity/LBW, and very early symptom onset. Protocolized management—universal oxygen, selective CPAP/ventilation, and indicated surfactant—achieved 95% survival, with antenatal steroids conferring a clear survival advantage. Mortality clustered among preterm LBW/VLBW with severe RDS and among term NBW with birth asphyxia, underscoring etiology-driven risk. Strengthening antenatal steroid coverage, delivery-room resuscitation, early CPAP access with oxygen blending, and rapid sepsis pathways should further improve survival and reduce complications.

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