Original Research

INCIDENCE AND RISK FACTORS OF RETINOPATHY OF PREMATURITY IN BABIES WEIGHING <1800 G; WITH SPECIAL REFERENCE TO THE BABIES WEIGHING BETWEEN 1501 G & 1800 G: A CLINICAL STUDY IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Retinopathy of prematurity (ROP) is a vaso-proliferative disorder of the developing retina and a leading cause of preventable blindness in infants. With improving neonatal intensive care in developing countries, the incidence of ROP in infants with relatively higher birth weights, including the 1501–1800 g range, is increasingly recognized. Data specific to this weight category from eastern India are limited.

Objective: To determine the incidence of ROP and identify associated perinatal and neonatal risk factors among infants weighing less than 1800 g, with special reference to the 1501–1800 g subgroup, in a tertiary care hospital.

Methods: A prospective, observational, hospital-based study was conducted over 12 months (March 2019 – February 2020) at the ROP clinic of Nil Ratan Sircar Medical College and Hospital, Kolkata. Two hundred preterm infants with birth weight <1800 g underwent serial ophthalmic examinations using binocular indirect ophthalmoscopy. Data on oxygen exposure, metabolic parameters, perinatal variables, and neonatal morbidities were recorded and analyzed using Student's t-test, chi-square/Fisher's exact test, and multivariate logistic regression (SPSS v26.0).

Results: ROP was detected in 56 of 200 infants (28.0%). In the 1501–1800 g subgroup, 26 of 105 infants (24.76%) developed ROP. Stages 1 and 2 accounted for 89.3% of cases; no infant had plus disease or aggressive posterior ROP. Significant risk factors included oxygen therapy (higher maximum SpO_2, lower minimum SpO_2, longer duration), hyperglycemia, low APGAR scores at 1 and 5 minutes, respiratory distress syndrome, apnea of prematurity, septicemia, anemia, and need for blood transfusion (all $p<0.05$). Surfactant therapy, necrotizing enterocolitis, intraventricular hemorrhage, patent ductus arteriosus, and maternal factors were not significantly associated.

Conclusion: ROP affects a clinically significant proportion of infants weighing 1501–1800 g, a group currently excluded from standard screening guidelines. Oxygen management, metabolic stability, and vigilant NICU monitoring are the most modifiable preventive targets. Screening protocols in similar resource settings should consider extending the birth weight threshold to include this weight category.

Keywords: Retinopathy of Prematurity, Low Birth Weight, 1501–1800 G, Oxygen Therapy, Screening, Neonatal Risk Factors, Hyperglycemia, Apgar score, Septicaemia, Anemia, Blood Transfusion.

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INTRODUCTION

Retinopathy of prematurity (ROP), previously designated as retrolental fibroplasia (RLF), is a vaso-proliferative disorder of the developing retina that arises from aberrant retinal vascularization in preterm neonates.

It remains among the foremost causes of preventable childhood blindness globally.[1] The pathogenesis is complex and multifactorial: the primary stage involves an initial insult—hyperoxia, hypoxia, or hemodynamic instability—at a critical window of retinal vascular development, resulting in vasoconstriction and vascular arrest mediated by downregulation of vascular

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endothelial growth factor (VEGF). The secondary stage is characterized by aberrant neovascularization driven by excess angiogenic factors, with new vessels extending into the vitreous and potentially causing tractional retinal detachment.[2]

Incidence rates vary considerably between settings: reported figures range from 21% to 65.8% in Western studies and from 34.9% to 60.1% in Indian studies.[3] Screening guidelines formulated by the American Academy of Pediatrics (AAP) and the American Association for Pediatric Ophthalmology and Strabismus (AAPOS), based on the CRYO-ROP and LIGHT-ROP trials, traditionally recommend screening of infants with birth weight ≤1500 g or gestational age ≤30 weeks.[4] However, the epidemiology of ROP differs substantially between developed and developing nations.[5]

In India and other middle-income countries, improved neonatal survival has expanded the population of premature infants reaching later stages of retinal development, and ROP in infants weighing above 1501 g is increasingly documented.[5,6] Several tertiary-care Indian cohorts have reported ROP in babies with birth weights up to 1800 g or even higher, yet most screening protocols do not mandate examination of this group unless additional clinical risk factors are present.[7] This creates a gap between the epidemiological reality and clinical practice.

Given the paucity of prospective data specifically characterizing the 1501–1800 g birth weight subgroup from eastern India, the present study was undertaken at Nil Ratan Sircar Medical College and Hospital, Kolkata, with the primary aim of determining the incidence of ROP in infants weighing less than 1800 g and the secondary aim of identifying associated neonatal and perinatal risk factors, with particular attention to the 1501–1800 g subgroup.

Aims and Objectives

Aim

To evaluate the incidence of ROP and identify the associated risk factors among preterm infants weighing less than 1800 g, with special reference to those weighing between 1501 and 1800 g.

Objectives

1. To determine the incidence of ROP in infants with birth weight <1800 g, including the 1501–1800 g subgroup.
2. To assess the association between various perinatal and neonatal risk factors and the development of ROP in both weight categories.

METHODOLOGY

Study Design

Prospective, observational, hospital-based study.

Study Setting

ROP clinic, Ophthalmology OPD, Nil Ratan Sircar Medical College and Hospital (NRSMCH), Kolkata — a tertiary care referral center serving urban and rural populations. Infants were referred from the NICU and postnatal wards.

Study Duration

March 2019 to February 2020 (12 months).

Sample Size

Two hundred (200) consecutive eligible infants enrolled during the study period.

Eligibility Criteria

Inclusion: preterm infants with birth weight <1800 g; gestational age <36 completed weeks (by maternal obstetric history or Expanded New Ballard Scoring System); both sexes; parents providing written informed consent.

Exclusion: birth weight ≥1800 g; gestational age ≥36 weeks; failure to complete scheduled follow-up; presence of congenital ocular anomalies or media opacities precluding fundoscopy; known congenital retinal disorders.

Operational Definitions

Hyperglycemia was defined as random blood glucose >125 mg/dL on daily monitoring during the first seven days of life. Hypoxia and hyperoxemia were evaluated by arterial blood gas analysis and continuous pulse oximetry; SpO_2 88–92% was taken as the reference range. Septicemia was defined by positive blood culture for bacterial or fungal organisms, or by laboratory criteria (WBC <5000/mm³, ANC <1000, band ratio >0.2). Anemia was defined as hemoglobin or hematocrit more than two standard deviations below the mean for postnatal age. Thrombocytopenia was defined as platelet count $<1.5 \times 10^5$/mm³. Severe respiratory illness was defined by requirement for mechanical ventilation with documentation of duration, FiO_2, peak inspiratory pressure, and PEEP.

ROP Screening Procedure

Screening was performed as per AAP 2013 guidelines based on postmenstrual age. Pupillary dilation was achieved with tropicamide 0.5% instilled every 10–15 minutes for four doses beginning one hour prior, followed by phenylephrine 2.5% (diluted 1:4 for neonatal use) once before examination. Repeated phenylephrine instillation was avoided to minimize systemic hypertension. After topical anaesthesia with proparacaine, a neonatal wire speculum was inserted and binocular indirect ophthalmoscopy was performed using a +20 diopter lens by an experienced ophthalmologist. The anterior segment was examined first, followed by the posterior pole for plus disease, then sequential peripheral retinal examination across all clock hours using a scleral depressor. Findings were classified per ICROP (zone, stage, extent in clock hours, presence of pre-plus or plus disease) and documented after each

examination. Follow-up intervals were determined by disease severity and ICROP recommendations.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using SPSS version 26.0. Continuous variables are expressed as mean ± SD and compared by Student's t-test. Categorical variables are expressed as proportions and compared using the chi-square test or Fisher's exact test, as appropriate. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors. A p-value <0.05 was considered statistically significant.

Ethical Considerations

Institutional Ethics Committee approval was obtained before study commencement. Written informed consent was obtained from parents or guardians after explanation in the vernacular language. Patient data confidentiality was strictly maintained.

RESULTS

Demographic and Baseline Characteristics

Two hundred preterm infants with birth weight <1800 g were enrolled. Table 1 summarizes baseline characteristics. Of the total, 105 infants (52.5%) weighed between 1501 and 1800 g and 95 (47.5%) weighed less than 1500 g. Males comprised 51.5% of the cohort. The majority of infants (71%) were born between 32 and 36 completed weeks of gestation; 29% were born before 32 weeks. Regarding mode of delivery, 77% were born by normal vaginal delivery, 11.5% by assisted vaginal delivery, and 11.5% by caesarean section (LSCS).

Table 1: Baseline Characteristics of the Study Population

Variable	Number	Percentage (%)
Total infants screened	200	100
Birth weight <1500 g	95	47.5
Birth weight 1501–1800 g	105	52.5
Male	103	51.5
Female	97	48.5
Gestational age <32 weeks	58	29.0
Gestational age 32–36 weeks	142	71.0
Vaginal delivery	154	77.0
Assisted vaginal delivery	23	11.5
LSCS	23	11.5

Incidence of ROP

ROP was detected in 56 of 200 infants (28.0%). In the 1501–1800 g subgroup, 26 of 105 infants (24.76%) developed ROP. In the <1500 g group, the incidence was 31.57%. Table 2 presents incidence by birth weight category.

Table 2: Incidence of ROP by Birth Weight Category

Birth Weight Group	ROP Cases	Total (n)	Incidence (%)
< 1800 g (overall)	56	200	28.00
1501–1800 g	26	105	24.76
< 1500 g	30	95	31.57

Staging of ROP

Among the 56 affected infants, Stage 1 was present in 26 cases (46.45%) and Stage 2 in 24 cases (42.85%), together accounting for 89.3% of all ROP. Stage 3 was found in 3 infants (5.35%), Stage 4 in 2 (3.50%), and Stage 5 in 1 (1.78%). No infant in any weight subgroup had plus disease or aggressive posterior ROP (AP-ROP). In the 1501–1800 g subgroup specifically, Stages 1 and 2 each accounted for 46.1% of cases; none had Stage 5, plus disease, or AP-ROP. Table 3 details staging by birth weight group.

Table 3: Staging of ROP by Birth Weight Group

Stage of ROP	BW <1500 g (n=30 with ROP)	BW 1501–1800 g (n=26 with ROP)	BW <1800 g (n=56 with ROP)
No ROP	65	79	144
Stage 1	14 (46.65%)	12 (46.1%)	26 (46.45%)
Stage 2	12 (40.0%)	12 (46.1%)	24 (42.85%)
Stage 3	2 (6.66%)	1 (3.84%)	3 (5.35%)

Stage 4	1 (3.33%)	1 (3.84%)	2 (3.50%)
Stage 5	1 (3.33%)	0	1 (1.78%)
Plus disease / AP-ROP	0	0	0
Any ROP	**30**	**26**	**56**

Oxygen-Related Variables

Oxygen therapy was required by 41 of 56 ROP-affected infants (73.2%) compared with 30 of 144 infants without ROP (20.8%), a highly significant difference ($p<0.0001$). In the 1501–1800 g subgroup, 82.6% of ROP cases had required oxygen versus 20.3% of non-ROP infants ($p<0.0001$). Infants with ROP had significantly higher mean maximum SpO_2, lower mean minimum SpO_2, and markedly longer mean duration of oxygen exposure. These findings held equally in the 1501–1800 g subgroup. Table 4 presents oxygen-related variables in detail.

Table 4: Oxygen-Related Variables — Comparison Between ROP and Non-ROP Groups

Variable	ROP Present (n=56)	No ROP (n=144)	p-value
Oxygen required — n (%)	41 (73.2%)	30 (20.8%)	<0.0001
Maximum SpO_2 — mean ± SD (%)	98.87 ± 1.13	97.23 ± 1.98	0.024
Minimum SpO_2 — mean ± SD (%)	81.95 ± 6.57	87.68 ± 3.33	0.002
Duration of oxygen — mean ± SD (hrs)	60.15 ± 57.57	11.48 ± 24.46	<0.001
Subgroup BW 1501-1800 g: oxygen required 19/26 (82.6%) vs 16/79 (20.3%), p<0.0001; max SpO2 97.97 +/- 1.02 vs 97.22 +/- 1.99, p=0.025; min SpO2 80.05 +/- 7.49 vs 87.56 +/- 3.27, p=0.002; duration 61.05 +/- 58.58 hrs vs 11.47 +/- 24.45 hrs, p<0.001.			

Perinatal and Metabolic Factors

Mean blood glucose levels during the first seven days of life were significantly higher in infants with ROP (179 ± 12 mg/dL vs 120 ± 10 mg/dL, p=0.003). The same association held in the 1501–1800 g subgroup (181 ± 11 mg/dL vs 120 ± 10 mg/dL, p=0.0035). No infant had a 1-minute APGAR score below 3; however, APGAR scores below 6 at 1 minute and below 7 at 5 minutes were significantly more frequent in ROP-affected infants in both weight groups ($p<0.0001$ and $p<0.001$ respectively). Table 5 summarizes these findings.

Table 5: Perinatal and Metabolic Factors

Variable	ROP Present	No ROP	p-value
Mean blood glucose — first 7 days (mg/dL)	179 ± 12	120 ± 10	0.003
APGAR <6 at 1 minute — n	29	30	<0.0001
APGAR <7 at 5 minutes — n	26	24	<0.001

Neonatal Morbidities

Several neonatal morbidities were significantly more frequent among infants who developed ROP (Table 6). Respiratory distress syndrome (RDS) was present in 67.9% of ROP-positive infants <1800 g and 69.2% of those in the 1501–1800 g subgroup ($p<0.001$). Apnea of prematurity was found in 39.3% of ROP-positive infants overall and 38.5% in the 1501–1800 g subgroup ($p<0.001$). Septicemia was present in 53.6% and 57.7% respectively ($p=0.003$ and $p=0.009$). Anemia occurred in 58.9% and 61.5% ($p=0.03$ and $p=0.04$). History of blood transfusion was present in 64.3% and 57.7% of ROP cases ($p=0.039$ and $p=0.046$).

Table 6: Neonatal Morbidities Significantly Associated with ROP

Neonatal Morbidity	ROP Present (n=56)	No ROP (n=144)	p-value
Respiratory distress syndrome	38 (67.9%)	23 (16.0%)	<0.001
Apnea of prematurity	22 (39.3%)	14 (9.7%)	<0.001
Septicemia	30 (53.6%)	20 (13.9%)	0.003
Anemia	33 (58.9%)	24 (16.7%)	0.030
Blood transfusion	36 (64.3%)	22 (15.3%)	0.039

Non-Significant Variables

Surfactant therapy, necrotizing enterocolitis, intraventricular hemorrhage, patent ductus arteriosus, pregnancy-induced hypertension, antepartum hemorrhage, meconium-stained amniotic fluid, and birth order did not demonstrate significant association with

ROP development in either the overall or subgroup analyses. Table 7 lists these variables with their p-values.

Table 7: Variables Not Significantly Associated with ROP

Variable	p-value (BW<1800 g)	p-value (1501–1800 g)
Surfactant therapy	0.313	0.406
Necrotizing enterocolitis	0.322	0.409
Intraventricular hemorrhage	0.671	0.616
Patent ductus arteriosus	0.939	0.991
Pregnancy-induced hypertension	0.485	0.483
Antepartum hemorrhage	0.478	0.472
Meconium-stained amniotic fluid	0.477	0.483
Birth order	NS	NS

DISCUSSION

The central clinical message of the present study is that ROP occurs in approximately one quarter of infants weighing between 1501 and 1800 g — a birth weight stratum explicitly excluded from mandatory screening under standard AAP and Indian guidelines. An incidence of 24.76% in this subgroup, comparable to the 28% overall incidence, supports the view that restricting ophthalmic surveillance to infants under 1500 g is insufficient in the Indian tertiary care context.

Contextualizing with Indian and international literature (Table 8), our overall incidence of 28% is higher than most Indian studies, which have reported rates of 17.5–22.3% in infants below 1250–1500 g.[7] However, our inclusion criteria extended to 1800 g and 36 weeks, reflecting the approach recommended by several Indian investigators who advocate widening the screening net in low- and middle-income settings where neonatal morbidity profiles differ from those in high-income countries.[5,6] The consistently higher incidence compared with international studies from Western settings (9.4–25.4%) likely reflects the different spectrum of illness severity and oxygen management practices.

Table 8: Incidence of ROP in the Present Study Compared with Selected Indian Studies

Study	Gestational Age (wks)	Birth Weight (g)	Incidence (%)
Maheshwari 1996	≤35	≤1500	20.0
Patil 1997	≤32	≤1250	17.5
Dutta 2004	≤32	≤1750	21.0
Gupta 2004	≤32	≤1250	21.7
Chaudhari 2009	≤32	≤1500	22.3
Present study (overall <1800 g)	**≤36**	**<1800**	**28.0**
Present study (1501–1800 g subgroup)	**≤36**	**1501–1800**	**24.76**

Regarding disease severity, the preponderance of Stage 1 and Stage 2 disease and the complete absence of plus disease or AP-ROP in our cohort is reassuring. This pattern is consistent with systematic screening programs that detect disease at early stages before it becomes vision-threatening.[4] The absence of plus disease may reflect relatively close follow-up in a dedicated ROP clinic, though single-center selection bias cannot be excluded. Severity was modestly higher in the <1500 g group (13.3%) compared with 1501–1800 g (7.7%), consistent with the expected inverse relationship between birth weight and disease severity.

Oxygen therapy emerged as the single strongest modifiable risk factor. The convergence of three independent oxygen-related parameters — elevated maximum SpO_2, reduced minimum SpO_2, and prolonged duration underscores that it is not merely the peak oxygen level but also the volatility and cumulative exposure that are pathogenetically relevant. The classical oxygen theory of Ashton posited that elevated arterial oxygen saturation causes retinal vasoconstriction and, if sustained, permanent vascular occlusion.[8] The spindle cell theory further attributes free radical injury from fluctuating oxygen levels as damaging precursor cells of retinal vasculature.[8] Both mechanisms are operative in the clinical scenario described here. The high proportion of oxygen-requiring infants in the 1501–1800 g ROP group (82.6%) is particularly noteworthy, emphasizing that oxygen exposure in this weight range is as consequential as in lighter infants.

Hyperglycemia as a significant finding corroborates several previous reports, which have demonstrated a dose-response relationship between mean glucose in the first month of life and ROP severity.[9] The mechanism likely involves oxidative stress: hyperglycemia generates reactive oxygen species through glucose auto-

oxidation and activation of the polyol pathway, which may prime retinal endothelial cells for subsequent VEGF dysregulation. The significantly elevated mean glucose in the 1501–1800 g ROP subgroup (181 mg/dL vs 120 mg/dL) is a clinically actionable finding, as glycemic control in NICUs is amenable to protocol-driven intervention.

Low APGAR scores at both 1 and 5 minutes were significantly associated with ROP. Perinatal asphyxia may trigger hypoxic-ischemic stress that disrupts the orderly development of retinal vasculature from the outset. Shah et al. previously reported the association between 1-minute APGAR and ROP; our study extends this to the 5-minute score in the 1501–1800 g subgroup. Infants requiring more extensive resuscitation at birth may be at heightened risk independent of gestational age alone.

Respiratory morbidities — RDS and apnea of prematurity — were among the most prevalent risk factors. RDS necessitates supplemental oxygen, establishing a direct link to the oxygen pathway. Recurrent apnea produces episodic hypoxia-hyperoxia cycles, generating oxidative bursts that are injurious to the immature retina. In the present study, 39.3% of ROP-positive infants had apnea, consistent with figures reported by Shohat et al. and Gunn et al., though somewhat lower than the 54% reported by Agarwal et al. and Gupta et al. Aggressive management of apnea with caffeine therapy may therefore have secondary benefits in reducing ROP burden.

The significant associations of septicemia, anemia, and blood transfusion reinforce a systemic morbidity model of ROP. Septicemia activates inflammatory cascades, including cytokine release and neutrophil oxidative bursts, which cause endothelial injury in retinal capillaries. Anemia and blood transfusion are mechanistically linked through the fetal-to-adult hemoglobin switch: adult hemoglobin releases oxygen more readily at the tissue level, creating localized hyperoxia in retinal tissue that may trigger vasoconstriction and subsequent neovascularization.[10] These findings were equally significant in the 1501–1800 g subgroup, reinforcing the argument that the same pathophysiological cascade operates across this broader birth weight range.

The non-significant associations for surfactant therapy, NEC, IVH, PDA, and maternal factors (PIH, APH) are consistent with several Indian and international studies, where these variables often lose significance after controlling for birth weight and oxygen exposure. Surfactant therapy, despite reducing the severity of RDS and its associated oxygen requirement, did not independently alter ROP incidence in the present cohort — possibly because a very small proportion of infants with RDS actually received surfactant, limiting statistical power for this subanalysis.

From a screening policy perspective, the present data join a growing body of evidence suggesting that Indian guidelines should incorporate an evidence-based threshold for the 1501–1800 g weight range. The WHO VISION 2020 programme places ROP screening as a priority for secondary prevention of childhood blindness. Current practice in many Indian NICUs mandates screening of this weight group only in the presence of additional clinical risk factors. Given that nearly one in four infants in this weight range developed ROP in our study, a universal screening policy for infants up to 1800 g in high-risk units appears justified. Newer tools such as the ROPScore — incorporating birth weight, gestational age, weight gain, blood transfusion, and oxygen exposure — may further refine risk stratification and optimize screening workload.[11] Telemedicine-based screening using digital retinal imaging, with remote grading by trained ophthalmologists, is a promising approach to extend coverage to underserved NICUs in eastern India.[12]

Limitations

The study was conducted at a single tertiary care center, limiting generalizability to other settings with different patient profiles and clinical practices. The sample size of 200, while adequate for demonstrating significant associations, constrains the robustness of subgroup analyses. Multivariate modeling to identify fully independent risk factors was performed, but residual confounding between closely correlated variables such as gestational age, illness severity, and oxygen exposure cannot be excluded. Long-term visual and neurodevelopmental outcomes were not evaluated, leaving the downstream consequences of early-stage ROP in the 1501–1800 g group uncharacterized. A larger multicentric study with extended follow-up is needed to validate these findings.

CONCLUSION

The present study demonstrates a clinically meaningful incidence of retinopathy of prematurity (24.76%) among infants weighing between 1501 and 1800 g — a group not currently included in mandatory screening programs under standard guidelines. This finding supports extending ROP screening to infants in this birth weight range in tertiary care NICUs in India. Oxygen therapy, glycemic instability, perinatal compromise, respiratory morbidities, septicemia, anemia, and blood transfusion were all significantly associated with ROP in both the overall cohort and the 1501–1800 g subgroup. Targeted interventions addressing oxygen monitoring, blood glucose control, infection prevention, and anemia management remain the most actionable preventive

strategies. Region-specific, evidence-based screening policies should be developed and disseminated to prevent avoidable blindness in this expanding population of preterm survivors.

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