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ORIGINAL RESEARCH

# Delayed Screening and Advanced-Stage Retinopathy of Prematurity at an Indonesian Tertiary Referral Hospital: A Cross-Sectional Study

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**ABSTRACT**

**Background:** Retinopathy of prematurity (ROP) is a leading preventable cause of childhood blindness, with its burden shifting toward middle-income countries where neonatal survival outpaces screening capacity. Indonesian data on programme performance are scarce.

**Objective:** To describe the screened population, quantify screening timeliness, and compare the observed ROP burden with Indonesian gestational-age-specific reference rates.

**Methods:** A cross-sectional study at M. Djamil General Hospital, Padang, ran from January to December 2025. Total sampling enrolled 278 preterm infants undergoing initial screening; 277 had complete funduscopic data, classified by ICROP3. Proportions carry Wilson 95% confidence intervals (CI). Indirect standardisation against Indonesian gestational-age-stratum rates gave a standardised morbidity ratio (SMR); as the records hold only marginal distributions, multivariable parameters were bounded by partial identification.

**Result:** Infants were mostly male (161/277; 58.1%, 95% CI 52.2–63.8) and born at 28–32 weeks (53.1%); 98.9% weighed 1500–2500 g, 75.5% had oxygen beyond 7 days. ROP was detected in 31/277 infants (11.2%, 95% CI 8.0–15.4). No infant had stage 1 disease, whereas 22/31 (71.0%) had stage 3 or worse and 12/31 (38.7%) stage 4–5. Observed cases exceeded the 19.2 expected (SMR 1.61, 95% CI 1.10–2.29; p = 0.016), but excluding stage 4–5 returned the ratio to unity (0.99, 0.60–1.54). Two-factor standardisation held it at 1.48–1.85 across every admissible oxygen distribution. First examination occurred beyond 8 weeks chronological age in 42.4%.

**Conclusion:** ROP prevalence was well below pooled global estimates, yet the cohort carried an excess of advanced disease that adjustment for oxygen and birth weight does not explain, and none of the early disease a timely programme detects. This indicates delayed detection, not low risk; timeliness is the priority.

## 1. Introduction

Retinopathy of prematurity (ROP) is a vasoproliferative disorder of the developing retina and one of the few causes of childhood blindness that is almost entirely preventable when detection is timely.[1,2] A spatiotemporal analysis of the Global Burden of Disease data quantified the resulting childhood and adolescent visual loss and showed that it remains concentrated in countries whose neonatal survival is improving fastest.[3] A systematic review and meta-analysis spanning four decades reported a pooled ROP prevalence of approximately 31.9% among screened preterm infants, with heterogeneity driven by screening criteria, neonatal-unit quality and population maturity.[4] National surveillance data show the same heterogeneity over time: the proportion of preterm infants examined and treated in the United States shifted substantially between 2003 and 2019[5] and again across a decade of screening practice,[6] while single-centre series in extremely low birth weight infants continue to report incidences well above the pooled figure.[7] The epidemiological centre of gravity has moved decisively: as neonatal intensive care expands across middle-income countries, survival of very preterm infants improves faster than the ophthalmic services required to protect their vision, producing what has been described as a third epidemic of ROP.[5,8,9]

Indonesia sits squarely within this transition. A multicentre national survey of preterm infants reported ROP incidence of 18% below 28 weeks of gestation, 7% at 28–32 weeks and 3.8% above 32 weeks.[10] These figures are considerably lower than pooled international estimates, but they were derived from participating centres with established screening pathways and may not represent programme performance more broadly. Indonesian national guidance admits a wider population to screening than North American criteria, encompassing infants of gestational age 34 weeks or less or birth weight 1500 g or less, together with infants weighing 1500–2000 g who carry additional risk factors.[11] This deliberately broad threshold reflects the local reality that severe ROP is not confined to the smallest infants, but it also imposes a substantial and growing workload on a limited number of paediatric ophthalmologists.

The pathophysiology of ROP is conventionally described in two phases.[12,13] In the first, exposure of an immature retina to a relatively hyperoxic extrauterine environment suppresses hypoxia-inducible factor 1-alpha and, with it, vascular endothelial growth factor (VEGF) and insulin-like growth factor 1, arresting vascular growth and leaving a peripheral avascular retina. In the second, as the infant grows and the avascular retina becomes metabolically demanding, tissue hypoxia drives a VEGF surge and disordered neovascularisation at the vascular-avascular junction, which may progress to fibrovascular proliferation and tractional retinal detachment. Prolonged supplemental oxygen is the principal modifiable exposure, and its effect is dose-dependent: each additional day of oxygen supplementation incrementally raises the odds of ROP independent of gestational age,[14] with the first seven weeks of life constituting the period during which exposure most strongly predicts severe disease.[15] Saturation levels achieved in the neonatal unit,[16] the specific oxygen-therapy parameters recorded in early life[2] and the saturation targets adopted as policy[17] have each been related to ROP stage. The extent of peripheral avascular retina at first examination is itself

a determinant of subsequent course.[13] Antenatal factors contribute risk before any postnatal oxygen is delivered, which helps explain why ROP is not exclusive to the most immature infants: prenatal maternal characteristics[18] and placental histopathology[19] are both associated with subsequent ROP.

Because the therapeutic window is narrow, screening performance is inseparable from clinical outcome. International guidance recommends that infants meeting eligibility criteria undergo a first dilated examination at a post-menstrual age of 31 weeks or at 4 weeks chronological age, whichever is later, with re-examination every one to two weeks until vascularisation is complete.[1] Laser photocoagulation and intravitreal anti-VEGF agents are highly effective for type 1 disease,[20,21] but neither restores vision once tractional detachment has occurred; surgical outcomes in stage 4 and 5 disease remain poor.[22] A screening programme is therefore judged not only by how many cases it finds but by the stage at which it finds them. Screening delay has been shown to shift the detected disease spectrum toward treatment-requiring stages,[23] and both attendance at scheduled follow-up[24] and the social circumstances that determine it[25] remain persistent challenges. Where surveillance fails, retinal detachment continues to occur even in well-resourced national systems.[26]

M. Djamil General Hospital in Padang is the principal tertiary referral centre for West Sumatra and receives preterm infants from across the province. Its paediatric ophthalmology and strabismus subdivision conducts the region's ROP screening service. Despite the volume of infants passing through this pathway, the clinical profile of infants presenting for screening, and the operational performance of the screening programme itself, have not previously been described. Published Indonesian work has concentrated on disease incidence rather than on the timeliness of detection, leaving an important gap: a low reported prevalence may reflect either a genuinely low-risk population or a programme examining infants too late to observe early disease.

This study was undertaken to address that gap. Its aims were threefold: to describe the demographic, perinatal and funduscopic profile of preterm infants undergoing initial ROP screening at this centre; to quantify the timeliness of first examination against international and national guideline windows; and to determine whether the observed ROP burden was consistent with, or in excess of, that expected from published Indonesian gestational-age-stratum-specific rates. Reporting prevalence, severity and timeliness together, and standardising the observed burden against a national reference, allows these two explanations to be separated.

## 2. Methods

### *Study design and reporting*

This was an observational, descriptive cross-sectional study of preterm infants undergoing initial ROP screening. Reporting follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies, and the completed checklist is available from the corresponding author. The study addressed three pre-specified objectives: characterisation of the screened population, quantification of screening timeliness against guideline windows, and comparison of the observed ROP burden with a national reference expectation.

### *Setting and participants*

The study was conducted at the paediatric ophthalmology and strabismus outpatient clinic of M. Djamil General Hospital, Padang, Indonesia, the tertiary referral centre for West Sumatra Province, over a twelve-month period from January to December 2025. The centre receives preterm infants referred from its own neonatal intensive care unit and from secondary hospitals and birthing facilities across the province.

Total sampling was applied: every preterm infant presenting for an initial ROP screening examination during the study period and meeting the eligibility criteria was enrolled, with no sampling fraction and no consecutive-recruitment ceiling. Eligibility followed Indonesian national guidance, which admits infants of gestational age 34 weeks or less or birth weight 1500 g or less, together with infants of birth weight 1500–2000 g who carry additional risk factors such as prolonged oxygen therapy, sepsis or haemodynamic instability.[11] Infants were excluded if the medical record was incomplete for outcome ascertainment or if ROP treatment had been received before the index examination, since prior treatment precludes classification of the natural disease stage. Two hundred and seventy-eight infants underwent initial screening and 277 had complete funduscopic outcome data, forming the analytic sample (Figure 1).

### *Variables and data sources*

Data were extracted retrospectively from clinical records using a standardised extraction form. Recorded variables were sex; chronological age at first screening examination in completed weeks; gestational age at birth in completed weeks; birth weight in grams; duration of supplemental oxygen therapy, dichotomised at 7 days in accordance with the exposure window identified as most predictive of severe ROP;[15] post-menstrual age at screening, calculated as gestational age at birth plus chronological age; funduscopic findings; and management disposition.

It should be noted explicitly that the oxygen variable records the total duration of supplemental oxygen exposure as documented in the record. It does not capture the fraction of inspired oxygen, target saturation ranges, the mode of respiratory support, or the number of hyperoxic or hypoxic excursions, none of which were recorded consistently. The variable is therefore a crude marker of exposure duration and not a measure of oxygen dose or of the quality of oxygen governance.

Record completeness varied slightly between variables. The recovered denominators indicate that missingness affects at most one record out of 278, consistent with the single infant excluded for incomplete outcome ascertainment. No imputation was performed or required, and a complete-case analysis is unbiased under any plausible mechanism at this scale.

### *Funduscopic examination and outcome definition*

Funduscopic examination was performed by an experienced paediatric ophthalmologist using a binocular indirect ophthalmoscope with a 28 D condensing lens following pharmacological pupillary dilation. Retinal findings were classified according to the International Classification of Retinopathy of Prematurity, third edition (ICROP3).[27] Each infant was assigned to one of three mutually exclusive categories on the basis of the worse eye: immature retina, defined as incomplete peripheral vascularisation without ROP and further characterised by the most posterior zone reached (zone I, II or III); mature retina, defined as complete peripheral vascularisation; or ROP, staged from 1 to 5. The three categories are exhaustive, so their counts sum to the analytic sample. Two secondary outcome definitions were applied. Sight-threatening ROP was defined as stage 3 or worse, and advanced ROP as stage 4 or 5, that is, partial or total retinal detachment. Management was categorised as observation with scheduled re-examination, or referral for treatment.

### *Screening timeliness*

Timeliness was assessed against two guideline anchors. International guidance recommends a first examination at a post-menstrual age of 31 weeks or at 4 weeks chronological age, whichever is later, with the recommended chronological window spanning 4 to 6 weeks.[1] Two timeliness indicators were therefore pre-specified: the proportion of infants first examined beyond 8 weeks chronological age, a threshold chosen to allow generous latitude beyond the recommended window; and the proportion first examined beyond a post-menstrual age of 40 weeks, by which point term-equivalent age has been reached and the

second, proliferative phase of the disease is typically well established.

### Sample size and precision

No a priori sample size was calculated, because total sampling over a fixed twelve-month period determined the achieved sample. Precision was instead assessed post hoc. With 277 infants and an observed ROP prevalence of 11.2%, the half-width of the 95% confidence interval is 3.71 percentage points, and 153 infants would have sufficed for a half-width of 5 percentage points. The achieved sample therefore estimates the primary outcome with adequate precision for a descriptive programme evaluation. It should be noted that stratified risk-factor analysis is precluded here not by sample size but by identification: with only marginal distributions recorded, no exposure-outcome association is estimable at any sample size, so power is not the operative constraint.

### Statistical analysis

Analyses were performed in Python 3.11 using SciPy 1.15. Categorical variables are reported as counts with percentages, and every proportion is accompanied by a Wilson score 95% confidence interval, which maintains nominal coverage for proportions near the boundaries and for the small denominators arising within ROP stage strata. Because record completeness varied by variable, each table reports the valid denominator for that variable rather than assuming a single denominator throughout; denominators are stated in every table heading and figure caption.

The observed number of ROP cases was compared with the number expected under published Indonesian gestational-age-stratum-specific rates by indirect standardisation.[10] Indirect rather than direct standardisation was used because direct standardisation would require stratum-specific outcome counts from this cohort, which were not recorded, whereas indirect standardisation requires only the cohort's stratum sizes and an external rate set. Expected counts were computed as the sum across strata of the stratum size multiplied by its published national rate, and the standardised morbidity ratio (SMR) was calculated as observed divided by expected. The 95% confidence interval was derived using Byar's approximation to the Poisson distribution; this was checked against the exact interval obtained from the chi-square relationship and the two agreed to two decimal places. The two-sided p value was obtained by doubling the one-sided Poisson tail probability, which is the more conservative of the available constructions.

Three pre-specified benchmark comparisons were made using exact binomial tests: the observed ROP prevalence against the pooled global estimate of 31.9%;[4] the referral proportion against the midpoint of the 5–10% range reported internationally;[24] and the proportion of ROP cases at stage 4–5 against a 10% benchmark. Effect sizes for differences between two proportions are reported as Cohen's h, for which values of 0.20, 0.50 and 0.80 conventionally denote small, medium and large effects; because effect sizes computed on the 31-case ROP subgroup carry substantial uncertainty, h is reported with the interval obtained by evaluating it at the Wilson limits of the underlying proportion. No test of the cohort's gestational-age composition against a national reference distribution is reported, because the published national survey gives stratum-specific rates rather than the composition of its screened population; the composition difference is described narratively instead, and the standardisation performs the corresponding adjustment.

Five sensitivity analyses of the SMR were pre-specified to test its dependence on the assumptions it rests upon: inflation of the national reference rates by 25% and by 50%, to represent under-ascertainment in the reference survey; partial and complete reclassification of the sub-28-week stratum into the 28–32-week stratum, to represent gestational-age misclassification; substitution of the un-reconciled source count; and exclusion of stage 4–5 cases, to test whether any excess is concentrated in advanced disease.

The number needed to screen to detect one case was computed as the reciprocal of the corresponding proportion, with confidence limits obtained by inverting the Wilson limits of that proportion. Five inferential procedures and more than forty confidence intervals are reported. All comparisons were pre-specified and address distinct questions, and no adjustment for multiplicity was applied; the confidence intervals should therefore be interpreted marginally rather than simultaneously. All tests were two-sided with alpha set at 0.05, and exact p values are reported to three decimal places except where p was below 0.001.

$$CI_{1-\alpha} = [\, \hat{p} + z^2/2n \pm z\sqrt{(\, \hat{p}(1-\hat{p})/n + z^2/4n^2\,)}\,] \,/\, (\,1 + z^2/n\,) \quad (1)$$

*Wilson score interval used for every proportion*

$$E = \Sigma_{g=1}{}^{3}\, n_g\, r_g{}^{\mathrm{nat}} \qquad \mathrm{SMR} = O \,/\, E. \quad (2)$$

*Expected count and standardised morbidity ratio*

$$L = O[\,1 - 1/9O - z/(3\sqrt{O})\,]^3 \,/\, E \qquad U = (O{+}1)[\,1 - 1/9(O{+}1) + z/(3\sqrt{(O{+}1)})\,]^3 \,/\, E. \quad (3)$$

*Byar's approximation to the Poisson limits of the SMR*

$$h = 2\arcsin\sqrt{p_1} - 2\arcsin\sqrt{p_2}. \quad (4)$$

*Cohen's effect size for the difference between two proportions*

### Multivariable analysis by partial identification

Let the unobserved joint distribution of gestational-age stratum, oxygen exposure and ROP status be represented by the twelve cell counts of a 3 × 2 × 2 contingency table. The observed margins impose seven linear equality constraints on those twelve non-negative unknowns, so the set of joint tables consistent with the data is a bounded convex polytope of dimension six. Every multivariable parameter is a function on that polytope, and its sharp identification region is the image of the polytope under that function. For parameters linear in the cell counts — stratum-specific case counts and risks, exposure-specific risks and risk differences, all of which have known denominators — the region's endpoints were obtained exactly by linear programming, and are sharp by linear-programming duality. For the Mantel-Haenszel adjusted odds ratio, which is a ratio of quadratic forms, the region was characterised analytically and corroborated by a verified inner approximation: several thousand feasible tables were generated from random vertex solutions and their convex combinations, every one re-checked against all margins to a tolerance of 1e-7 before its parameter value was retained, so every reported attainable value is genuinely attained.

Bounds derived from margins alone are wide by construction, so two monotonicity assumptions drawn from established biology were imposed in a stated hierarchy, allowing the reader to see exactly what each assumption buys. The first is that ROP risk does not increase with gestational-age stratum; the second is that ROP risk is not lower among infants exposed to more than seven days of supplemental oxygen. Both are ordering assumptions rather than parametric ones, both are supported by the evidence cited in the Introduction, and both are expressed as linear inequalities in the cell counts because the relevant denominators are observed.

Three further multivariable analyses were pre-specified. First, a one-parameter proportional-excess model, which

assumes that the stratum-specific risks in this cohort equal the published national rates multiplied by a common factor; the factor is identified by the observed total, and the resulting stratum risks were checked to lie inside the assumption-free identification region. Second, a two-factor indirect standardisation in which the expected count carries both the national gestational-age rates and an external rate ratio for prolonged oxygen; because the gestational-age by oxygen table is itself unobserved, the expected count was bounded by linear programming over its transportation polytope for each assumed rate ratio, and the rate ratio was varied across the plausible range from 1.0 to 3.0. Third, an exact bound on the extent to which birth weight could confound any reported estimate, obtained from the mass available for reallocation between birth-weight strata.

$$\mathcal{P} = \{\, n \in \mathbb{R}^{12}_{\geq 0} : \Sigma_{x,y}\, n_{gxy} = N_g,\ \Sigma_{g,y}\, n_{gxy} = M_x,\ \Sigma_{g,x}\, n_{gxy} = K_y \,\} \quad (5)$$

*Set of joint tables consistent with the observed margins*

$$\Theta(\theta) = [\, \min_{n^* \in \mathcal{P}} \theta(n)\ ,\ \max_{n^* \in \mathcal{P}} \theta(n) \,] \quad (6)$$

*Sharp identification region of a multivariable parameter $\theta$*

### *Ethical considerations*

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the institutional ethics committee of M. Djamil General Hospital, Padang. The study comprised a retrospective review of existing medical records; patient identifiers were removed before analysis, and no additional patient contact, examination or intervention was undertaken for the purposes of this research.

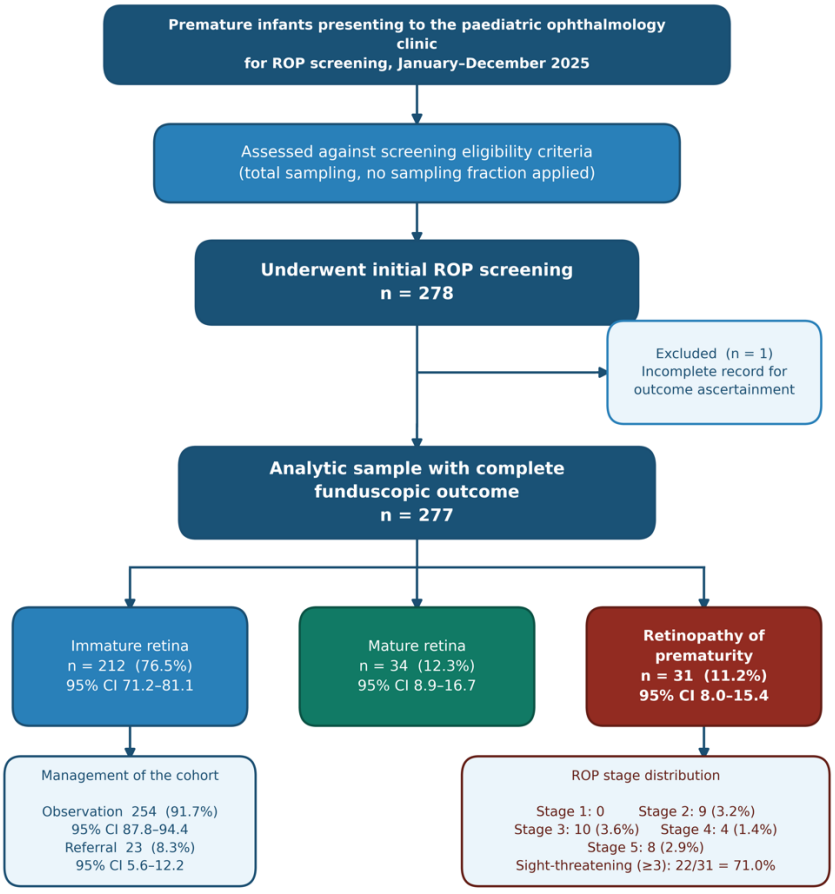


***Figure 1.*** *Flow of preterm infants through retinopathy of prematurity screening at M. Djamil General Hospital, Padang, January to December 2025. Proportions are of the analytic sample (n = 277) with Wilson score 95% confidence intervals.*

## 3. Results

### *Study population*

Two hundred and seventy-eight preterm infants underwent initial ROP screening at the paediatric ophthalmology clinic of M. Djamil General Hospital, Padang, between January and December 2025. One infant was excluded because the record was incomplete for outcome ascertainment, leaving 277 infants with complete funduscopic data in the analytic sample. The flow of infants through screening and their distribution across outcome categories are shown in Figure 1.

### *Demographic and perinatal characteristics*

Baseline characteristics are presented in Table 1. Male infants predominated, accounting for 161 of 277 infants (58.1%, 95% CI 52.2–63.8) against 116 female infants (41.9%, 95% CI 36.2–47.8). The single largest group was first examined between 4 and 8 weeks of chronological age (101/278; 36.3%), followed by infants examined before 4 weeks (59/278; 21.2%) and beyond 20 weeks (54/278; 19.4%).

Gestational age was 28–32 weeks in 147 of 277 infants (53.1%, 95% CI 47.2–58.9), 32 to under 37 weeks in 102 infants (36.8%, 95% CI 31.4–42.6) and below 28 weeks in 28 infants (10.1%, 95% CI 7.1–14.2). Birth weight was concentrated almost entirely in a single stratum: 275 of 278 infants (98.9%, 95% CI

96.9–99.6) weighed between 1500 and 2500 g, while 3 infants (1.1%) weighed 1000 to under 1500 g, and no infant weighed below 1000 g or 2500 g or more. Supplemental oxygen therapy exceeding 7 days was documented in 209 of 277 infants (75.5%, 95% CI 70.1–80.1), with the remaining 68 infants (24.5%, 95% CI 19.9–29.9) exposed for 7 days or less.

Compared with the populations from which pooled international estimates are drawn, this cohort contained a smaller proportion of extremely preterm infants and a larger proportion of moderately preterm infants. No formal test of this composition difference is reported; the indirect standardisation described below performs the corresponding adjustment directly.

**Table 1. Demographic and perinatal characteristics of preterm infants undergoing initial retinopathy of prematurity screening.**

| Characteristic | n (%) | 95% CI |
|---|---|---|
| **Sex (n = 277)** | | |
| Male | 161 (58.1) | 52.2–63.8 |
| Female | 116 (41.9) | 36.2–47.8 |
| **Chronological age at first examination (n = 278)** | | |
| <4 weeks | 59 (21.2) | 16.8–26.4 |
| 4–8 weeks | 101 (36.3) | 30.9–42.1 |
| 9–12 weeks | 39 (14.0) | 10.4–18.6 |
| 13–16 weeks | 13 (4.7) | 2.8–7.8 |
| 17–20 weeks | 12 (4.3) | 2.5–7.4 |
| >20 weeks | 54 (19.4) | 15.2–24.5 |
| **Gestational age (n = 277)** | | |
| <28 weeks | 28 (10.1) | 7.1–14.2 |
| 28–32 weeks | 147 (53.1) | 47.2–58.9 |
| 32–<37 weeks | 102 (36.8) | 31.4–42.6 |
| **Birth weight (n = 278)** | | |
| <1000 g | 0 (0.0) | 0.0–1.4 |
| 1000–<1500 g | 3 (1.1) | 0.4–3.1 |
| 1500–2500 g | 275 (98.9) | 96.9–99.6 |
| ≥2500 g | 0 (0.0) | 0.0–1.4 |
| **Supplemental oxygen therapy (n = 277)** | | |
| ≤7 days | 68 (24.5) | 19.9–29.9 |
| >7 days | 209 (75.5) | 70.1–80.1 |

## *Funduscopic findings*

Funduscopic findings are presented in Table 2 and Figure 2. Immature retina was the most frequent finding, present in 212 of 277 infants (76.5%, 95% CI 71.2–81.1). Within this group, vascularisation had reached zone II in 140 infants (50.5%, 95% CI 44.7–56.4) and zone III in 72 infants (26.0%, 95% CI 21.2–31.5); no infant had zone I immature vascularisation. Fully mature retina was documented in 34 infants (12.3%, 95% CI 8.9–16.7).

Retinopathy of prematurity was detected in 31 of 277 infants, giving a screening-detected prevalence of 11.2% (95% CI 8.0–15.4). The stage distribution was strikingly asymmetric. No infant was recorded with stage 1 disease. Stage 2 was present in 9 infants (3.2%, 95% CI 1.7–6.1), stage 3 in 10 infants (3.6%, 95% CI 2.0–6.5), stage 4 in 4 infants (1.4%, 95% CI 0.6–3.7) and stage 5 in 8 infants (2.9%, 95% CI 1.5–5.6).

Expressed as a proportion of the infants with ROP, 22 of 31 (71.0%, 95% CI 53.4–83.9) had sight-threatening disease of stage 3 or worse, and 12 of 31 (38.7%, 95% CI 23.7–56.2) had advanced disease with partial or total retinal detachment. Advanced disease affected 12 of the whole analytic sample of 277 infants (4.3%, 95% CI 2.5–7.4).

Three cells in the funduscopic data are structurally zero: zone I immature vascularisation, stage 1 ROP, and, in the perinatal data, birth weight below 1000 g. A zero count among 277 infants carries an upper Wilson 95% bound of 1.4%, so none of these constitutes strong evidence of true absence. Their likely explanations differ: the absence of infants below 1000 g plausibly reflects survival in the referring units, whereas the absence of zone I vascularisation and of stage 1 disease would both be expected if first examination occurred after the retinal periphery had vascularised beyond zone I and after early lesions had progressed or regressed.

**Table 2. Funduscopic findings at first screening examination and severity summary (n = 277).**

| Funduscopic finding | n (%) | 95% CI |
|---|---|---|
| Immature retina | 212 (76.5) | 71.2–81.1 |
| Zone I | 0 (0.0) | 0.0–1.4 |
| Zone II | 140 (50.5) | 44.7–56.4 |
| Zone III | 72 (26.0) | 21.2–31.5 |
| Mature retina | 34 (12.3) | 8.9–16.7 |
| Retinopathy of prematurity | 31 (11.2) | 8.0–15.4 |
| Stage 1 | 0 (0.0) | 0.0–1.4 |
| Stage 2 | 9 (3.2) | 1.7–6.1 |
| Stage 3 | 10 (3.6) | 2.0–6.5 |

| Funduscopic finding | n (%) | 95% CI |
|---|---|---|
| Stage 4 | 4 (1.4) | 0.6–3.7 |
| Stage 5 | 8 (2.9) | 1.5–5.6 |
| **Severity summary** | | |
| Sight-threatening ROP (stage ≥3), of cohort | 22 (7.9) | 5.3–11.7 |
| Sight-threatening ROP (stage ≥3), of ROP cases | 22 (71.0) | 53.4–83.9 |
| Advanced ROP (stage 4–5), of cohort | 12 (4.3) | 2.5–7.4 |
| Advanced ROP (stage 4–5), of ROP cases | 12 (38.7) | 23.7–56.2 |

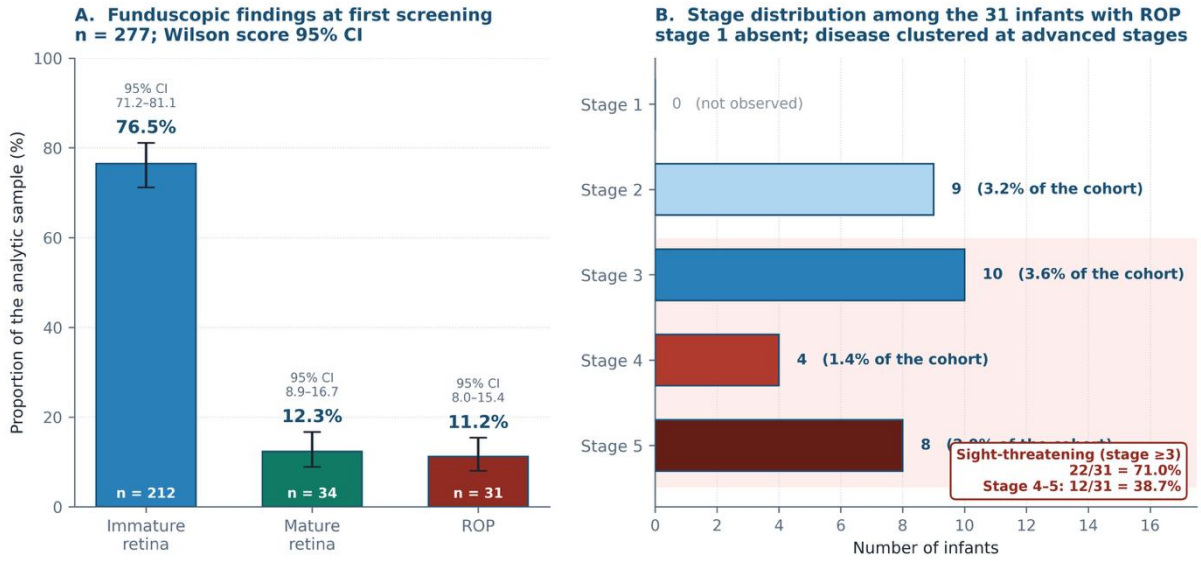


***Figure 2.*** *Funduscopic outcomes at first screening. (A) Distribution of the three mutually exclusive funduscopic categories with Wilson score 95% confidence intervals (n = 277). (B) Stage distribution among the 31 infants with retinopathy of prematurity; stage 1 was not observed, and 71.0% of cases were already stage 3 or worse.*

### *Timing of screening examination*

Post-menstrual age at screening and chronological age at first examination are summarised in Table 3 and displayed in Figure 3. Screening was most frequently performed at a post-menstrual age of 31–35 weeks, comprising 147 of 277 infants (53.1%, 95% CI 47.2–58.9), followed by 36–40 weeks in 67 infants (24.2%, 95% CI 19.5–29.6) and 41–49 weeks in 28 infants (10.1%, 95% CI 7.1–14.2). Twenty-two infants (7.9%, 95% CI 5.3–11.7) were screened before a post-menstrual age of 31 weeks, and 13 infants (4.7%, 95% CI 2.8–7.9) were screened beyond 50 weeks.

Against the two pre-specified timeliness thresholds, 118 of 278 infants (42.4%, 95% CI 36.8–48.3) received their first examination beyond 8 weeks of chronological age, and 41 of 277 infants (14.8%, 95% CI 11.1–19.5) were first examined beyond a post-menstrual age of 40 weeks. Conversely, 160 of 278 infants (57.6%, 95% CI 51.7–63.2) were first examined within 8 weeks of birth.

**Table 3. Timing of the first screening examination, with pre-specified timeliness indicators assessed against international guideline windows (first examination at post-menstrual age 31 weeks or 4 weeks chronological age, whichever is later). CI, confidence interval; PMA, post-menstrual age.**

| Timing variable | n (%) | 95% CI |
|---|---|---|
| **Post-menstrual age at screening (n = 277)** | | |
| <31 weeks | 22 (7.9) | 5.3–11.7 |
| 31–35 weeks | 147 (53.1) | 47.2–58.9 |
| 36–40 weeks | 67 (24.2) | 19.5–29.6 |
| 41–49 weeks | 28 (10.1) | 7.1–14.2 |
| >50 weeks | 13 (4.7) | 2.8–7.9 |
| **Timeliness indicators** | | |
| First examination within 8 weeks (n = 278) | 160 (57.6) | 51.7–63.2 |
| First examination beyond 8 weeks (n = 278) | 118 (42.4) | 36.8–48.3 |
| Screened at PMA 31–35 weeks (n = 277) | 147 (53.1) | 47.2–58.9 |
| Screened beyond PMA 40 weeks (n = 277) | 41 (14.8) | 11.1–19.5 |
| Screened beyond PMA 50 weeks (n = 277) | 13 (4.7) | 2.8–7.9 |

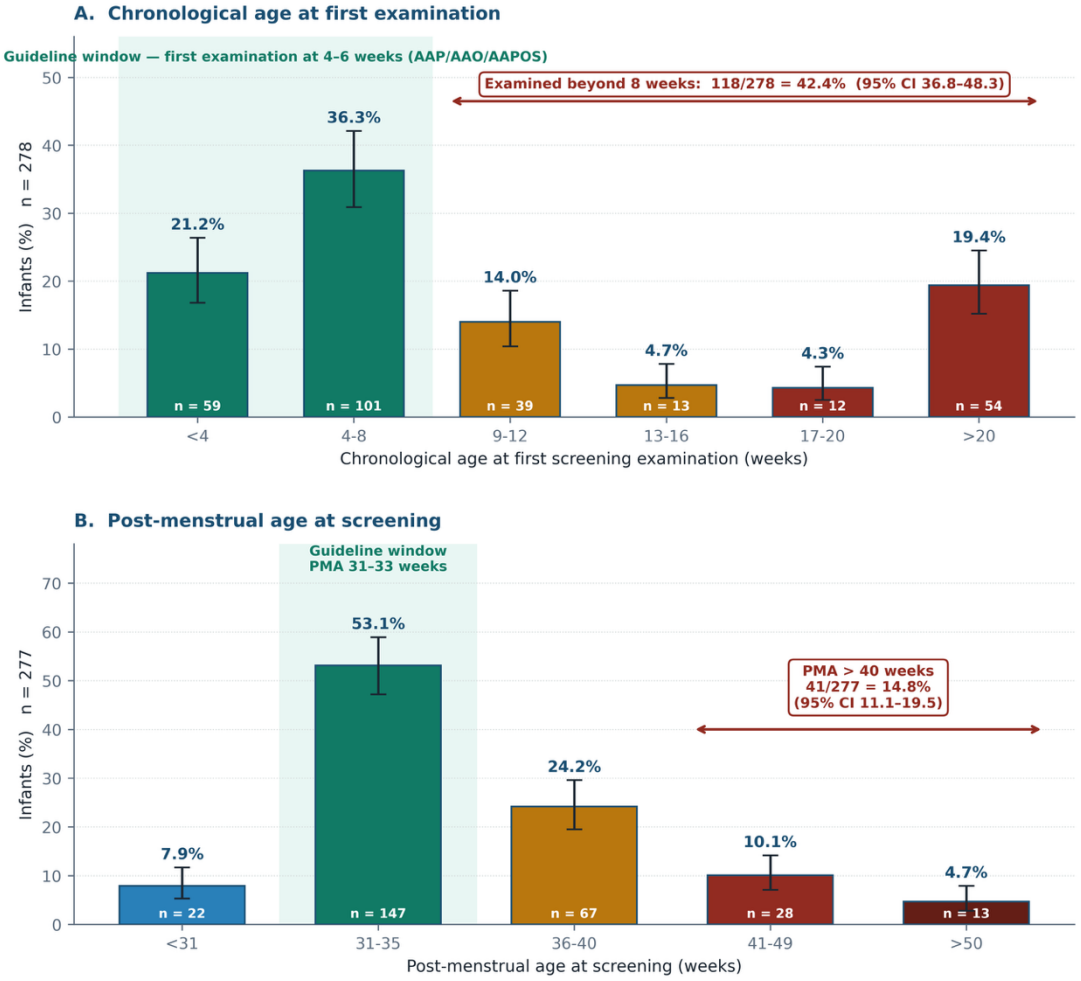


*Figure 3. Timeliness of the first screening examination. (A) Chronological age at first examination (n = 278); the shaded band marks the guideline window of 4 to 6 weeks. (B) Post-menstrual age at screening (n = 277); the shaded band marks the guideline window of 31 to 33 weeks. Error bars are Wilson score 95% confidence intervals.*

## *Management*

Management dispositions are shown in Table 4. Two hundred and fifty-four infants (91.7%, 95% CI 87.8–94.4) were managed by observation with scheduled re-examination, and 23 infants (8.3%, 95% CI 5.6–12.2) were referred for treatment. The number referred slightly exceeded the 22 infants with stage 3 or worse disease.

The 23 referrals correspond closely to the 22 infants with stage 3 or worse disease. The eight infants with ROP who were managed by observation all had stage 2 disease, for which observation with scheduled re-examination is the guideline-concordant management in the absence of plus disease. Because plus disease was not recorded, referral decisions could not be independently audited against type 1 criteria.

Expressed as screening yield, 8.9 infants (95% CI 6.5–12.5) had to be screened to detect one case of ROP of any stage, 12.6 (95% CI 8.5–18.9) to detect one case of sight-threatening disease, and 23.1 (95% CI 13.5–40.1) to detect one infant with retinal detachment.

**Table 4. Management disposition at first screening examination (n = 277). CI, confidence interval.**

| Management disposition | n (%) | 95% CI |
|---|---|---|
| Observation with scheduled re-examination | 254 (91.7) | 87.8–94.4 |
| Referral for treatment | 23 (8.3) | 5.6–12.2 |

## *Comparison with national and international reference values*

Applying the published Indonesian gestational-age-stratum-specific ROP rates to this cohort's gestational-age distribution yielded an expected count of 19.21 ROP cases, comprising 5.04 cases expected among the 28 infants below 28 weeks, 10.29 among the 147 infants at 28–32 weeks and 3.88 among the 102 infants at 32 to under 37 weeks. The 31 cases observed represent an excess of 11.79 cases and a standardised morbidity ratio of 1.61 (95% CI 1.10–2.29; p = 0.016). The observed and expected distributions are compared in Figure 4A, and the numerical detail is given in Table 5.

The three pre-specified benchmark comparisons are shown in Figure 4B. The observed ROP prevalence of 11.2% was substantially and significantly lower than the pooled global estimate of 31.9% (p < 0.001), with a moderate effect size (Cohen's h = -0.52). The referral proportion of 8.3% was consistent with the internationally reported range of 5–10%, showing no significant departure from its 7.5% midpoint (p = 0.569; h = 0.03). By contrast, the proportion of ROP cases

presenting at stage 4–5 was 38.7% against a 10% benchmark, a large and highly significant excess ($p < 0.001$; $h = 0.70$).

The composition of the excess is examined in the multivariable analysis below.

**Table 5. Indirect standardisation of the observed retinopathy of prematurity burden against published Indonesian gestational-age-stratum-specific rates, and pre-specified benchmark comparisons. Expected counts are the sum across strata of stratum size multiplied by the published national rate. SMR, standardised morbidity ratio, with Byar 95% confidence interval; h, Cohen effect size for the difference between two proportions; V, Cramer V.**

| Comparison | Observed | Reference | Effect (95% CI) | p |
|---|---|---|---|---|
| **Indirect standardisation by gestational-age stratum** | | | | |
| <28 weeks (n = 28) | — | 18.0% | 5.04 expected | — |
| 28–32 weeks (n = 147) | — | 7.0% | 10.29 expected | — |
| 32–<37 weeks (n = 102) | — | 3.8% | 3.88 expected | — |
| **All strata combined (n = 277)** | **31 cases** | **19.21 expected** | **SMR 1.61 (1.10–2.29)** | **0.016** |
| **Benchmark comparisons** | | | | |
| ROP prevalence | 11.2% (8.0–15.4) | 31.9% | h = -0.52 | <0.001 |
| Referral proportion | 8.3% (5.6–12.2) | 7.5% | h = 0.03 | 0.569 |
| Advanced ROP among ROP cases | 38.7% (23.7–56.2) | 10.0% | h = 0.70 | <0.001 |
| Gestational-age distribution vs national survey | — | — | $\chi^2 = 17.82$, V = 0.179 | <0.001 |

## *Sensitivity of the standardised morbidity ratio*

The five pre-specified sensitivity analyses are presented in Table 6. The standardised morbidity ratio was robust to gestational-age misclassification in the conservative direction: reclassifying half the sub-28-week stratum into the 28–32-week stratum raised the ratio to 1.75 (95% CI 1.19–2.49), and reclassifying the whole stratum raised it to 1.92 (95% CI 1.31–2.73). Because last-menstrual-period dating tends to overestimate maturity in this setting, any such error would make the reported ratio an underestimate.

The ratio was not robust to two other assumptions. If the national reference rates under-ascertained ROP by 25%, the ratio fell to 1.29 (95% CI 0.88–1.83); at 50% under-ascertainment it fell to 1.08 (95% CI 0.73–1.53). Substituting the un-reconciled source count of 26 cases gave 1.35 (95% CI 0.88–1.98).

The most informative analysis was the exclusion of stage 4–5 disease. Counting only the 19 infants with stage 2 or stage 3 ROP against the same expected count returned the ratio to 0.99 (95% CI 0.60–1.54). The excess burden identified in the primary analysis is therefore located entirely in advanced disease: this cohort did not carry more early ROP than national rates predict, but it carried substantially more retinal detachment.

**Table 6. Sensitivity of the standardised morbidity ratio to the assumptions on which it rests.**

| Scenario | Observed | Expected | SMR (95% CI) | p | Interpretation |
|---|---|---|---|---|---|
| **Primary analysis** | **31** | **19.21** | **1.61 (1.10–2.29)** | **0.016** | **Reconstructed counts; N = 277** |
| Denominator 278 assumed | 31 | 19.21 | 1.61 (1.10–2.29) | 0.016 | SMR compares counts, so it is unchanged |
| Source count 26 used instead | 26 | 19.21 | 1.35 (0.88–1.98) | 0.161 | If the un-reconciled ROP total were correct |
| All <28 wk reclassified to 28-32 wk | 31 | 16.13 | 1.92 (1.31–2.73) | 0.001 | Extreme GA-misclassification scenario |
| Half of <28 wk reclassified | 31 | 17.67 | 1.75 (1.19–2.49) | 0.005 | Moderate GA-misclassification scenario |
| National reference rates +25% | 31 | 24.01 | 1.29 (0.88–1.83) | 0.192 | If national rates under-ascertained ROP |
| National reference rates +50% | 31 | 28.81 | 1.08 (0.73–1.53) | 0.732 | Severe national under-ascertainment |
| **Excluding stage 4-5 (n = 19)** | **19** | **19.21** | **0.99 (0.60–1.54)** | **1.000** | **Counting only stages 2-3 as screen-detected** |

## *Multivariable partial identification*

Results of the multivariable analysis are given in Table 7 and displayed in Figure 4C and Figure 4D.

Stratum-specific risk. The observed margins alone place the ROP risk of the 28 infants born before 28 weeks anywhere in 0.0–100.0%, that of the 147 infants born at 28–32 weeks in 0.0–21.1%, and that of the 102 infants born at 32 to under 37 weeks in 0.0–30.4%. Adding the assumption that risk does not increase with gestational age narrows these to 11.2–100.0%, 1.2–17.7% and 0.0–11.2% respectively, corresponding to 3.13–28.00, 1.77–26.04 and 0.00–11.42 cases. Expressed against the nationally expected counts, the stratum-specific standardised morbidity ratios are bounded by 0.62–5.56, 0.17–2.53 and 0.00–2.95.

This produces an instructive asymmetry. The aggregate standardised morbidity ratio is identified and significantly above unity, yet not one of the three stratum-specific ratios it decomposes into is identified: each region contains unity. The excess is established for the cohort as a whole and cannot be attributed to any particular maturity band from these data.

Proportional-excess model. Under the one-parameter model the common factor is 1.614, implying stratum risks of 29.1%, 11.3% and 6.1% and case counts of 8.13, 16.61 and 6.26. All three lie inside the assumption-free identification region, so the model is admissible; it is nonetheless a model, and its stratum values are not data.

Adjusted association with oxygen. The Mantel-Haenszel odds ratio for prolonged oxygen adjusted for gestational age is not identified, and this can be shown directly rather than numerically. Assigning all 31 ROP cases to the 68 infants exposed for 7 days or less is consistent with every margin and sets the numerator to zero; assigning all 31 to the 209 infants exposed for longer is likewise consistent and sets the denominator to zero. The identification region is therefore the whole of the non-negative half-line, and imposing both monotonicity assumptions does not alter it. No adjusted association between oxygen exposure and

ROP can be recovered from these data at any level of precision, and none is reported.

Birth weight as a confounder. Because 275 of 278 infants fall in a single birth-weight stratum and none falls below 1000 g or at or above 2500 g, the mass available for reallocation between birth-weight strata is 3 infants. Re-weighting to any birth-weight distribution can therefore move any birth-weight-adjusted proportion by at most 1.08 percentage points, confining the ROP prevalence to 10.1–11.2% under every possible adjustment. Birth weight is effectively constant in this cohort and cannot confound; this is why the standardisation is carried out on gestational age alone.

Two-factor standardisation. Carrying an external rate ratio for prolonged oxygen alongside the national gestational-age rates, and bounding the expected count over every admissible gestational-age by oxygen table, the two-factor standardised morbidity ratio is 1.55–1.72 at an assumed rate ratio of 1.5, 1.52–1.78 at 2.0 and 1.48–1.85 at 3.0 (Figure 4D). The ratio remains above unity throughout, so the excess is not explained by the cohort's high prevalence of prolonged oxygen exposure under any external effect size in the plausible range.

Two negative findings are recorded for completeness. The maximum-entropy joint distribution consistent with the margins is the independence table, whose stratum risks are all 11.19% and whose adjusted odds ratio is exactly 1.00; it asserts the null by construction and is therefore useless as a point estimate. And the national figure of 4–5% for severe ROP admits two readings that the source does not disambiguate — a proportion of all screened infants, or a proportion of ROP cases — which yield standardised ratios for advanced disease of 0.87–1.08 and 12.5–15.6 respectively, differing by a factor of about 18. No standardised ratio for advanced disease is therefore reported, and the severity findings rest on internal comparisons that do not use that figure.

**Table 7. Multivariable analysis by partial identification.**

| Multivariable parameter | Margins only | + monotonicity in GA | Model-based reference | Status |
|---|---|---|---|---|
| **Stratum-specific ROP risk** | | | | |
| <28 weeks | 0.0–100.0% | 11.2–100.0% | 29.1% | Not identified |
| 28–32 weeks | 0.0–21.1% | 1.2–17.7% | 11.3% | Not identified |
| 32–<37 weeks | 0.0–30.4% | 0.0–11.2% | 6.1% | Not identified |
| **Stratum-specific SMR** | | | | |
| <28 weeks | — | 0.62–5.56 | 1.61 | Contains 1 — not identified |
| 28–32 weeks | — | 0.17–2.53 | 1.61 | Contains 1 — not identified |
| 32–<37 weeks | — | 0.00–2.95 | 1.61 | Contains 1 — not identified |
| **Adjusted association** | | | | |
| Mantel–Haenszel OR, oxygen >7 d, adjusted for GA | 0 to ∞ | 0 to ∞ | — | Provably unidentified |
| **Confounding by birth weight** | | | | |
| Maximum shift in any adjusted proportion | — | 1.08 percentage points | — | Bounded — cannot confound |
| **Two-factor standardised morbidity ratio** | | | | |
| external oxygen rate ratio 1.0 | — | 1.61–1.61 | — | Excess retained |
| external oxygen rate ratio 1.5 | — | 1.55–1.72 | — | Excess retained |
| external oxygen rate ratio 2.0 | — | 1.52–1.78 | — | Excess retained |
| external oxygen rate ratio 2.5 | — | 1.50–1.82 | — | Excess retained |
| external oxygen rate ratio 3.0 | — | 1.48–1.85 | — | Excess retained |

## 4. Discussion

### *Principal findings*

Among 277 preterm infants screened for retinopathy of prematurity at M. Djamil General Hospital, Padang, over twelve months, ROP was detected in 11.2% (95% CI 8.0–15.4) — roughly a third of the pooled global estimate of 31.9%.[4] Read alone, that figure suggests a favourable disease burden. Three further findings make that reading untenable. First, no infant presented with stage 1 disease, while 71.0% of all ROP was already at stage 3 or worse and 38.7% had progressed to partial or total retinal detachment. Second, the observed case count exceeded the number expected from published Indonesian stratum-specific rates by 61% (SMR 1.61, 95% CI 1.10–2.29; p = 0.016), despite a cohort composition weighted toward more mature infants who should have generated fewer, not more, cases — and, decisively, that excess disappeared when stage 4–5 disease was excluded (SMR 0.99, 95% CI 0.60–1.54), locating all of it in advanced disease. Third, 42.4% of infants were first examined beyond 8 weeks of chronological age. Taken together these observations point to a programme detecting disease late rather than encountering it rarely.

### *Interpreting the prevalence estimate*

The gap between the 11.2% observed here and the 31.9% pooled global estimate is real but must be interpreted with care. Pooled international estimates are dominated by cohorts of very low birth weight infants in high-income neonatal units, where survival at 24–27 weeks is routine and the denominator is therefore enriched for the highest-risk infants.[4,6] Series restricted to extremely low birth weight infants report incidences far above the pooled figure,[7] whereas programmes screening a broader and more mature population report lower ones.[28] The present cohort is quite different: only 10.1% were born before 28 weeks and 98.9% weighed 1500–2500 g. A lower crude prevalence in such a population is expected on composition alone, and the moderate effect size for the comparison (h = -0.52) is consistent with a difference driven substantially by case mix.

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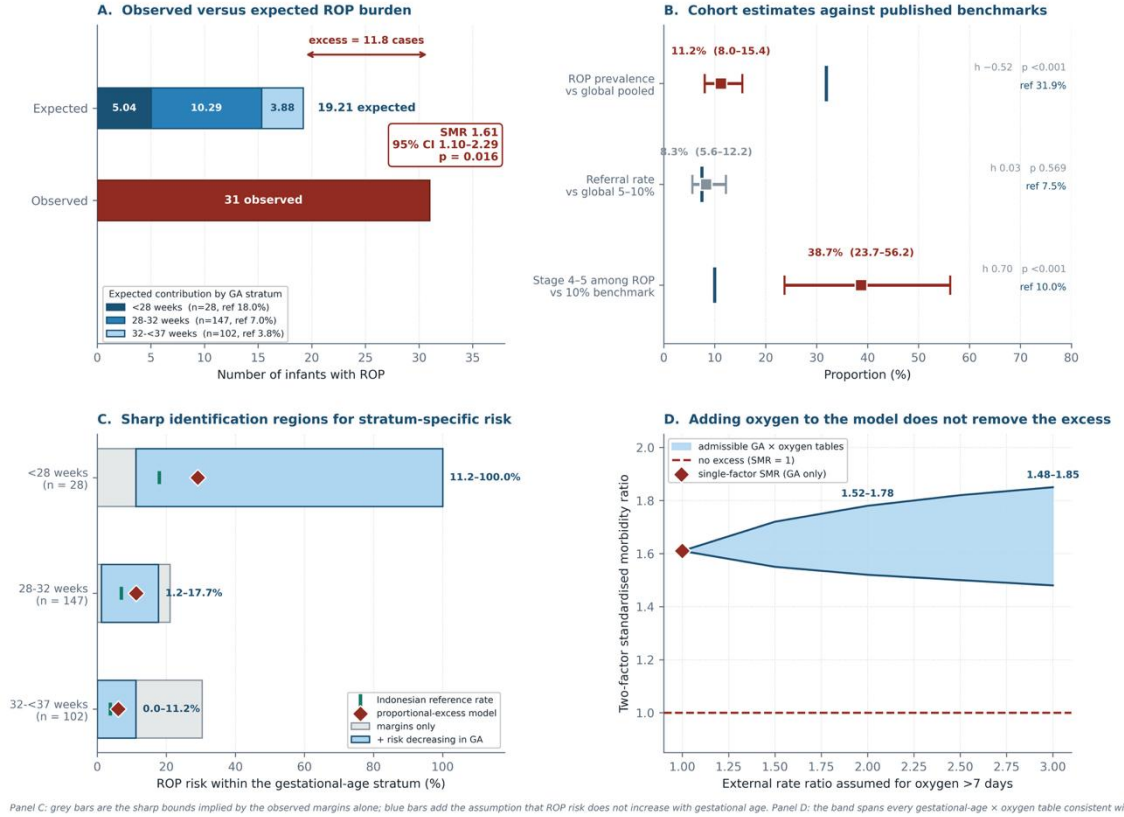


***Figure 4.*** *Observed burden against reference expectations, and what the margins do and do not identify. (A) Indirect standardisation: the expected count decomposed by gestational-age stratum against the observed count. (B) Cohort estimates against published benchmarks; squares are point estimates with 95% confidence intervals and vertical rules mark reference values. (C) Sharp identification regions for stratum-specific ROP risk; grey bars are implied by the observed margins alone, blue bars add the assumption that risk does not increase with gestational age, the green rule marks the Indonesian reference rate and the diamond the proportional-excess model. (D) Two-factor standardised morbidity ratio as a function of the external rate ratio assumed for prolonged oxygen; the band spans every gestational-age by oxygen table consistent with the observed margins.*

This is precisely why indirect standardisation matters. By applying Indonesian stratum-specific rates to this cohort's own gestational-age distribution, the reference expectation is adjusted for the very composition difference that deflates the crude comparison. The resulting SMR of 1.61 reverses the direction of the naive conclusion: relative to what national data predict for a population of this maturity, this cohort carried more ROP, not less. Crude prevalence and standardised burden thus tell opposite stories, and only the latter is adjusted for who was actually screened.

The sensitivity analysis then localises that excess precisely, and this is the finding on which the paper turns. Excluding stage 4–5 disease returns the ratio to 0.99 (95% CI 0.60–1.54). The programme is therefore not encountering more retinopathy of prematurity than national rates predict; it is encountering the expected amount of early disease and a large excess of retinal detachment. That is not a description of elevated incidence. It is a description of disease that was allowed to progress before it was seen.

Two caveats bound this interpretation. The ratio is sensitive to the accuracy of the national reference rates: if the reference survey under-ascertained ROP by a quarter or more, the primary excess would no longer be statistically distinguishable from unity, although the stage-specific asymmetry would remain. And the reference figures are described in their source as incidence over a screening course, whereas the present study measures ascertainment at a single first examination. If so, a first-examination figure should be lower than a full-course cumulative incidence, and the excess reported here is conservative rather than inflated. The comparison should nonetheless be read with that mismatch in mind.

## *Disease severity and the signature of delayed detection*

The stage distribution is the most clinically arresting finding. In a screening programme operating within guideline windows, the modal detected lesion should be early — stage 1 or stage 2 disease at the vascular-avascular junction — because infants are examined during the transition between the vaso-obliterative and vaso-proliferative phases.[1,12] The complete absence of stage 1 disease, combined with 12 infants already carrying tractional retinal detachment at their first recorded examination, is difficult to reconcile with timely surveillance. Two mechanisms could produce it, and they are not mutually exclusive: early lesions may have arisen and progressed before the infant ever reached the clinic, or early lesions may have arisen and spontaneously regressed, leaving only the subset that progressed to be counted. Both mechanisms require that the first examination occurred late, and the timeliness data show that it frequently did.

A third mechanism must be considered before delay is accepted: stage 1 disease may have been present but not recorded. A flat demarcation line at the vascular-avascular junction is a subtle finding, easily missed without scleral indentation, in an unsettled infant, or through the tunica vasculosa lentis common in this population. Since the source records do not document whether indentation was used, this possibility cannot be excluded. Three considerations nonetheless favour delay as the principal explanation. Twelve retinal detachments cannot be an ascertainment artefact. The timeliness data provide an independent line of evidence that first examination frequently occurred late. And zone I vascularisation was likewise absent, which under-detection of a subtle ridge would not explain but late examination would. The definitive test would compare the stage distribution between infants examined early and late, which requires the cross-tabulation these records

do not contain; it is the single most valuable analysis a prospective successor study could perform.

Strelnikov and colleagues documented exactly this severity shift, reporting that screening delay increases the proportion of treatment-requiring disease at first presentation,[23] and multicentre work has shown how often scheduled follow-up is not completed.[24,25] Even in national systems with organised surveillance, retinal detachment from ROP persists and is concentrated among infants whose screening deviated from protocol.[26] Against a 10% benchmark for stage 4–5 among ROP cases, the 38.7% observed here represents a large effect ($h = 0.70$, $p < 0.001$). The clinical consequence is severe and largely irreversible: laser photocoagulation and intravitreal anti-VEGF therapy are highly effective for type 1 disease,[20,21,29] but neither restores vision once the retina has detached. Non-surgical management of stage 4A retains some prospect of anatomical success,[22] whereas surgical outcomes in detachment complicating aggressive ROP remain poor,[29] and aggressive disease frequently requires retreatment even when treated promptly.[30] Twelve infants in a single year at a single centre reached that point. Where anti-VEGF therapy is used, prolonged surveillance is mandatory because of late reactivation, which lengthens rather than shortens the follow-up burden.[30] The mechanism and the pathway on which it fails are summarised in Figure 5.

It is worth stating what these data cannot establish. The dataset records the stage at first examination but not the interval between referral request and examination, nor the reason for any delay. Whether the delay originated in late referral from peripheral facilities, in clinic capacity, in transport and cost barriers to families, or in some combination, cannot be determined here and should be the subject of a dedicated pathway audit.

The argument of this paper rests on the conjunction of three observations rather than on any one of them. Prevalence below expectation on a crude comparison could reflect a low-risk population. A severity distribution skewed toward advanced disease could reflect referral bias. Examination outside the guideline window could occur without consequence. Each signal is weak alone, and each admits an innocent explanation. Together, and with the excess burden confined to advanced disease, they are difficult to reconcile with any mechanism other than delayed detection.

### *Demographic and perinatal profile in context*

The male predominance of 58.1% is somewhat higher than the approximately 53% male composition of screening populations reported in the meta-analysis by Hoyek and colleagues of more than 170,000 infants.[31] Interpretation should be cautious: Hundscheid and colleagues, in a Bayesian meta-analysis, found male sex unrelated to overall ROP incidence (RR 1.00) while modestly increasing the risk of severe disease (RR 1.14).[32] The male excess observed here is therefore more plausibly a feature of which infants were referred than evidence of sex-related susceptibility, and no cross-tabulation of sex against outcome was available to test the question directly.

The birth-weight distribution is the most distinctive feature of this cohort and the one that most clearly locates it internationally. That 98.9% of infants weighed 1500–2500 g, with none below 1000 g, is the inverse of the profile seen in high-income settings, where ROP overwhelmingly affects infants under 1000 g. This is the pattern Gilbert and colleagues described as the third epidemic: in middle-income countries, severe ROP occurs in heavier and more mature infants because neonatal survival has improved while oxygen governance and monitoring have not kept pace.[8] Comparable programmes in South Africa[33] and in other resource-constrained tertiary settings[9] report the same phenotype, and a provincial screening audit has documented how it interacts with limited service capacity.[34] Pooled analyses nonetheless continue to identify very low gestational age as the strongest single predictor of ROP,[35] which is why the standardisation in this study was anchored on gestational-age strata. The broader Indonesian national screening criteria, which admit infants up to 34 weeks and up to 2000 g with risk factors, are an appropriate response to this reality,[11] and the present data support retaining them.

One internal inconsistency in the source record deserves explicit mention rather than silent accommodation. Twenty-eight infants were recorded at a gestational age below 28 weeks, yet only three weighed less than 1500 g. These two distributions are not readily compatible, since median birth weight at 27 weeks of gestation is approximately 1000 g and a weight of 1500–2500 g at that gestation would be far above the 97th centile. The most probable explanation is misclassification of gestational age, which in this setting is frequently estimated from the last menstrual period rather than from early ultrasound biometry. Because the direction and magnitude of any such error cannot be recovered from the record, no analysis in this study relies on the joint distribution of gestational age and birth weight, and the standardisation result should be regarded as sensitive to gestational-age misclassification.

Supplemental oxygen beyond 7 days was documented in 75.5% of infants. Estrada and colleagues demonstrated a dose-dependent relationship in which each additional day of oxygen incrementally raises ROP odds independent of gestational age,[14] and Choi and colleagues identified the first seven weeks of life as the interval in which oxygen exposure most strongly predicts severe disease.[15] Mechanistically, sustained relative hyperoxia suppresses hypoxia-inducible factor 1-alpha and VEGF, arresting vascular growth; the subsequent hypoxia of the enlarging avascular retina then drives the VEGF surge that produces pathological neovascularisation.[12,13] A prolonged-oxygen prevalence of three quarters is high and identifies oxygen governance in referring neonatal units as a target for intervention. The available data cannot support any stronger claim: without recorded inspired oxygen fractions, saturation targets or cross-tabulation against outcome, no association between oxygen duration and ROP is estimable in this cohort, and the partial-identification analysis below shows that this is a matter of identification rather than of precision.

### *What the multivariable analysis establishes, and what it cannot*

Reporting that a dataset cannot support multivariable regression is common; establishing what it can support is less so. The partial-identification analysis presented here separates three questions that a conventional 'no adjusted analysis was possible' statement leaves fused together.

The first is whether the aggregate excess survives adjustment for the cohort's other major exposure. It does. Prolonged oxygen exposure was documented in three quarters of these infants, and a sceptical reader might reasonably suspect that the excess relative to national rates simply reflects an unusually heavily oxygen-exposed population. The two-factor standardisation answers that directly: across every admissible gestational-age by oxygen table and every external oxygen rate ratio between 1.0 and 3.0, the standardised ratio stays between 1.48 and 1.85. Oxygen exposure cannot explain the excess away. Birth weight cannot either, and for a stronger reason — with 98.9% of infants in one stratum it is not a variable in this cohort at all, and the exact bound of 1.08 percentage points quantifies the point.

The second is where within the cohort the excess sits. Here the answer is that the data do not say. Each stratum-specific standardised ratio has an identification region containing unity, even under the monotonicity assumption. The aggregate is identified while none of its components is — an asymmetry worth stating plainly, because a reader who saw only the overall ratio of 1.61 might reasonably assume it could be attributed to the most immature infants, and it cannot be.

The third is whether anything can be said about oxygen as a risk factor in these infants. Nothing can. The adjusted odds ratio's identification region is the entire non-negative half-line, and that is a proof rather than a numerical limitation: the margins are consistent both with every case having occurred among the briefly exposed and with every case having occurred among the prolongedly exposed. No assumption available from the literature closes this. The high prevalence of prolonged oxygen exposure in this cohort is a finding about the referring neonatal units; it is not evidence of an association with ROP in these data, and it has not been presented as such.

Two further results deserve brief mention because they are the kind of quantity a reader may expect to see and should know were considered and rejected. The maximum-entropy joint distribution consistent with these margins is exactly the independence table, so it asserts the null by construction and cannot serve as a point estimate of any association. And the national reference figure for severe ROP admits two readings differing by a factor of about eighteen, so no standardised ratio for advanced disease is reported; the severity findings rest on the internal stage distribution and the decomposition of the aggregate ratio, neither of which uses it.

The practical implication is specific and is the strongest argument in this paper for a change in record-keeping. Had the gestational-age by outcome cross-tabulation been recorded — a single additional column in the screening register — the stratum risks would have been point-identified and the maturity gradient testable. Under the proportional-excess reconstruction such a table would have yielded a trend statistic of z = -3.05 across the three strata, but that figure is a property of the assumed model rather than of the data, and it is quoted only to indicate what was forgone. The cost of recording the pairing is negligible; the analytical cost of not recording it is that the central risk-factor questions in this cohort are unanswerable at any sample size.

### *Implications for clinical practice and the Indonesian health system*

The operational implication of these findings is specific. A programme whose referral rate of 8.3% already matches international expectations (p = 0.569) and whose eligibility criteria are appropriately broad does not have a case-finding problem; it has a timing problem. Effort directed at widening eligibility would add workload without addressing the mechanism generating advanced disease, whereas effort directed at compressing the interval between birth and first examination targets it directly.

A distinct and arguably more important implication concerns oxygen rather than screening. Screening detects disease; oxygen governance prevents it. Supplemental oxygen beyond seven days was documented in three quarters of this cohort, in a population of predominantly moderately preterm infants weighing 1500–2500 g who should not ordinarily require such prolonged support. No association between oxygen duration and ROP can be estimated from these data, and none is claimed. But the exposure prevalence is itself a finding, and the dose-dependent relationship established elsewhere,[14,15] together with evidence relating achieved saturation levels[16] and modelled saturation targets[17] to ROP, makes saturation targeting, alarm-limit setting and blender availability in referring neonatal units a rational focus for intervention. A screening programme that detects disease earlier reduces blindness; oxygen governance reduces the disease.

Three measures follow. First, responsibility for initiating ROP screening should sit with the referring neonatal unit rather than with the family, with a documented screening due date entered in the discharge summary of every eligible infant and a named individual accountable for it. Second, tele-ROP imaging, in which retinal images are captured in the neonatal unit and reviewed remotely, is safe and feasible in routine practice[36] and its severity scoring performs consistently across sites,[37] which suits a province served by a single tertiary centre. Neonatal nursing staff are central to such a pathway, and their knowledge and confidence in ROP screening are themselves modifiable.[38] Third, the Postnatal Growth and Retinopathy of Prematurity (G-ROP) criteria retain full sensitivity for type 1 ROP while reducing the number of infants requiring examination, and have now been validated in European,[39] North American[40] and East Asian[41] populations. Comparable algorithmic approaches include the East London criteria[42] and weight-gain-based models such as WINROP,[43] and a population-based cohort has been used to derive national criteria directly.[44] Any of these offers a route to release clinic capacity that could be redirected toward earlier first examinations rather than a larger total volume.

These measures are financially realistic within the Indonesian system. Universal coverage under Jaminan Kesehatan Nasional, administered by BPJS Kesehatan, already funds neonatal intensive care, ophthalmological examination and ROP treatment, so the barrier is one of pathway design and coordination rather than of reimbursement. Embedding a mandatory screening due date within the national perinatal record, and aligning the PNPK ROP guidance with an explicit, auditable timeliness standard, would convert an existing entitlement into reliable delivery. The cost comparison is stark. Restructuring a screening programme around validated criteria has been shown to reduce its cost without loss of sensitivity,[45] while the in-hospital costs of the morbidities of prematurity, ROP among them, are substantial in their own right.[46] A screening examination is inexpensive and repeatable; a child blinded by stage 5 ROP incurs decades of educational, social and economic cost.

Two qualifications should temper these recommendations. First, they address scheduling rather than coverage, and this study cannot establish whether coverage is also deficient: the number of eligible preterm infants in the province is not recoverable from these records, so the 278 infants screened cannot be expressed as a proportion of those who should have been. A programme that examines most eligible infants late requires different remedies from one that never sees them at all, and distinguishing the two should be the first task of any follow-on evaluation. Second, implementation requires a named instrument. The maternal and child health handbook and the national perinatal record already accompany every Indonesian infant, and a mandatory screening due-date field within them would attach the obligation to the infant rather than to the family's initiative. Whether tele-ROP review attracts a JKN tariff is a separate question that would need to be settled before such a service could be sustained.

### *Strengths*

This study has six principal strengths. First, total sampling over a complete twelve-month period at the sole tertiary referral centre for West Sumatra minimises selection bias and captures the full annual screening workload of the province. Second, all examinations were performed by an experienced paediatric ophthalmologist using a uniform technique and a single classification standard (ICROP3), which limits observer and instrument variability. Third, every proportion is reported with a Wilson score confidence interval, and the primary burden comparison is standardised against a national reference rather than presented as a crude figure, which is what allowed the apparent discrepancy between low prevalence and high severity to be resolved. Fourth, the primary inferential result was subjected to five pre-specified sensitivity analyses, which is what allowed the excess burden to be localised to advanced disease rather than asserted in general. Fifth, the constraint imposed by marginal-only data was addressed by formal partial identification rather than by assertion, which allowed three multivariable questions to be separated and answered differently: the excess survives adjustment for oxygen, birth weight cannot confound it, and the adjusted association with oxygen is provably unidentified. Sixth, the entire dataset underwent a documented

arithmetic reconciliation before analysis, and all reported values were re-derived programmatically and independently verified in the final manuscript, giving an auditable chain from source counts to printed number.

### *Limitations*

Several limitations qualify these findings. First, the cross-sectional design captures the first screening examination only. Infants were not followed longitudinally, so the natural history of the detected lesions, treatment outcomes and visual function in later childhood are all unknown, and no causal inference is possible. This matters because the outcomes that follow screening are what ultimately justify it: long-term visual outcomes after regressed ROP,[47] refractive error and amblyopia in preterm children,[48] vision-related quality of life,[49] and the ocular safety of agents used during screening[50] all lie beyond this dataset.

Second, and most consequentially, only marginal distributions were available. No cross-tabulation of exposure against outcome was recorded, so no individual-level multivariable model, odds ratio or receiver operating characteristic statistic is estimable. The partial-identification analysis establishes what the margins do determine — that the aggregate excess survives adjustment for oxygen and cannot be confounded by birth weight — and proves what they do not: the adjusted association with oxygen is unidentified over the entire non-negative half-line, and no stratum-specific standardised ratio excludes unity. Consequently this study can describe, standardise and bound burden, but it cannot identify risk factors, and no finding here should be read as evidence of an exposure-outcome association.

Third, exposure measurement is crude. Oxygen therapy was recorded only as a duration dichotomised at 7 days, without inspired oxygen fraction, saturation targets, mode of respiratory support or documentation of hyperoxic excursions. Other established contributors to ROP risk were not recorded at all: early and late onset sepsis,[51] anaemia in more mature and heavier infants,[52] platelet count,[53] and neonatal hyperglycaemia[54] have each been associated with ROP, and their omission may materially affect the interpretation of the standardised burden.

Fourth, several clinically important variables were absent from the source records and could not be analysed. Plus disease was not documented, so referral decisions could not be audited against type 1 criteria; the zone of ROP was not recorded for affected infants, although zone determines prognosis and treatment urgency; the outcome of the 23 referred infants — attendance, treatment received and anatomical result — lies outside this dataset, so the consequences of the delay this study documents cannot be quantified here; and whether scleral indentation was used at examination was not documented, which bears directly on the confidence with which the absence of stage 1 disease can be interpreted. It should also be noted that referral is not equivalent to treatment received, and loss to follow-up between the two would compound the delay reported here.

Fifth, the eligibility criteria as applied could not be fully reconstructed. National guidance admits infants of birth weight 1500–2000 g with risk factors, yet the recorded birth-weight stratum extends to 2500 g, so it is not possible to determine from these records how many infants entered screening by the gestational-age route, the birth-weight route or the risk-factor route. A broader effective net than the stated criteria would dilute the crude prevalence, which is a further reason to prefer the standardised comparison.

Sixth, this is a single-centre study at a tertiary referral hospital, which introduces referral bias. Infants reaching this clinic are not representative of all preterm infants in West Sumatra, and the severity profile observed is likely to be enriched relative to the underlying population. The prevalence estimate should therefore be read as a property of this screening pathway rather than as a population prevalence, and the SMR compares this pathway with a national reference rather than one population with another.

The direction of the referral bias in the final limitation is worth stating: infants reaching a tertiary clinic are likely to be more severely affected than those who do not, so the severity profile is probably enriched. The countervailing possibility is more troubling — that the most delayed infants in the province never arrive at all, in which case the timeliness failure documented here understates the provincial picture.

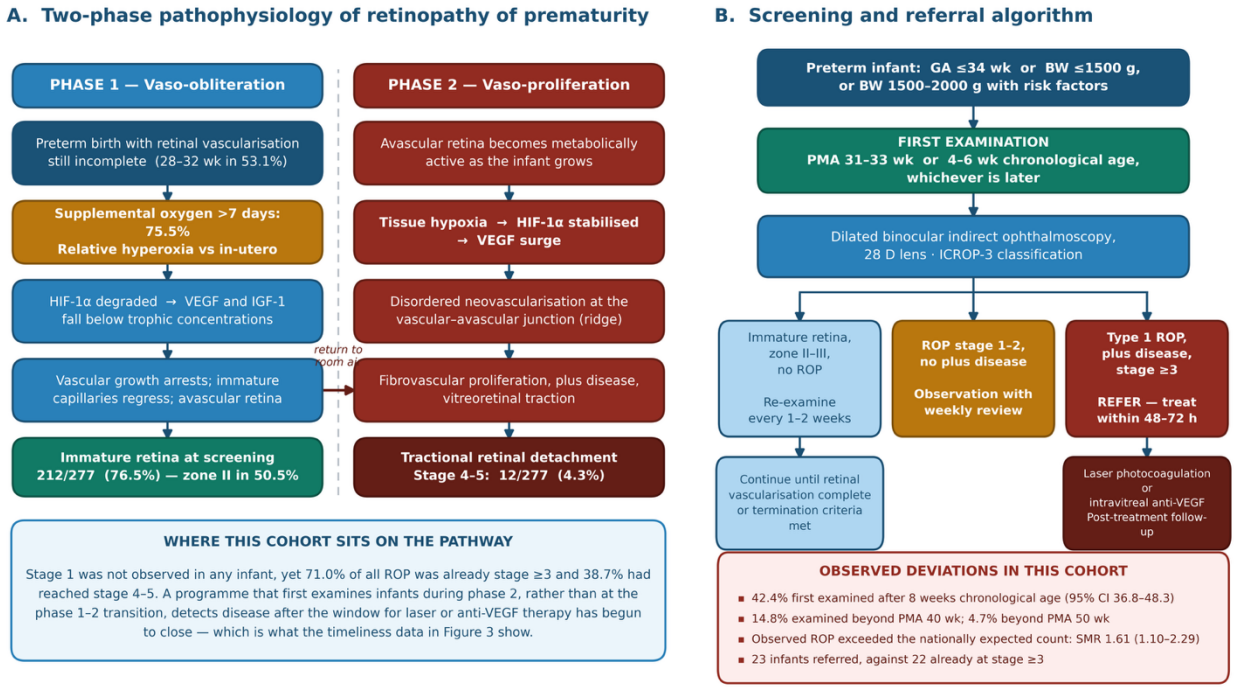


***Figure 5.*** *Mechanism and pathway. (A) The two-phase pathophysiology of retinopathy of prematurity, annotated with the corresponding observations from this cohort. (B) The screening and referral algorithm, with the deviations quantified in this study listed beneath.*

## 5. Conclusion

Among 278 preterm infants screened over twelve months at a tertiary referral hospital in Padang, retinopathy of prematurity was detected in 11.2% of the 277 infants with complete data — well below pooled global estimates, yet 61% above the count expected from Indonesian gestational-age-stratum-specific rates (SMR 1.61, 95% CI 1.10–2.29). That excess is robust to the cohort's other exposures: two-factor standardisation holds the ratio between 1.48 and 1.85 across every admissible gestational-age by oxygen table, and birth weight, being effectively constant here, can move any adjusted proportion by at most 1.08 percentage points. Sensitivity analysis then locates the excess: excluding stage 4–5 returns the ratio to 0.99 (95% CI 0.60–1.54). The severity profile agrees. No infant had stage 1 disease, 71.0% of cases were already stage 3 or worse, and 38.7% had reached retinal detachment, while 42.4% of infants were first examined beyond 8 weeks of chronological age. This programme is not encountering more retinopathy of prematurity than expected; it is encountering the expected amount of early disease and an excess of blindness-threatening disease, which is the signature of late detection rather than of high risk.

The priority is therefore timeliness rather than coverage or eligibility. Assigning responsibility for initiating screening to the referring neonatal unit, recording an auditable screening due date in the national perinatal record and maternal and child health handbook, and using tele-ROP imaging and validated G-ROP criteria to release clinic capacity are concrete steps within existing JKN coverage. Alongside these, oxygen governance in referring units addresses the disease rather than its detection. One change to the screening register would repay itself immediately: recording each infant's gestational age against its funduscopic outcome. That single pairing, absent from the present records, is what makes every risk-factor question in this cohort unanswerable, and its absence cannot be repaired by a larger sample. Future work should establish a prospective cohort recording exposure-outcome pairs, plus disease, zone, treatment received and visual outcome, so that risk factors can be identified, the stage distribution compared between infants examined early and late, and the effect of any timeliness intervention measured directly.

## Declarations

***Ethics approval and consent to participate.*** This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from Ethical Committee of CMHC Research Center, Indonesia (approval number 2025/339). The study comprised a retrospective review of existing medical records; patient identifiers were removed before analysis, and no additional patient contact, examination or intervention was undertaken for the purposes of this research.

***Availability of data and materials.*** The reconciled dataset, the analysis code and the verification harness supporting the findings of this study are available from the corresponding author on reasonable request.

***Competing interests.*** The authors declare that they have no competing interests.

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***Authors' contributions.*** NRM conceived the study, collected and curated the data, performed the analysis and drafted the manuscript. J supervised the clinical examinations, provided subspecialty interpretation of the funduscopic findings and critically revised the manuscript. Both authors read and approved the final manuscript.

***Generative artificial intelligence use.*** The authors did not use generative artificial intelligence to generate the scientific content, data extraction, or statistical analysis reported in this manuscript. The authors take full responsibility for the content of the published article.

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