Zone of retinal vascularization and refractive error in premature eyes with and without spontaneously regressed retinopathy of prematurity

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PURPOSE To evaluate the relationship between zone of retinal vascularization and refractive error in premature infants without retinopathy of prematurity (ROP) or with spontaneously regressed ROP.

METHODS The medical records of neonates screened for ROP between 2009 and 2015 at a tertiary academic center were reviewed retrospectively. Cases included untreated eyes with spontaneously regressed ROP; premature eyes without a diagnosis of ROP were control subjects. Primary outcomes were zone of retinal vascularization and refractive error, determined by cycloplegic retinoscopy (CR).

RESULTS Of 378 eyes evaluated, 184 had ROP, 24 of which underwent treatment and were excluded. Mean corrected age at first CR was 7.5 months. Seventeen eyes without ROP were myopic at first CR (8.8%), compared to 35 eyes with regressed ROP (21.9%). No untreated eyes had halted vasculature in zone I; notably, 44% of spontaneously regressed zone II eyes were myopic. Irrespective of ROP status, CR significantly differed by zone of vascularization ($P < 0.001$), with more myopia occurring with posterior halting of vascularization. For all eyes, CR significantly differed between complete vascularization versus zone II ($P < 0.0001$) and zone III versus zone II ($P = 0.001$); zone III versus complete vascularization did not statistically differ ($P = 0.15$). This relationship held true for untreated, spontaneously regressed ROP eyes ($P < 0.01$, $P = 0.01$, $P = 0.8343$).

CONCLUSIONS More myopic refraction occurred in neonates screened for ROP with posterior halting of vascularization. Patients with halted vascular growth in zone II should be closely monitored for myopia and refractive amblyopia. (J AAPOS 2019;23:211.e1-6)

Population-based studies suggest a rising prevalence of premature births worldwide, including industrialized countries.[1] As neonatal care advances, premature infants of younger gestational age and lower birth weight are surviving at higher rates, leading to an increase in the incidence of associated conditions such as retinopathy of prematurity (ROP),[2] which is the leading cause of visual impairment in premature infants, accounting for approximately 20,000 cases of blindness and 12,300 cases of mild to moderate visual impairment in 2010.[3] In the United States, ROP incidence grew from 14.7% in 2000 to 19.88% in 2012; it is also rising in other regions of the world.[2]

ROP screening in premature infants is primarily aimed at preventing blindness from vitreoretinopathy and retinal detachment. However, even with successful treatment or spontaneous resolution of ROP, there is a high risk of other complications, including amblyogenic refractive error, anisometropia, strabismus, reduced visual field, and deficiency in color vision and contrast sensitivity.[4,5] Historically, much attention has been given to the development of myopia in ROP, because it is otherwise uncommon in infants or preschool children. Associations include decreased birth weight, younger gestational age at birth, advanced stage of disease, and posterior pathology including macular dragging, retinal detachment, and proliferative vitreoretinopathy.[6] Reported anatomic correlations that may contribute to myopia in this population include increased lens thickness, shallow anterior chamber depth, and increased axial

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length.[7-9] Importantly, increasing evidence has shown that retinal activity may play a central role in controlling ocular growth when image quality is degraded.[10,11] Given the retinal pathology in ROP and impaired central and peripheral vision,[5] this study aimed to investigate whether the extent of retinal disease, seen as the extent of retinal vascularization, is associated with refractive outcomes.

Subjects and Methods

The study adhered to the tenets of the Declaration of Helsinki and was approved by the Institutional Review Board of the University of Illinois at Chicago. All requirements of the US Health Insurance Portability and Accountability Act of 1996 were met.

The medical records of all premature neonates screened for ROP at the University of Illinois at Chicago between January 1, 2009, and April 1, 2015, were reviewed retrospectively. Multiple variables were recorded, including gestational age, birth weight, maternal age, length of hospital stay, results of ROP screening examinations, and spherical equivalent results of cycloplegic retinoscopy (CR). Grading of ROP was stratified into zone of vascularization and stage, as previously described by the International Classification of ROP.[12] Given that myopia may independently associate with retinal diode laser treatment or anti–vascular endothelial growth factor treatment, ROP analysis was limited to eyes with untreated, spontaneously regressed disease. Control eyes include premature eyes that never developed any stage of ROP. Primary outcomes included zone of retinal vascularization and CR. Patients lost to follow-up prior to CR and patients treated for ROP were excluded.

Dilated fundus examinations were carried out by one of 4 full-time pediatric and retinal subspecialty ophthalmologists who routinely screen for ROP at least 30 minutes after administration of 0.2% cyclopentolate hydrochloride with phenylephrine 1%. The frequency of ROP examinations was at the discretion of the screening ophthalmologist, and examinations were performed as necessary until retinal vasculature matured or the ophthalmologist deemed screening was no longer necessary and not requiring treatment. Halted vascularization was defined as the zone of vascularization at the last examination recorded if the eye was not observed to have a mature, fully vascularized retina. Retinal vascular angiographic studies were not completed as part of the examination process in untreated, regressed ROP eyes. CR was performed by pediatric ophthalmologists as part of standard care at a time of their discretion at least 45 minutes after administration of cyclopentolate hydrochloride 1%, phenylephrine hydrochloride 2.5%, and tropicamide 1%.

Previous studies have suggested that myopia related to ROP begins within the first few months and progresses through the first few years of life, while myopia related to prematurity stabilizes earlier.[9] In acknowledgment of possible differences in pathophysiologic development of myopia and limitation of patient follow-up, an individual's first CR was selected for study. Pearson correlation was used to determine the correlation between age at CR and refractive error. QQ-plots and Shapiro-Wilk test for normality were used to test for normality. The nonparametric Kruskal-Wallis test was used to compare refractive errors between zones of vascularization in patients with and without ROP. This was followed by pairwise comparisons (Dwass-Steel-Chritchlow-Fligner) to determine the difference between zone groups. A P value of <0.05 was considered significant. As both eyes from each patient are reported, interocular correlation was taken into consideration by performing the same analysis with one eye per patient. The eye with more myopic CR was selected for this subanalysis, and when refractive error was equal between eyes, preference was given to the eye with ROP and lower zone of vascularization, followed by worse stage.

Table 1. Patient demographics: 354 eyes of 189 subjects[a]

Characteristic	Result
Race/ethnicity, no. (%)	
African American	106 (56)
Hispanic	64 (34)
White	10 (5)
Asian	9 (5)
Mean age at birth, weeks (range)	
Eyes without ROP	30.1 (26-35)
Untreated, spontaneously regressed ROP eyes	27.1 (24-34)
Total eyes	28.8 (24-35)
Mean birth weight g (range)	
Eyes without ROP	1249.1 (690-1950)
Untreated, spontaneously regressed ROP eyes	907.2 (495-1720)
Total eyes	1094 (495-1950)
Mean hospital stay, days (range)	
Eyes without ROP	52.7 (13-198)
Untreated, spontaneously regressed RfOP eyes	109.3 (34-550)
Total eyes	78.6 (13-550)
Mean CA age at first CR, months (range)	
Eyes without ROP	6.8 (−0.25-23.25)
Untreated, spontaneously regressed ROP eyes	8.3 (2-25)
Total eyes	7.5 (−0.25-25)
Median SE at first CR, diopters (range)	
Eyes without ROP	1.5 (−6.5-5.8)
Untreated, spontaneously regressed ROP eyes	1 (−5-5.75)
Total eyes	1.375 (−6.5-5.75)

CA, corrected age: (gestational age at birth + chronological age) − 40 weeks; *CR*, cycloplegic retinoscopy; *ROP*, retinopathy of prematurity; *SE*, spherical equivalent in diopters.
[a]Of the total of 378 eyes, 24 treated ROP eyes were excluded, leaving 354 for analysis. Of these, 194 were without ROP; 160 were untreated (ROP regressed spontaneously).

Results

A total of 378 eyes of 189 infants (96 males [51%) were identified and reviewed. Demographic information is reported in Table 1. Of the 378 eyes, 184 had ROP of any stage, and 24 of those required treatment for type 1 ROP, excluding them from this series. Staging of ROP occurred through an average of 6 inpatient and outpatient (combined with CR) dilated fundus examinations. Given

Table 2. Features of ROP and non-ROP eyes per zone of vascularization

	No ROP				ROP			
	Halted zone II	Halted zone III	Complete vascularization	*P* value[a]	Halted zone II	Halted zone III	Complete vascularization	*P* value
No. eyes (% of ROP or no ROP)	1 (0.5)	36 (18.6)	157 (80.9)		25 (15.6)	76 (47.5)	59 (36.9)	
Median age at birth, weeks[b]	27	30	30	0.48	25	27	27	<0.001
Median birth weight, g[b]	960	1165	1230	0.48	650	870	960	0.003
Median hospital stay, days[b]	59	55	47	0.04	117	100	88	0.001
Median age at first CR, CA months[b]	6.8	7.75	5.75	<0.001	8.25	7.50	7.0	0.37
Mean SE ± SD	−0.25	1.31 ± 2.31	1.63 ± 1.50	0.46	−0.54 ± 1.94	1.01 ± 1.52	1.14 ± 1.38	0.03
Median SE[b]	−0.25	1.44	1.50		0.50	1.00	1.25	
Range	NA	−6.5 to 5.75	−4.5 to 5.75		−5 to 2.1	−5 to 5.75	−2.5 to 4.88	
Myopic eyes (% within subgroup)		2 (5.6)	14 (8.9)		11 (44)	13 (17.1)	11 (18.6)	

CA, corrected age: (gestational age at birth + chronological age) − 40 weeks; *CR*, cycloplegic retinoscopy; *NA*, not applicable; *ROP*, retinopathy of prematurity; *SD*, standard deviation; *SE*, spherical equivalent, diopters.
[a]Comparisons were only performed for zone III and complete vascularization for the no ROP group, given n = 1 for zone II.
[b]Nonparametric ANOVA (Kruskall-Wallis) test of medians was performed to compare the distributions of the variables.

the established correlation of low birth weight and gestational age at birth on ROP risk, these characteristics, along with hospital stay, are reported in Tables 1 and 2.

Spontaneously regressed ROP eyes had disease ranging from regressed stage 1 to regressed stage 3 ROP, with similar frequencies. No untreated eyes had spontaneously halted vascularization in zone 1. One eye without ROP halted in zone II; this eye was excluded from analysis because of its rare occurrence and the uncertainty of effect of this singular value; the fellow eye of this subject had regressed zone II, stage 1 ROP, suggesting the possibility of similar pathology amongst both eyes. No eyes in this series showed evidence of confounding retinal disease (familial exudative vitreoretinopathy, incontinentia pigmenti, sickle cell disease, etc).

The mean gestational age at last ROP examination increased with less completed retinal vascularization. The mean gestational age was 42.5 weeks for fully vascularized eyes, 56.3 weeks for eyes halted in zone III, and 68.9 weeks for eyes halted in zone III regardless of the presence or absence of ROP. After this time, scleral depression was no longer performed, although there are cases where the zone of vascularization were confirmed because it could be visualized without scleral depression. Mean age and standard deviation at first CR was 10.3 ± 4.1 months (range 3-29); corrected age at first CR was 7.5 ± 4.0 months (range, −0.25 to 25). To investigate correlation between age at first CR and refractive error in this series, a correlation coefficient was calculated and found to be low overall ($r = -0.17$), with similar results for ROP ($r = -0.17$) and non-ROP subgroups ($r = -0.11$).

Overall, 17 of 194 premature eyes without ROP were myopic (8.8%), and 35 of 160 spontaneously regressed eyes with ROP were myopic (21.9%). Breakdown of myopic CR per zone of vascularization for non-ROP and ROP eyes are represented in Table 2. Notably, 11 of the 25 halted zone II eyes with a history of ROP (44%) were myopic.

Values of spherical equivalents (SE) per zone of vascularization in non-ROP and ROP eyes are also represented in Table 2, with similar trends of less hyperopia or more myopia with posterior zone of halted vascularization. SE data points are represented in Figure 1A (spontaneously regressed ROP eyes) and Figure 1B (no ROP). When evaluating all eyes, SE significantly differed by zone of retinal vascularization ($P < 0.0001$). Pairwise comparisons showed a significant difference between both complete vascularization versus zone II ($P < 0.0001$) and zone III versus zone II ($P = 0.001$). There was no statistically significant difference between complete vascularization and zone III ($P = 0.15$). Subgroup analysis of the 160 eyes with spontaneously regressed ROP revealed a similar, significant difference between SE and zone of vascularization ($P < 0.001$); pairwise comparison in these eyes revealed a significant difference between complete vascularization and zone II ($P < 0.01$) and zone III versus zone II ($P = 0.01$), and no difference between complete vascularization and zone III ($P = 0.8343$).

Pairwise two-sided multiple comparison analysis of the eye with the more myopic refractive error per patient similarly revealed significant differences in SE between complete vascularization versus zone II ($P < 0.005$), zone III

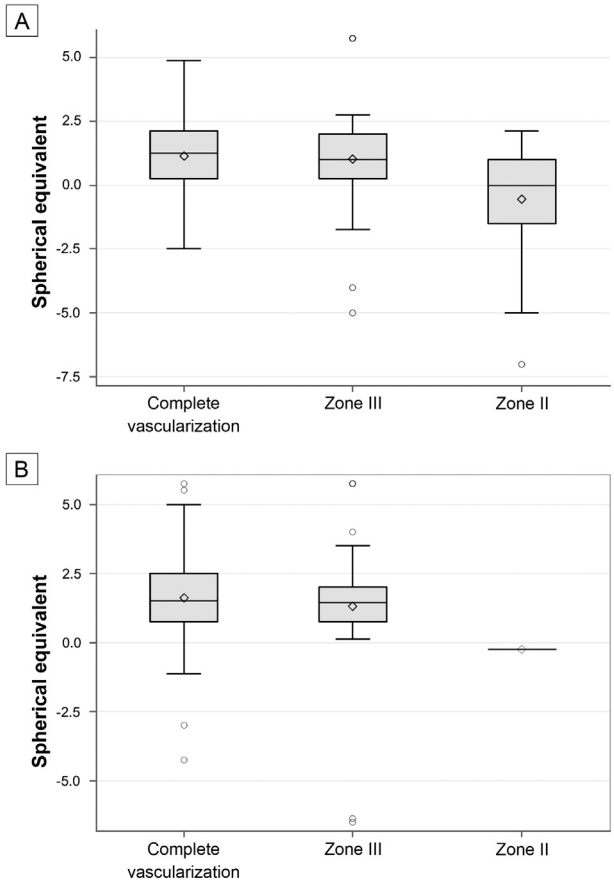

FIG 1. Distribution of spherical equivalent refractive errors in eyes with spontaneously regressed ROP (A [n = 160]) and eyes without ROP (B [n = 194]). Eyes with halted zone II ROP had a more myopic spherical equivalent refractive error ($P < 0.0001$).

versus zone II ($P = 0.02$), while complete vascularization versus zone III were not significantly different ($P = 0.49$). Kruskal-Wallis test of Wilcoxon rank-sum scores per zone of vascularization in this subanalysis revealed a statistically significant difference in SE per zone.

Discussion

Neonatal refractive error follows a normal, Gaussian distribution, averaging 2–3 D of spherical equivalent hyperopia.[13] Accordingly, mild forms of congenital myopia with full-term birth are rare, typically reported in 4%-6% of neonates; myopia over 3 D is exceedingly rare, reported in <1% of otherwise healthy infants <4 years of age.[13] In both neonatal hyperopia and myopia, changes to corneal curvature, lens power, and axial length over the first few years of life typically lessen the refractive error.[13] This process of diminished refractive error through dynamic ocular growth, or emmetropization, was first described in 1909 and originally thought to be of lenticular control. Contemporary theories, building from animal studies starting in the 1960s, suggest that optical defocus on the retina may be the central impetus affecting ocular growth.[4,10,14] The retina may locally control choroidal and posterior scleral remodeling and thickness, as well as provide afferent cortical input for control of other ocular anatomy.[10] Retinal control of scleral and choroidal thickness has been purported to be the dominant factor of emmetropization and has been thought to be governed more by peripheral than foveal retina defocus; primate studies have shown no alteration of axial length after foveal ablation, whereas opposing results occur from selective blurring of the peripheral retina.[15,16] This may be because of more robust spatial summation provided by the peripheral retina or from eccentrically concentrated signaling elements.[16] For example, dopaminergic amacrine cells, proposed to play a role controlling axial length in animal emmetropization models, have been found to be evenly distributed across the retina thereby providing a more heavily weighted extrafoveal response.[10,17] Correspondingly, a previous study has shown higher refractive error in children with peripheral versus macular retinal disease such as coloboma and retinitis pigmentosa.[18] This implication is important for diseases that affect the retinal periphery, such as ROP.

Although myopia is associated with ROP, prematurity is an independent risk factor for myopia.[19] Halting of axial growth (axial hyperopia) has been directly correlated with earlier gestational age at birth.[20-22] The shortened axial length and equatorial diameter that may arise with prematurity has been theorized to decrease zonular tension on the lens capsule, allowing a thicker lens and decreased anterior chamber depth, both of which have been reported in premature infants.[22] Furthermore, Fielder and colleagues[23] have suggested that early removal of the fetus from uterine conditions may lead to premature cooling of the cornea and an inability of the cornea to flatten, consistent with biometric studies. Cortical injury and damage to an immaturely developed visual system from systemic conditions associated with prematurity may also influence refractive outcome, perhaps from aberrant cortical control of uveal tone, with secondary effect on lenticular and axial status.[10]

Despite these innate predispositions for myopia in prematurity, ROP eyes have been shown to have a higher prevalence of and increased severity of myopia.[4,6] Although halting of retinal vascularization occurs in both eyes of premature infants and those with a history of ROP, the retinal pathophysiology differs between the two groups. Along with immature retinal vascular development that has been classically described in ROP, optical coherence tomography studies have revealed several other anatomic abnormalities. Irregular macular development with a shallower foveal pit, retained inner nuclear layers, and abnormal cellular distribution pattern have been described and are thought to be related to failure of centrifugal bipolar movement during foveal development.[4,24] Peripheral retinal rod photoreceptor dysfunction and post-receptor intralaminar reorganization and dysfunction have also been described.[4] Of note,

electroretinogram studies have correlated decreased peripheral rod sensitivity in ROP to myopic outcome.[4]

In a study by Garcia-Valenzuela and Kaufman,[7] ROP patients aged 2-18 were found to have increased lens thickness and decreased anterior chamber depth compared to premature counterparts. Inclusion criteria of −5 to −22 D of myopia and inclusion of eyes treated with cryotherapy and laser in this series highlight the variability of subject inclusion that may occur in biometric ROP studies and the conflicting results that currently exist, which the authors note.[7] With the growing evidence placed on the role of the peripheral retina governing ocular anatomy and refractive outcome, there is a comparative lack of clinical investigation controlling for the anatomic extent of vascularization and refractive outcome. Lue and colleagues[25] previously looked at refractive outcomes in premature eyes with and without untreated ROP, and other reports have investigated associations of myopia in ROP by the severity or stage of disease.[6]

Refractive outcome controlled by zone of disease is novel to this series. Here, rates of myopia were higher than expected in both groups compared to healthy subjects of similar age as reported in the literature, and a higher prevalence of myopia in all zones of vascularization was seen in ROP versus non-ROP eyes in this series, as is consistent with other studies.[6,25] A significantly higher rate of myopia occurred with zone II halting of vascularization in ROP eyes (44%). Although refractive outcomes between zone III and fully vascularized eyes did not achieve statistical significance in this study, a trend for less hyperopia (more myopia) in zone III versus mature vascularization existed. A lack of statistical significance between these two groups may signify the possibility that the small anterior crescent of retina defined by zone III is less significant in driving emmetropization, or that some zone III eyes may have eventually fully matured by the time CR was performed. Difficulty in performing peripheral retinal examinations in patients at older age and accurately and precisely separating complete vascularization versus zone III may also explain lack of statistical significance between groups. It is also possible that without angiographic confirmation of the zone of vascularization that some subjects may have vascularized beyond what was recorded. Nonetheless, high rates of myopia in halted zone II and similar outcomes between zone III and fully vascularized eyes are both important clinically with regard to CR screening and suggest that patients halted in zone II need to be followed more closely for development of significant refractive error.

Limitations of this study include its retrospective nature, the disparate number of eyes per zone of vascularization, and the potential for interexaminer variability for ROP examinations and CR. Furthermore, biometric measurements are lacking; this may be of interest for future analysis. Notably, although mean corrected gestational age at first CR was 7.5 ± 4.0 months, this series had a relatively wide range of corrected gestational ages at first CR. Various factors are implicated. Regressed ROP patients and those without ROP were on different follow-up schedules, given differences in ocular disease and comorbid, systemic disease. Those with ROP tended to follow up at younger corrected gestational ages. Furthermore, at this inner city, tertiary care center, patient's caregivers were not uniformly adherent to requested follow-up schedules, and some patients were lost to follow-up after one CR. It is unknown whether patients with suboptimal prenatal care or other risk factors for premature birth had differences in follow-up adherence; to limit potential selection bias, the authors chose to include all eligible eyes. This decision was also supported by the relatively low correlation coeffecient between CGA at first CR and spherical equivalent for all eyes in this series (−0.17), with similarly low correlation values for ROP and premature subgroups. We understand the negative value to coincide with physiologic lessening of neonatal hyperopia with age. Finally, this series had a relatively high proportion of African American and Hispanic patients, potentially limiting its generalizability. Despite the limitations, this study provides useful clinical information regarding the anatomic extent of retinal vascularization and refractive error, with specific consideration given to the high prevalence of myopia occurring in eyes halting in zone II and the similar refractive outcomes in eyes halting in zone III halting or completely vascularizing.

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