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Anisometropia and refractive status in children with unilateral congenital nasolacrimal duct obstruction

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Abstract:

OBJECTIVE: The objective of the study was to evaluate the refractive status and thereby assess anisometropia in children with unilateral congenital nasolacrimal duct obstruction (CNLDO).

STUDY DESIGN: This study design was a descriptive cross-sectional study.

PLACE AND DURATION: this study was conducted at the Department of Pediatric Ophthalmology and Strabismology, Al-Shifa Trust Eye Hospital, Rawalpindi; from August 2013 to July 2014.

METHODOLOGY: This study assessed consecutive children with unilateral CNLDO. Cycloplegic refraction on all children with CNLDO was performed followed by appropriate intervention. Refractive errors of the affected and normal eyes were compared.

RESULTS: One hundred and twenty-four children with a mean age of 29.69 ± 21.12 months (range, 2 months to 8 years) were studied. Based on spherical equivalent (SE), hypermetropia was more common in the affected eyes ($P < 0.001$). Anisometropia of >1.5 diopters (D) was present in $n = 17$ (13.7%). Interocular difference was significant for spherical error and SE ($P < 0.001$) but not cylindrical errors.

CONCLUSION: Unilateral CNLDO is associated with statistically significant anisometropia, especially anisohypermetropia which has amblyogenic potential. It is vital to perform cycloplegic refraction routinely and counsel parents regarding prognosis and regular follow-ups.

Keywords:

Anisometropia, congenital nasolacrimal duct obstruction, refractive, status

Introduction

Congenital nasolacrimal duct obstructions (CNLDO) are one of the most common cases seen in pediatric ophthalmology clinics. CNLDO occurs in 5%–15% of full-term newborns.[1] It is characterized by epiphora and intermittent discharge. It is usually unilateral or asymmetric and is mainly due to a persistent membrane at the level of Hasner valve. Approximately, 90% experience spontaneous resolution before the age of 1 year. It becomes symptomatic in merely 5%–6% of infants.[2] Intervention is usually done when CNLDO becomes persistent and/or once the child is older than 1 year of age. Our previous study on outcome of primary intubation in CNLDO showed success in 92% children <2 years of age and 90% in children between 2 and 3 years of age.[3]

NLDO has usually been considered a benign condition that does not influence visual development. It is indefinite what part, if any; persistent tearing has on visual development, refractive status, and amblyopia. A number of authors have recently described a relationship between CNLDO and the development of amblyopia and strabismus secondary to anisometropia.[4-7] The major visual concern in CNLDO is the presence of

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significant anisometropia during vital period of visual development in these infants. The objective of this current study was to describe the type, frequency of refractive error, and the severity of anisometropia in a successive series of children diagnosed with unilateral CNLDO.

Methodology

The study includes children with unilateral CNLDO who presented to our institute from August 1, 2013 to July 31, 2014. The study was approved by the Institutional Ethics Review Board of Pakistan Institute of Ophthalmology. A written informed consent was attained from all parents/guardians before enrollment.

Inclusion criteria were epiphora and/or discharge from birth which did not respond to nasolacrimal duct massage, till 1 year and up to 6 months of age when there was mucopurulent discharge. Children with unilateral involvement from the start were only studied. If the child had a previously confirmed diagnosis of CNLDO since birth; we included them in our study, no matter at what age they presented. Exclusion criteria consisted of any ocular deformity which could influence refractive status for instance ptosis, strabismus, media opacities, glaucoma, or keratopathy. Syndromic children with CNLDO and craniosynostosis were excluded from the study. Children who had a history of surgery were also excluded from the study.

Cycloplegic refraction was done with 1% cyclopentolate drops, instilled in the conjunctival sac three times, spaced out at 5, 15, and 30 min on the day of presentation; refraction was performed 30 min after last drop by means of streak retinoscopy. Children who did not achieve cycloplegia with 1% cyclopentolate were excluded from the study. No cycloplegic refraction was done under EUA. Uncooperative children were sedated with chloral hydrate syrup after pediatric consultation. In most cases owing to the epiphora and discharge, it was impossible to mask the examiner/optometrist. Complete anterior and posterior segment examinations were done by senior pediatric ophthalmologist.

All the data were analyzed using SPSS (Statistical Package for the Social Science, IBM, USA) version 17. Descriptive statistics are presented as percentages, frequencies, median, and interquartile range (IQR). The continuous data were tested for normality using Kolmogorov–Smirnov test. As the continuous data were nonparametric, the Wilcoxon signed-rank test was employed to compare the sphere, cylinder, axis, and spherical equivalent (SE) of the affected and fellow eyes. $P < 0.05$ was considered statistically significant.

Results

One hundred and twenty-four patients ($n = 124$) were included in this study with a mean age (in months) and standard deviation of 29.69 ± 21.12. The minimum age was 2 months and maximum 96 months. The number of male patients was 76 (61.3%) and female patients were 48 (38.7%). The left eye was affected in 72 (58.1%) patients and right eye was affected in 52 (41.93%). The highest hypermetropic refractive error was +6 D and highest myopic error was −5.50 D. Discharge was present in 19 cases (15.3%) whereas 105 (84.7%) patients presented with epiphora. The current study shows that the highest number of patients ($n = 75$) with unilateral CNLDO presented in the first 2 years of life (0–24 months). Three children were under 6 months of age (2, 4, and 6 months). Figure 1 shows the distribution of patients according to different age groups.

The largest interocular difference in sphere was 3 D, cylinder was 1 D, and SE was 2.5 D. Based on SE, hypermetropia was more common in affected eyes whereas myopia was more prevailing in the fellow eyes [Table 1]. Anisometropia (spherical or cylindrical >1.5 D) was present in 17 cases (13.7%).

A Wilcoxon signed-rank test revealed a statistically significant difference between spheres of affected eyes (median = 0.75, IQR = 1.50) and fellow eyes (median = 0.5, IQR = 1), $z = -4.643$, $P < 0.001$. There was also a statistically significant difference between SEs of affected eyes (median = 0.63, IQR = 1.25) and fellow eyes (median = 0.5, IQR = 1), $z = -3.831$, $P < 0.001$; and axis of affected eyes (median = 0, IQR = 0) and fellow eyes (median = 0, IQR = 75), $z = -0.760$, $P = 0.448$. However, no statistically significant difference was observed between the cylinder of affected eyes (median = 0, IQR = 0) and fellow eyes (median = 0, IQR = 0), $z = -1.892$, $P > 0.05$ [Table 2].

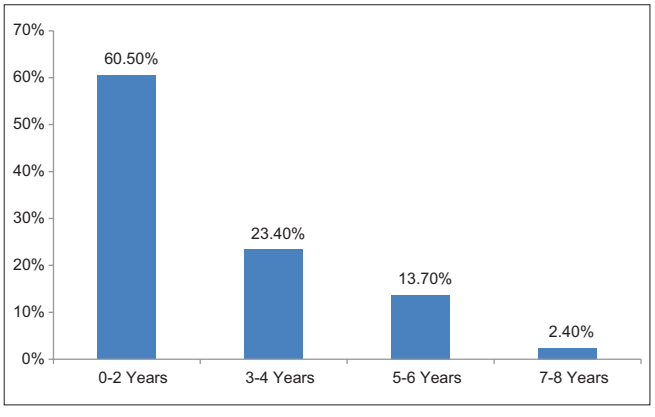

Figure 1: Age distribution of children with unilateral congenital nasolacrimal duct obstruction with corresponding frequencies

Discussion

CNLDO has been at the hub of recent debate on its proposed relationship with anisometropia, strabismus, and amblyopia. No cause-effect relationship linking CNLDO and anisometropia has been studied and the precise method by which CNLDO might cause refractive error, anisometropia, and amblyopia is indistinct. The proper focusing of images on the retina early in life is vital for emmetropization. CNLDO rarely if ever results in complete visual obstruction. Besides, early unilateral visual deprivation has been linked with myopia not hypermetropia.[8] It is postulated that accumulation of discharge, excessive tears, and antibiotic ointments may result in the deformation of retinal images. This image disparity may lead to a lack of appropriate emmetropization, and as a result, the repeated finding of anisometropia in the affected eye. It is also proposed that this anisometropia is refractory. However, recent studies reveal that this is not necessarily true.[9]

The significance of anisometropia as a source of amblyopia is well documented. Donahue suggests that 1 D of anisometropia can be considered as clinically significant anisometropia.[10] The American Association for Pediatric Ophthalmology and Strabismus guidelines state that anisometropia (spherical or cylindrical) >1.5 D is an amblyopic risk factor.[11] Nevertheless, due to individual physiologic variability's, amblyopia can even be seen with milder degree of anisometropia. The prevalence of anisometropia in the general pediatric population ranges from 2.3% to 3.4%, based on literature review.[12] Amblyopia has been reported to occur in approximately 1.6%–3.6% of the normal population.[13]

Table 1: Type of refractive error, frequencies, and comparison with fellow eye

	Affected eyes, *n* (%)	Fellow eyes, *n* (%)
Emmetropia	20 (16.1)	22 (17.7)
Myopia	1 (0.8)	6 (4.8)
Hypermetropia	80 (64.5)	61 (49.2)
Simple myopic astigmatism	1 (0.8)	1 (0.8)
Compound myopic astigmatism	4 (3.2)	2 (1.6)
Simple hyperopic astigmatism	4 (3.2)	8 (6.5)
Compound hyperopic astigmatism	5 (4.0)	15 (12.1)
Mixed astigmatism	9 (7.2)	9 (7.2)
Total	124	124

The prevalence is even higher in medically underserved populations with reported rate as high as 22.7%.[14]

Anisometropia disturbs binocularity causing reduced stereoacuity; hence, its management is more complex compared to strabismal and deprivational amblyopia. Studies demonstrate that the most important factors in treatment results are age and depth of amblyopia that are directly related to the degree of anisometropia.[15] Therefore, as the child gets older management becomes more complex and time consuming, particularly in hypermetropic anisometropes in whom a less encouraging treatment results are seen, in contrast to myopes. Based on our results, we believe that it is vital to check refractive status of children with CNLDO to assess visually significant anisometropia at an early age to prevent these children from amblyopia and visual morbidity.

First Chalmers and later Ellis questioned the relationship between CNLDO and visual maturation. Chalmers found anisometropia in 3.8%, in eyes with CNLDO; all their participants were hypermetropic in the affected eye.[16] Ellis found no appreciable increased incidence of amblyopia (1.6%) in a large series of 2249 patients with nasolacrimal duct obstruction (NLDO) compared with controls. They also found no correlation between refractive error and NLDO, including no significant increase in the incidence of anisometropia.[17]

Our prevalence of anisometropia (>1.5 D) in NLDO patients of 13.7% is approximately thrice that of the general population. It is also higher than reported studies on this subject matter.[4-6,16-19] Similarly, a study of around 1200 CNLDO patients found twice the rate of anisometropia in the unilateral CNLDO patients (7.6%) compared with bilateral NLDO patients (3.6%) that the rate of anisometropia and amblyopia are greater in NLDO patients.[4]

Matta and Silbert reviewed 375 patients with CNLDO and reported that 22% of the children with CNLDO had amblyopia risk factors.[5] Piotrowski *et al.* described a high prevalence (9.8%) of anisometropia with or without amblyopia in an 8-year consecutive case series which included 305 children with CNLDO.[6] Furthermore, Eshraghi *et al.* studied 433 cases with CNLDO that underwent probing. They reported that 5.5% had

Table 2: Median interquartile ranges of affected and fellow eyes

	n	Median of affected eyes (IQR)	Median of fellow eyes (IQR)	*Z*	*P*
Sphere (D)	124	0.75 (1.5)	0.5 (1.00)	−4.64	<0.001
Cylinder (D)		0.00 (0.00)	0.00 (0.00)	−1.89	0.059
Axis (°)		0.00 (0.00)	0.00 (75)	−0.76	0.448
Spherical equivalent (D)		0.63 (1.25)	0.5 (1.00)	−3.83	<0.001

IQR=Interquartile range

anisometropia and 9.46% had amblyopia risk factors. They also found more anisometropia in failed probing cases and theorized that structural abnormality may have a role in anisometropia.[18]

Bagheri *et al*. evaluated refractive state in children with unilateral CNLDO; they reported that, in children aged 4 years and older, the interocular difference between spherical error and SE was considerable as compared to children younger than 4 years.[19] Contrary to this, our study found no significant association between the age (in months) of the patients and the interocular difference in sphere, cylinder, and SE of affected and nonaffected eyes. We also observed that difference between the affected and fellow eyes was significant in terms of spherical refractive error and SE and that hypermetropia was more common in the eye with CNLDO. These findings suggest that when unilateral CNLDO becomes chronic, the likelihood and severity of hypermetropia increases, which as detailed, is a risk factor for amblyopia. This finding is clinically significant, as management and prognosis of amblyopia becomes intricate in older children.

Recently, Pyi Son studied 244 cases and found that early and spontaneous resolution of CNLDO is more likely to have a higher (not lower) rate of anisometropia compared to spontaneous or surgical resolution.[20] They proposed that the eye with CNLDO proceeds to emmetropization differently than the unaffected eye. Early resolution can hinder the process of emmetropization in the affected eye, making it lag behind the normal eye in achieving emmetropization. These findings negate the fact that anisometropia in CNLDO is transient and refractory. Further studies need to be done to determine the timing of resolution of CNLDO and its effect on the development of anisometropic amblyopia. In our study, we did not determine whether anisometropia persisted or not after surgical intervention or in later life. Nonetheless, Simon reported that even after CNLDO has improved, anisometropic hypermetropia is a regular finding in patients with a history of unilateral CNLDO.[7] Our results also show a high rate of anisometropia which concomitantly has amblyogenic effect.

Studies mention that emmetropia is achievable in anisometropes with appropriate management.[21] However, the precise cause why studies find high prevalence of anisometropia in subjects even after CNLDO has resolved is still contentious. Nevertheless, the results endorse the fact that patients of CNLDO should be regularly reviewed for refractory status. Furthermore, as some studies state that, in older participants, the interocular difference becomes more significant compared to younger children, this places them at high risk for developing amblyopia.[6,18-20] These facts may support the benefit of early intervention in CNLDO. However, further studies with larger sample size longer follow-up time are required to establish this effect. A cohort, visual status documentation and longer follow-up are required to answer the relationship between CNLDO and amblyopia. The cross-sectional nature of this study limits us to draw any conclusions on the relationship between CNLDO and amblyopia.

Conclusion

Unilateral CNLDO is a risk factor for anisometropia, particularly hypermetropic anisometropia with amblyogenic potential. Keeping in view that CNLDO is a common presentation in pediatric ophthalmology clinics, we recommend that all children with CNLDO should be regularly followed, even after the obstruction has anatomically and functionally resolved. These children should undergo cycloplegic refraction on each visit and should be monitored for the development of amblyopia and other ocular abnormalities.

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Conflicts of interest
The authors declare that there are no conflicts of interests of this paper.

References

1. American Academy of Ophthalmology Basic and Clinical Science Course Subcommittee. Basic and Clinical Science Course. Sec. 6. Pediatric Ophthalmology and Strabismus; 2015-2016.
2. Schnall BM. Pediatric nasolacrimal duct obstruction. Curr Opin Ophthalmol 2013;24:421-4.
3. Memon MN, Siddiqui SN, Arshad M, Altaf S. Nasolacrimal duct obstruction in children: Outcome of primary intubation. J Pak Med Assoc 2012;62:1329-32.
4. Kipp MA, Kipp MA Jr., Struthers W. Anisometropia and amblyopia in nasolacrimal duct obstruction. J AAPOS 2013;17:235-8.
5. Matta NS, Silbert DI. High prevalence of amblyopia risk factors in preverbal children with nasolacrimal duct obstruction. J AAPOS 2011;15:350-2.
6. Piotrowski JT, Diehl NN, Mohney BG. Neonatal dacryostenosis as a risk factor for anisometropia. Arch Ophthalmol 2010;128:1166-9.
7. Simon JW, Ngo Y, Ahn E, Khachikian S. Anisometropic amblyopia and nasolacrimal duct obstruction. J Pediatr Ophthalmol Strabismus 2009;46:182-3.
8. Weiss AH. Unilateral high myopia: Optical components, associated factors, and visual outcomes. Br J Ophthalmol 2003;87:1025-31.
9. Ozgur OR, Sayman IB, Oral Y, Akmaz B. Prevalence of amblyopia in children undergoing nasolacrimal duct irrigation and probing. Indian J Ophthalmol 2013;61:698-700.
10. Donahue SP. Relationship between anisometropia, patient age and the development of amblyopia. Am J Ophthalmol 2006;142:132-40.
11. Donahue SP, Arnold RW, Ruben JB, AAPOS Vision Screening Committee. Preschool vision screening: What should we be detecting and how should we report it? Uniform guidelines for

reporting results of preschool vision screening studies. J AAPOS 2003;7:314-6.
12. Borchert M, Tarczy-Hornoch K, Cotter SA, Liu N, Azen SP, Varma R, *et al.* Anisometropia in hispanic and African American infants and young children the Multi-Ethnic Pediatric Eye Disease Study. Ophthalmology 2010;117:148-53.e1.
13. Simons K. Amblyopia characterization, treatment, and prophylaxis. Surv Ophthalmol 2005;50:123-66.
14. Soori H, Ali JM, Nasrin R. Prevalence and cause of low vision and blindness in Tehran Province, Iran. J Pak Med Assoc 2011;61:544-9.
15. Caputo R, Frosini R, De Libero C, Campa L, Magro EF, Secci J, *et al.* Factors influencing severity of and recovery from anisometropic amblyopia. Strabismus 2007;15:209-14.
16. Chalmers RJ, Griffiths PG. Is congenital nasolacrimal duct obstruction a risk factor for the development of amblyopia? Br Orthopt J 1996;53:29-30.
17. Ellis JD, MacEwen CJ, Young JD. Can congenital nasolacrimal duct obstruction interfere with visual development? A cohort case control study. J Pediatr Ophthalmol Strabismus 1998;35:81-5.
18. Eshraghi B, Akbari MR, Fard MA, Shahsanaei A, Assari R, Mirmohammadsadeghi A. The prevalence of amblyogenic factors in children with persistent congenital nasolacrimal duct obstruction. Graefes Arch Clin Exp Ophthalmol 2014;252:1847-52.
19. Bagheri A, Safapoor S, Yazdani S, Yaseri M. Refractive state in children with unilateral congenital nasolacrimal duct obstruction. J Ophthalmic Vis Res 2012;7:310-5.
20. Pyi Son MK, Hodge DO, Mohney BG. Timing of congenital dacryostenosis resolution and the development of anisometropia. Br J Ophthalmol 2014;98:1112-5.
21. Atilla H, Kaya E, Erkam N. Emmetropization in anisometropic amblyopia. Strabismus 2009;17:16-9.