Ocular findings in children attending occupational therapy clinic at Kenyatta National Hospital, Nairobi, Kenya

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ABSTRACT

Objectives: **To describe the pattern of ocular abnormalities, their correlation with the physical disorders and describe associated risk factors in children attending the Occupational therapy clinic at Kenyatta National Hospital.**

Design: **Cross sectional hospital based.**

Subjects: **A hundred and eighty seven children, aged between three months and 13 years with cerebral palsy and sensory integration deficits.**

Results: **Majority of the patients had cerebral palsy(CP), 160(85.6%), while in those with sensory integration deficit(SID), attention- deficit / hyperactive disorder and autism had almost equal proportions, 20(10.7%) and 18(9.6%) respectively. Among all the children, 62% had ocular anomalies. Children with CP had a much higher prevalence (58.3%) compared to SID group (3.7%). The common ocular abnormalities included cortical visual impairment (48.7%), refractive errors (39%) and squints (34.2%). Association between physical disability and ocular anomalies was noted more in patients with CP compared with SID. Strabismus, cortical visual impairment and myopia were more likely to occur in patients with CP. Significant hyperopia was noted only in CP patients. Strabismus and cortical visual impairment were more likely to occur in patient with neonatal jaundice, while refractive errors in patients with congenital causes and optic atrophy in patients with meningitis.**

Conclusion: **Visual disabilities in children with physical disabilities were common. Cortical visual impairment, refractive errors and squints were more common. Children with CP had a much higher prevalence compared to the SID group.**

Recommendation: **All Children with CP and SID should be referred to ophthalmologist and low vision specialist for assessment.**

INTRODUCTION

There are an estimated 1.5 million blind children in the world of which 75% live in Africa and Asia. Each year, an estimated half a million children become blind, of whom up to 60% are thought to die within a year of becoming blind. The prevalence therefore markedly underestimates the burden[1]. Almost half of all blindness in children particularly those in the poorest communities is due to avoidable causes that are amenable to cost effective interventions[2]. The control of blindness in children is complex, requiring community activities through to sophisticated tertiary eye care services[3].

There is a high prevalence of ocular defects in children with developmental disabilities. This group of children has their visual disabilities ignored or overlooked. They are not examined with care due to a difficulty in making an assessment due to their mental and physical disability or with the idea that nothing much can be done to help them cope up with their already poor condition. It is important to examine all children with this kind of multi-system involvement as the incidence of ocular abnormalities is high and corrective measures at the right stage may help in overall development of the child, more so because they rely heavily on visual stimulation for their education[4].

In children with cerebral palsy (CP), the common ocular abnormalities include refractive errors, strabismus, strabismic amblopia, visual field defects and nystagmus. The various ocular defects also vary depending on the type of CP. Spastic children are more likely to have ocular defects than athetoid and ataxic children because of more extensive diffuse involvement of brain. The squints may be essentially similar as in a normal child but deviation is always greater and also the incidence of squint increases with brain damage. Similarly the type of refractive error may vary with the aetiological type. Thus premature children, affected by toxemia and dystocia generally have myopia though in overall hypermetropia is more common[4]. Dyskinetic strabismus is seen exclusively in CP

patients especially in slow, variable, and highly inefficient visual function. Its most striking feature is the fluctuation from esotropia to exotropia under the same accommodative conditions with a slow tonic deviation similar to a vergence movement. Many of the patients have athetoid CP. The association of dyskinetic strabismus with athetosis and upward gaze palsy suggests that the basal ganglia may be the site of the malfunction. This strabismus responds poorly to surgery and the associated athetosis is important in the diagnosis and treatment[5].

Autism spectrum disorder has been associated with ocular and systemic malformations in three syndromes; Möbius, CHARGE and Goldenhar syndromes. An association of autism with Duane syndrome, facial nerve palsy and ear anomalies in thalidomide-exposed individuals has also been noted[6, 7].

Patients with attention-deficit/ hyperactivity disorder (ADHD) have unexplained difficulties on tasks requiring speeded processing of coloured stimuli. Colour vision mechanisms, particularly short-wavelength (blue-yellow) pathways, are highly sensitive to various diseases, toxins and drugs that alter dopaminergic neurotransmission. Thus, slow colour processing might reflect subtle impairments in the perceptual encoding stage of stimulus colour, which arise from hypodopaminergic functioning[8].

The most conspicuous common denominator in children with dyslexia is the convergence insufficiency type of exodeviation. This finding suggests a low accommodative convergence/ accommodation ratio in these children.[9]

METHODS

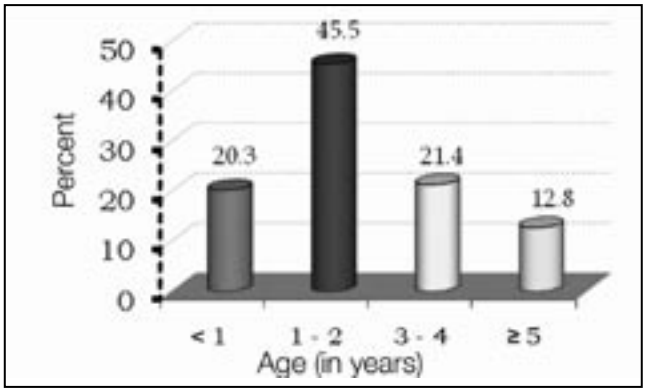
A cross sectional hospital based study was conducted at the Occupational Therapy Clinic based at Kenyatta National Hospital. The study period was between November 2007 and January 2008. Children (< 16 years) attending the sensory unit with sensory integration deficits(autism, ADHD, learning disabilities) and those from general paediatric unit with a diagnosis of cerebral palsy were seen. The minimum sample size was calculated to be 186. All the consecutive children who were present during the study period and also met the inclusion criteria were seen. Data was collected using pre designed questionnaires which was administered to the parents or guardians of the children selected for the study after obtaining an informed consent.

Data was also obtained from birth cards, MCH cards and treatment records. The children then had their eyes examined in the following order; Assessment of vision, and anterior segment examination using a torch. Where posterior segment was accessible, the eyes were dilated using tropicamide eye drops. Fundoscopy was done using indirect ophthalmoscope and a 20-dioptre loupe, followed by retinoscopy. Where poor attention of the child did not allow full assessment in one session, the examination was carried out in parts. Validation of the data was done before it was entered into the computer for analysis using the Statistical Package for social Scientists, SPSS. Results are presented in tables, bar graphs and pie charts.

RESULTS

A total of the 187 children were seen, males were 109(58%) and females were 78(42%). The male to female ratio was 1.4:1.

Figure 1: Grouped age distribution (n = 187)



The majority of the children were between one and two years (45.5%). The mean age for patients with CP was 2.3 years and that for sensory integration deficit was 4.8 years. There was a significant difference in age between the two groups (P<0.001).

Figure 2: Physical diagnosis (n = 187)

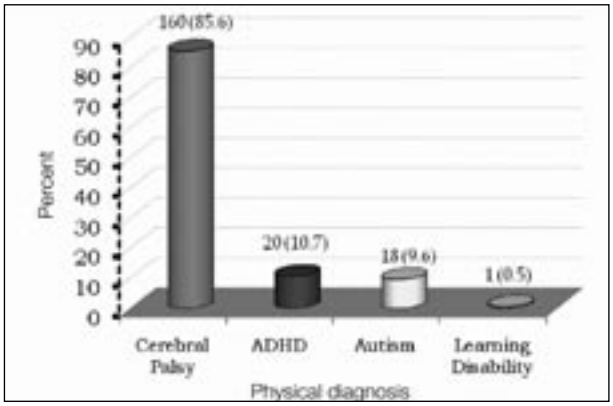


Table 1: Prevalence of ocular disorders

	Frequency	Prevalence
Adnexae	15	8.0%
Anterior segment	72	38.5%
Posterior segment	8	4.3%
Refractive errors	73	39.0%
Overall	116	62.0%

The overall prevalence of ocular disorders was 62.0% with refractive errors comprising the most (39%).

Table 2: Comparison of the prevalence of ocular disorder between sensory deficit (SID) and cerebral palsy groups

	No.	Prevalence	p-value
Cerebral palsy	164	109 (58.3%)	0.001
Sensory deficit	23	7 (3.7%)	

The prevalence of ocular disorders was much higher in the cerebral palsy group as compared to the sensory deficit with a statistically significant p- value(<0.001).

Table 3: Causes of visual impairments (n = 187)

Causes of visual impairment	Frequency	Percent
Cortical visual impairment	91	48.7
Strabismic amblyopia	25	13.4
Ocular causes	19	10.2
Not tested	9	4.8

Figure 3: Ocular signs (n = 187)

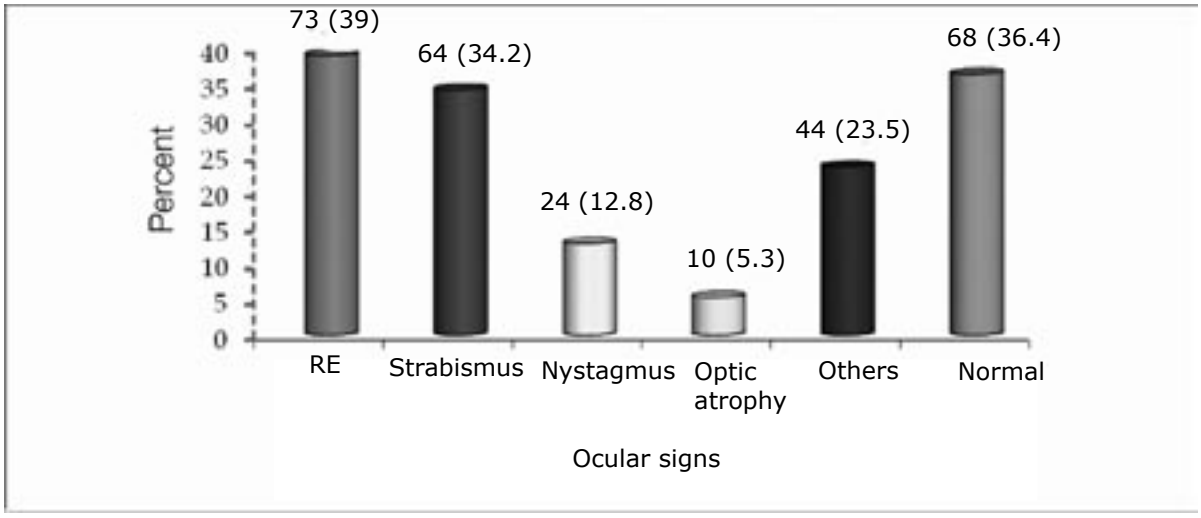


Table 4: Association between physical disease and ocular signs

	CP, n (%)	S.I.D, n (%)	OR (95% CI)	p-value
Squint	59 (92.2)	5 (7.8)	5.51 (0.82 - 15.5)	0.104
strabismic amblyopia	27 (90.0)	3 (10.0)	1.31 (0.34 - 5.99)	0.908
CVI	88 (96.7)	3 (3.3)	7.7 (2.06 - 34.05)	<0.001
Nystagmus	21 (87.5)	3 (12.5)	0.979 (0.27 - 3.58)	0.974
Myopia	43 (93.5)	3 (6.5)	2.40 (0.67 - 8.4)	0.169
Hyperopia	27 (100.0)	-	-	-
Optic atrophy	9 (90.0)	1 (10.0)	1.3 (0.15 - 10.71)	0.811

CVI=Cortical Visual Impairment

Table 5: Correlation between ocular signs and risk factors for physical disease squint, (n=48)

Factor	Squint, n (%)	OR (95% CI)	p-value
Meningitis	11 (39.3)	0.98 (0.39 - 2.45)	0.871
Birth asphyxia	25 (37.3)	0.83 (0.40 - 1.71)	0.705
Neonatal sepsis/jaundice	9 (62.5)	3.11 (0.21 - 88.76)	0.563
Congenital	3 (42.9)	1.15 (0.19 - 0.37)	0.824

Refractive errors, RE (n=63)

Factor	RE, n (%)	OR (95% CI)	p-value
Meningitis	18(54.5)	1.6 (0.75-3.50)	0.222
Birth asphyxia	32(46.4)	1.1 (0.58-2.36)	0.785
Neonatal sepsis /Jaundice	5 (38.5)	0.7 (0.23-2.37)	0.612
Congenital	7 (77.8)	4.6 (0.32 - 6.99)	0.043
Premature	1 (16.7)	0.23 (0.002-2.03)	0.153

Nystagmus, (n=17)

Factor	Nystagmus, n (%)	OR (95% CI)	p-value
Meningitis	5 (29.4)	1.26 (0.35 - 4.34)	0.766
Birth asphyxia	11 (64.7)	1.74 (0.54 - 5.72)	0.441
Neonatal sepsis/Jaundice	-		
Congenital	1 (5.9)	0.82 (0.13 - 5.56)	0.666
Premature	-		

strabismic amblyopia, (n=23)

Factor	Amblyopia, n (%)	OR (95% CI)	p-value
Meningitis	4 (12.1)	0.57 (0.15 - 1.98)	0.479
Birth asphyxia	14 (20.3)	1.47 (0.54 - 4.07)	0.552
Neonatal sepsis/Jaundice	3 (23.1)	1.45 (0.29 - 6.53)	0.700
Congenital	2 (22.2)	1.36 (0.18 - 7.99)	0.659
Premature	-		

Cortical visual impairment (CVI), (n=68)

Factor	CVI, n (%)	OR (95% CI)	p-value
Meningitis	17 (51.5)	0.96 (0.40 - 2.27)	0.932
Birth asphyxia	36 (52.2)	0.99 (0.47 - 2.09)	0.886
Neonatal sepsis/Jaundice	9 (92.5)	32.39(10.93 -149.29)	<0.001
Congenital	4 (44.4)	0.71 (0.15 - 3.26)	0.725
Premature	2 (33.3)	0.44 (0.05 - 2.94)	0.424

Optic atrophy (n= 9)

Factor	Atrophy, n (%)	OR (95% CI)	p-value
Meningitis	8 (21.2)	25.58 (2.92 - 578.57)	<0.001
Birth asphyxia	1 (1.4)	0.11 (0.00 - 0.95)	0.025
Neonatal sepsis/Jaundice	-		
Congenital	-		
Premature	-		

DISCUSSION

Children with cerebral palsy had a much higher prevalence of ocular disorders (58.3%) compared to the sensory integration deficit group (3.7%) P = 0.001. This is probably because they have more extensive brain injury evidenced by the multi- system involvement. Most studies have estimated that 54-90% of children with CP have visual disabilities[4,10,11]. The common ocular abnormalities included cortical visual impairment 48.7%, refractive errors 39% and squints 34.2%. Others included strabismic amblopia 13.4%, nystagmus 12.8%, and optic atrophy 5.3%. Corneal scars, cataracts, maculopathy and eyelid anomalies collectively comprised 5%. Of the refractive errors, myopia accounted for 60% while hyperopia occurred in 40%. Other studies report that the refractive errors are more evenly distributed or there is a bias towards hyperopia[4,10,12]. The discrepancy in our study could be due to the fact that cyclopentolate was not used for objective refraction due to increased hypersensitivity of central nervous system disturbance in patients with brain damage. Instead, tropicamide was used which leads to underestimation of hyperopia because only the manifest hyperopia is determined.

The type of refractive error varies with aetiology of CP. Fantl et al has shown that myopia predominates in spastic CP[13]. Black *et al* showed that in the dystonic cerebral palsies hyperopia predominates[14]. This has been explained as a failure of maturation of the visual system related to neonatal hypoxia. It seems clear that the normal changes with age that occur in hyperopia do not occur in cerebral palsied children. However, for this to happen it has been stated that the injury has to occur during the perinatal period; otherwise the incidence of refractive errors approaches that of a normal population[14]. In overall hyperopia predominates[4]. It was not possible to sub- classify the patients in our study according to the type of CP since the types of cerebral palsy were not indicated in the medical records for most of the patients. Significant refractive errors should be corrected with glasses where possible.

The high incidence of strabismus in the CP population is probably related to lesions in the subcortical oculomotor centres or cerebella lesions which disrupt binocular vision. Many authors have considered the treatment of squint unrewarding because of the low rate of success as assessed along conventional lines and the high incidence of consecutive squint following surgery, due in part at least to central obstacles to fusion[14]. However, Hiles et al treated 234 children with cerebral palsy and squint along conventional lines. They considered that 90% of their children achieved satisfactory ocular alignment during an average follow-up of just over four years. It thus seems reasonable that every attempt should be made to treat the squint as if the child was otherwise normal; even the purely cosmetic aspects assume importance, both for parent and patient, though squint surgery may have to be balanced against submitting a child to a surgical procedure[14].

Cortical visual impairment (CVI) was diagnosed where despite a normal ocular examination the vision was judged to be poor. The presence of CVI typically worsens the functional outcome in patients with CP. Acuity may be very poor in infancy and remain so. In others there is gradual improvement in acuity, orienting to peripheral stimuli, attention to and reaching for objects, and for social gaze. In most children with CVI, acuity does not reach normal levels. Glasses should be given if warranted, as visual abilities may improve, surprisingly so. Stimulation of the eyes by using lights and bright colours and keeping the child in brightly lit room when he /she is doing any activity can be of enormous benefit in helping the recovery process.

Other ocular anomalies reported in literature such as visual field defects and accommodation insufficiency were not assessed in this study.

These would require the subject's cooperation and we had lack of cooperation from a majority of the study patients due to their young age (87.2% were less than five years). Duckman reported reduced accommodative facility in a group of children with CP (aged 5–14 years) attending a special-need school. Those subjects with dyskinetic or ataxic CP had significantly reduced accommodative responses compared with the group of subjects with spastic CP at each accommodative demand[15]. The rare ocular syndromes associated with autism spectrum disorders (Möbius, CHARGE, Goldenhar and Duane syndromes) were not seen in this study.

Some of the known risk factors for the physical disabilities were observed to have an association with the ocular anomalies (Table 4-5). The wide range of odds ratio for some of the risk factor- ocular sign correlations was probably because the small number of patients used for comparison in those sub-groups. There is no data in literature that shows such associations between ocular anomalies and the two groups of physical disabilities examined in this study or their risk factors but Ashwal et al showed that children whose CP was due to periventricular leukomalacia were found to be more likely to have visual perceptual problems[11].

CONCLUSION

Visual disabilities in children with cerebral palsy were common. Refractive errors, squint, amblyopia, and cortical visual impairment were seen in a high percentage of these children. Lower prevalence of ocular anomalies was noted in the sensory integration deficit group compared to cerebral palsy.

RECOMMENDATIONS

- All Children with cerebral palsy and sensory integration deficits should be referred to an ophthalmologist and low vision specialist for assessment.
- The occupational, speech and hearing therapists should work closely with the low vision specialists in co-ordinating the physical and ocular rehabilitation.
- Low vision unit should be started at KNH and specialists should be trained to provide the much needed services to these children.
- Follow up of the patients for evaluation of long term outcome of the visual interventions.

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