Original Article

Cerebral visual impairment in children: Multicentric study determining the causes, associated neurological and ocular findings, and risk factors for severe vision impairment

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Purpose: To evaluate the causes, associated neurological and ocular findings in children with cerebral visual impairment (CVI), and to identify risk factors for severe vision impairment. **Methods:** A multicenter, retrospective, cross-sectional analysis was carried out from January 2017 to December 2019 on patients less than 16 years of age with a diagnosis of CVI. **Results:** A total of 405 patients were included of which 61.2% were male and 38.8% were female. The median age at presentation was 4 years (range 3 months to 16 years). Antenatal risk factors were present in 14% of the cases. The most common cause of CVI was hypoxic-ischemic encephalopathy (35.1%), followed by seizure associated with brain damage (31.3%). The most common neurological finding was seizure (50.4%), followed by cerebral palsy (13.6%). Associated ophthalmological findings were significant refractive error (63.2%), esotropia (22.2%), exotropia, (38%), nystagmus (33.3%), and optic nerve atrophy (25.9%). Severe visual impairment (<20/200) was associated with optic atrophy (odds ratio: 2.9, 95% confidence interval: 1.4–6.0; P = 0.003) and seizure disorder (odds ratio: 1.9, 95% confidence interval: 1.2–3.3; P = 0.012). **Conclusion:** The various ophthalmic, neurological manifestations and etiologies could guide the multidisciplinary team treating the child with CVI in understanding the visual impairment that affects the neuro development of the child and in planning rehabilitation strategies.

Key words: Cerebral visual impairment in children, hypoxic-ischaemic encephalopathy, severe visual impairment in children

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Cerebral visual impairment (CVI) is defined as a bilateral visual function deficit that is not limited to the visual cortex but usually involves white matter pathways. In the absence of any significant ocular pathology or anterior visual pathway diseases, it is caused by damage or malfunctioning of the retrogeniculate visual pathways. The prevalence of CVI in children and the number of visually impaired CVI children has been steadily increasing in the past few decades as modern perinatal care has increased the survival of premature and very sick full-term babies.[1-10] Improved management of other ocular diseases leading to blindness, such as cataracts, glaucoma, and retinopathy of prematurity, has decreased the burden of blindness in children.[11-13] However, there is little knowledge about CVI among ophthalmologists and unlike other diseases, this disease is not being adequately managed.

There have been only a few reports of large series of patients with CVI.[2,3,14-17] Late diagnosis and treatment can lead to childhood blindness, which has a significant impact on the psychomotor, social, and emotional development of the child. The goal of this study was to determine the causes, associated neurological and ocular findings of children with CVI, and factors associated with vision impairment. CVI affects the functional vision of the child, which interferes with overall development. Therefore, early diagnosis of CVI is important as treatment of associated ocular diseases prevents the development of amblyopia in the affected children helping in better rehabilitation.

Methods

Study design

This multi centre, retrospective study was conducted from January 2017 to December 2019 at three tertiary eye care institutes in north and central India associated with an eye consortium. These three participating institutes are tertiary eye

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care referral centres in their respective regions. The purpose of the eye consortium is to develop collaborative scientific research through consistent and robust big data from eye care institutes of central and north India. The ethics committees of all the three participating institutes and the scientific committee of the eye consortium (SCBEC/2021/April/22) approved the study. The study adhered to the tenets of the Declaration of Helsinki.

Data collection

All three participating institutes developed a common study protocol for data collection. The three institutes follow standard protocols of comprehensive eye checkups for all their patients. In all three institutes, electronic medical records are in place and the databases were searched using the diagnostic codes "cerebral visual impairment" and "cortical visual impairment." Patient data were entered in the common excel sheet, which was designed by all the participating institutes to ensure uniformity in data collection.

A review of case records of children less than 16 years with a diagnosis of CVI was done for age at presentation, gender, antenatal history, perinatal history, presenting complaint, visual acuity, cycloplegic refraction, ocular alignment and motility, and anterior and posterior segment findings. The diagnosis of CVI was made when the ocular findings did not collaborate with the vision of the child and there were associated neurological findings such as seizure, cerebral palsy, microcephaly, etc.

The causative diagnosis and neurological associations such as cerebral palsy, autism, hemiparesis, dystonia, mental retardation, etc., were taken from the history, physical findings, pediatrician or neurologist assessment, and radiology reports, as available in the records.

Visual acuity assessment in all the participating institutes followed a uniform protocol. For very young infants less than 6 months of age and children having poor vision and uncooperative children, visual behavior was measured with central, steady, and maintained (CSM) method. For infants more than 6 months of age and pre-verbal children, Lea paddle was used. Older cooperative children's vision was measured using Lea symbols and LogMAR visual acuity charts. Functional vision grading of patients was done according to the vision gradation in children with CVI suggested in the article by Huo *et al*.[1] [Appendix 1] All the patients underwent cycloplegic refraction. We divided patients into two groups based on uncorrected binocular functional vision. In one group, we included patients with equal to or better than 20/200 vision and in the other group, patients with vision poorer than 20/200.

Operational definitions

CVI

CVI was suspected in any child whose visual function could not be explained by the ophthalmological findings when the child had a history of an eventful perinatal period or neurological ailment. In these children, ocular examination revealed poor visual function bilaterally that could not be accounted for by refractive error, structural ocular examination findings, or optic atrophy.

Significant refractive error

This was graded as per the preferred practice pattern of the American Academy of Ophthalmology [Appendix 2].[18] Generally, we considered refractive error greater than +4.50 dioptres sphere and –3.00 dioptres sphere as a significant refractive error in children without squint or anisometropia, and +1.50 dioptre sphere and –3.00 dioptre sphere in children with squint or anisometropia.

Structural neurological malformations

Neurological malformations include agenesis of the corpus callosum, porencephalic cyst, lissencephaly, and chiari malformation [Appendix 3].[16]

Optic atrophy

Optic atrophy was diagnosed by the pallor of the optic nerve head as seen in dilated fundus examination by the examining ophthalmologist, and the diagnosis was supported by visual evoked potentials whenever there was a clinical doubt.

Statistical analysis

Data were collected and stored in a spreadsheet using Microsoft Excel software. Data management and coding were done in Excel. Data were analyzed using SPSS version 20.0. (SPSS Inc, Chicago, IL, USA). Descriptive analysis was primarily carried out, where categorical variables were presented in the form of frequencies, percentages, and continuous variables in the form of mean ± standard deviation. Pearson's Chi-square test was used to measure the association between individual variables using 2 × 2 or 3 × 2 contingency tables, and the effect size was calculated from these tables using Cramer's V test. Multiple logistic regression was carried out to identify independent risk factors. A P value <0.05 was considered statistically significant.

Results

Demographics

A total of 405 patients were identified with CVI during the study period of which 248 (61.2%) were male and 157 (38.8%) were female. The age at presentation ranged between 3 months and 16 years, with a median age of 4 years (mean 5.13, standard deviation [SD] ±9.19). Defective vision was the most common presenting complaint observed in 216 patients (53.3%), followed by deviated eyeball as the second most common presenting complaint in 211 (52%) patients.

Antenatal and perinatal risk factors

Antenatal and perinatal risk factors in CVI children are shown in Table 1. Normal antenatal history was observed in 240 (59.3%) patients, data were missing in 108 (6.7%) patients, and risk factors were present in 57 (14%) patients. The various risk factors were antenatal infection, gestational hypertension, and gestational diabetes. A history of premature delivery was present in 133 (37.6%) patients. An eventful perinatal period was present in 256 (63.2%) patients. The number of patients with a history of delayed cry after birth was 228 (67.5%). There were 196 (48.4%) patients with a history of admission to neonatal intensive care units for a mean duration of 12 h and oxygen was administered in 108 (26.7%) patients. In the neonatal period, seizure disorder was recorded in 184 (45.4%) patients and neonatal hypoglycemia in 44 (10.9%) patients. The delayed developmental milestone was present in 326 (80.5) patients.

Etiology

The causes of CVI are shown in Table 2. In our study population, common causes of CVI were hypoxic-ischemic encephalopathy in 142 (35.1%) patients, seizure associated with brain damage in 127 (31.3%) patients, neonatal hypoglycaemia

Table 1: Associated antenatal and perinatal risk factors in children with CVI

Risk Factors	Total Number (percent)	Vision Better Than and Equal to 20/200 Number (percent)	Vision Worse Than 20/200 Number (percent)	*P
Gestational diabetes mellitus	5 (1.2)	0 (0.0)	5 (100.0)	0.291
Gestational hypertension	9 (2.2)	1 (11.1)	8 (88.9)	0.585
Antenatal infection	10 (2.5)	2 (20.0)	8 (80)	0.869
Premature delivery <37 weeks	133 (37.6)	22 (16.5)	111 (83.5)	0.709
Birth weight				
Normal birth weight	154 (46.7)	26 (16.9)	128 (83.1)	0.659
Low birth weight <2.5 kg	137 (46.7)	24 (17.5)	113 (82.5)	
Very low birth weight <1.5 kg	39 (11.1)	9 (23.1)	30 (76.9)	
Delayed cry after birth	228 (67.5)	37 (16.2)	191 (83.8)	0.291
Perinatal seizure	184 (45.4)	26 (14.1)	158 (85.9)	0.063
Hypoglycemia	44 (10.9)	6 (13.6)	38 (86.4)	0.423
Perinatal infection	10 (2.5)	2 (20.0)	8 (80.0)	0.869
Foetal distress	8 (2.0)	2 (25.0)	6 (75.0)	0.604
Trauma	18 (4.4)	3 (16.7)	15 (83.3)	0.878

CVI=cerebral visual impairment. *P value was calculated using Chi-square test

Table 2: Etiology of children with CVI in our study population

Etiology	Total Number (percentage)	Vision Better Than and Equal to 20/200 Number (percent)	Vision Worse Than 20/200 Number (percent)	*P
Hypoxic ischemic encephalopathy	142 (35.1)	23 (16.2)	119 (83.8)	0.482
Seizure associated with brain damage	127 (31.3)	14 (11.1)	113 (88.9)	0.013
Neonatal hypoglycemia	44 (10.9)	6 (13.6)	38 (86.4)	0.423
Idiopathic	44 (10.9)	8 (18.2)	36 (81.8)	0.977
CNS structural malformation	39 (9.6)	7 (17.9)	32 (82.1)	0.990
Infection	13 (3.2)	3 (23.1)	10 (76.9)	0.630
Trauma	7 (1.7)	2 (28.6)	5 (71.4)	0.464
Hydrocephalus	5 (1.2)	1 (20.0)	4 (80.0)	0.908

CVI=cerebral visual impairment, CNS=central nervous system. *P value was calculated using Chi-square test

in 44 (10.9%) patients, structural neurological malformations in 39 (9.6%) patients, and infection in 13 (3.2%) patients. Seventy-two (17.8%) children in our study population had multiple etiologies.

Neurological findings

The associated neurological findings in our patients are summarized in Table 3. Most children with CVI had associated neurological abnormalities. The most common were seizure in 204 (50.4%) patients, cerebral palsy in 55 (13.6%) patients, and microcephaly in nine (2.2%) patients. Severe vision impairment (<20/200) was associated with seizure disorder ($P = 0.012$).

Refractive error

On the evaluation of cycloplegic refraction, significant refractive error was observed in 244 (63.2%) patients. Simple hyperopia was observed in 33 (8.5%) eyes and simple myopia was observed in 15 (3.8%) eyes. Simple myopic astigmatism was seen in 25 (6.4%) and simple hypermetropic astigmatism was present in 12 (3.1%) eyes. Compound myopic astigmatism and compound hypermetropic astigmatism were present in 56 (14.5%) eyes and 44 (11.4%) eyes had mixed astigmatism. Myopia ranged up to –21 dioptres, whereas hyperopia was found with a maximum of + 8 dioptres.

Ocular findings

The ocular abnormalities are summarized in Table 4. Associated ophthalmological findings were esotropia in 90 (22.2%) patients, exotropia in 154 (38%) patients, nystagmus in 135 (33.3%) patients, optic nerve atrophy in 105 (25.9%) patients, and temporal disc pallor in 78 (19.3%) patients. Severe vision impairment (<20/200) was associated with optic atrophy ($P = 0.003$).

Imaging findings

Of the 405 patients, neuro imaging findings were available in 321 (79.3%) patients. These were ischemic encephalopathic changes in 108 (26.7%) patients, periventricular leukomalacia in 84 (20.7%) patients, thinning of the corpus callosum in 28 (6.9%) patients, and global cortical gliosis in 25 (6.2%) patients.

When we compared vision with different variables by univariate analysis [Table 5], we found severe vision impairment (<20/200) to be significantly associated with optic atrophy (odds ratio [OR]: 2.9, 95% confidence interval [CI]: 1.4–6.0; $P = 0.003$) with an effect size of 0.149 ($P = 0.003$; Cramer's V test). Severe vision impairment (<20/200) was also significantly associated with seizure disorder (OR: 1.9, 95% CI: 1.2–3.3; $P = 0.012$), and the effect size was 0.131 ($P = 0.008$, Cramer's V test). However, there was no significant association

Table 3: Associated neurological deficits in children with CVI

Neurological Findings	Total Number (percentage)	Vision Better Than and Equal to 20/200 Number (percent)	Vision Worse Than 20/200 Number (percent)	*P
Seizure	204 (50.4)	27 (13.2)	177 (86.8)	0.012
Cerebral palsy	55 (13.6)	9 (16.4)	46 (83.6)	0.730
Microcephaly	9 (2.2)	0 (0.0)	9 (100.0)	0.155
Hemiparesis	7 (1.7)	2 (28.6)	5 (71.4)	0.464
Autism	7 (1.7)	5 (71.4)	2 (28.6)	<0.001
Hearing loss	5 (1.2)	0 (0.0)	5 (100.0)	0.291
Others (delayed speech, hypotonia, dystonia, hemiplegia, ADHD, mental retardation)	12 (2.96)	3 (25.0)	9 (75.0)	>0.05

ADHD=Attention deficit hyperactivity disorder, CVI=cerebral visual impairment. **P* value was calculated using Chi-square test

Table 4: Associated ocular findings in children with CVI

Ocular Findings	Total Number (percentage)	Vision Better Than and Equal to 20/200 Number (percent)	Vision Worse Than 20/200 Number (percent)	*P*
Refractive error	244 (63.2)	52 (21.3)	192 (78.7)	0.079
Strabismus				
Absent	161 (39.8)	25 (15.5)	136 (84.5)	0.566
Exotropia	154 (38)	30 (19.5)	124 (80.5)	
Esotropia	90 (22.2)	18 (20.0)	72 (80.0)	
Nystagmus	135 (33.3)	19 (14.1)	116 (85.9)	0.144
Cataract	20 (4.9)	4 (20.0)	16 (80.0)	0.814
Optic atrophy	105 (25.9)	9 (8.6)	96 (91.4)	0.003
Temporal disc pallor	78 (19.3)	15 (19.2)	63 (80.8)	0.758
Retinal disease	11 (2.7)	1 (9.1)	10 (90.9)	0.435
Others (microphthalmos, microcornea, etc)	6 (1.5)	2 (33.3)	4 (66.7)	>0.05

CVI=cerebral visual impairment. **P* value was calculated using Chi-square test

Table 5: Risk factors associated with severe visual impairment in children with CVI

Risk Factors	Odds Ratio	95% Confidence Interval	*P*
Univariate analysis (Chi-square test)			
Optic atrophy	2.9	1.4-6.0	0.003
Seizure	1.9	1.2-3.3	0.012
Birth asphyxia	1.4	0.8-2.4	0.291
Neonatal hypoglycemia	1.4	0.6-3.6	0.423
Autism	0.1	0.02-0.4	0.000
Infection	0.7	0.2-2.7	0.630
Cerebral_atrophy	1.0	0.4-2.4	0.990
Multivariate analysis (multiple logilistic regression)			
Optic atrophy	2.6	1.2-5.5	0.013
Siezure	2.1	1.2-3.7	0.007
Autism	0.1	0.0-0.5	0.006
Cerebral atrophy	0.3	0.1-1.3	0.097
Significant refractive error	0.7	0.4-1.2	0.173

CVI=cerebral visual impairment

with other variables, such as birth asphyxia (OR: 1.4, 95% CI: 0.8–2.4; *P* = 0.291), neonatal hypoglycemia (OR: 1.4, 95% CI: 0.6–3.6; *P* = 0.423), infection (OR: 0.7, 95% CI: 0.2–2.7; *P* = 0.630), and structural neurological malformations (OR: 1.0, 95% CI: 0.4–2.4; *P* = 0.990). Multivariate analysis [Table 5] also identified optic atrophy (OR: 2.6; 95% CI: 1.2–5.5; *P* = 0.013) and seizure disorder (OR: 2.1; 95% CI: 1.2–3.7; *P* = 0.007) to be independent risk factors for severe vision impairment.

Discussion

Our study comprises the largest cohort of patients with CVI in a developing country. It is a multicenter study that has been carried out at three tertiary eye care referral institutes in central and north India where around 50,000 children undergo eye examinations every year. This study population represents large parts of central and north India covering urban, semirural, and metropolitan regions.

The proportion of male to female was 1.6:1 in our study, which is similar to observations made in other studies.[14,17] As no sex predilection has been reported in CVI, this gender inequality may be because of the societal norms. It has been observed that ocular diseases in males are prioritized more than females in both adults and children in low- and middle-income countries, including India.[19,20] Our study reports a late age at presentation with a median age of 4 years in comparison to other studies where they reported a young age at presentation.[8,15,17] In the Pehere *et al*.[15] and Ganesh *et al*.[17] studies, median age at presentation was 3 years. The late age at presentation in our study can possibly be attributed to a late diagnosis and reporting of patients by pediatricians

and ophthalmologists in this region. In our study, 133 (37.6%) patients were prematurely born. Prematurity is not only a risk factor for retinopathy of prematurity but also a risk factor for CVI. In our study, one-third of the children gave a history of prematurity. Prematurely born babies should be screened at the early recommended age by trained ophthalmologists to rule out retinopathy of prematurity and these children should also be screened in their infancy to rule out CVI by checking their functional vision, refraction, ocular, and neurological findings. However, prematurity was not a risk factor for severe vision impairment in our study.

The most common etiology seen in our series was hypoxic-ischemic encephalopathy affecting 142 (35.1%) children. This result is similar to other reports by Huo *et al*.,[1] Khetpal *et al*.[16] and Pehere *et al*.[15] who found an etiology of perinatal hypoxia in 38 (22.35%), 35 (36%), and 50 (40.32%) patients, respectively. Early diagnosis and treatment of neonates at risk, the establishment of modern NICUs, and increased awareness in gynecologists, pediatricians, and general physicians can decrease the prevalence of hypoxic-ischemic encephalopathy and neuro developmental disabilities in these children. Compared to other western studies, in our study a low incidence of infection was observed. Infection was noted in 13 (3.2%) and hydrocephalus in five (1.2%) patients.[14,21,22] This could be attributed to the fact that children suffering from central nervous system infection have a high rate of mortality. Hence, delayed diagnosis and management of such patients is a common practice. The association between autism and vision impairment is complex.[23] In the present study, most children with autism did not have severe visual impairment. However, the number of patients with autism in the present study was few. Neonatal hypoglycemia was present in 44 (10.9%) patients. Ganesh *et al*.[17] also reported hypoglycemia in 19.31% patients. Counseling the parents by a neonatologist can help in reducing the prevalence of neonatal hypoglycemia by keeping babies warm and dry and offering early feeds.[24]

Ocular abnormalities were also commonly seen in our patients. Significant refractive error was present in 244 (63.2%) patients, which is similar to observations noted in other studies.[15,17] Myopia was the most common refractive error noted in other studies. However, in our study, the most common refractive error was astigmatism with compound hypermetropic astigmatism and compound myopic astigmatism in 56 (14.5%) eyes. Strabismus was observed in 244 (60%) patients and 154 (38%) had exotropia. This finding is similar to other studies.[15-17] In our patients, optic atrophy was noted in 105 (25.9%) patients and this finding is similar to other studies.[15-17]

Seizure disorders, cerebral palsy, and global developmental delay were quite common in our patients. Seizure disorders were significantly associated with severe vision impairment in our study. Also, optic atrophy was an independent risk factor. Despite their significance, the quantum of association was low. This may be because of patient selection where patients with severe neurological disorders were not brought for ophthalmic evaluation. Another factor may be the retrospective study design, which prevented the complete exploration of these risk factors. All the neurologic abnormalities in CVI patients are suggestive of associated diffuse brain damage along with damage to the visual pathways.

In our study, 135 (33.3%) patients had nystagmus. The incidence of nystagmus in our study is in concurrence with other studies.[1,25-27] In our study, 30 (6.97%) nystagmus patients had periventricular leukomalacia. Periventricular leukomalacia was equally associated with full-term (9.53%) and preterm (9.06%) babies. However, there was no significant association with other risk factors such as birth asphyxia, neonatal hypoglycemia, infection, structural neurological malformations, and trauma.

The limitations of our study are its retrospective design and the fact that a few important ocular findings such as accommodation, visual field, and contrast sensitivity data were not captured. These factors could also be grossly affected in CVI and should not be missed during evaluation. These findings are necessary for planning the rehabilitation of CVI children. However, our study was a multicenter study comprising the largest cohort of CVI patients from both urban and rural backgrounds, in which we emphasized the importance of the risk factors that cause visual impairment. A long-term follow-up study after rehabilitation for functional vision would provide more data to analyze the overall prognosis of these children.

Conclusion

In this large series of children with CVI from central and north India, male predominance, delayed presentation, significant refractive error, severe visual impairment, multiple ocular, and neurological abnormalities were some of the salient findings. A history of seizure disorder and presence of optic atrophy were significantly associated with poor vision. As many children with CVI have treatable ophthalmic conditions, early referrals by pediatrician and neurologist to an ophthalmologist are desirable to prevent permanent visual impairment. Similarly, ophthalmologists should take a neurology opinion in children diagnosed with CVI to evaluate and treat any underlying neurological abnormalities. Evaluation of children with neurological issues should be emphasized in ophthalmology residency programs for early detection of CVI and prompt referral to a pediatric ophthalmologist. Future prospective studies should explore causative risk factors of not only CVI but those associated with poor vision so as to develop treatment recommendations.

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Conflicts of interest

There are no conflicts of interest.

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Appendix 1: Vision gradation in children with CVI suggested by Huo *et al*[1]

Vision	Description
Level 1	Light perception only
Level 2	Occasional fixation on large objects, faces, or movement
Level 3	Occasional fixation on small objects (i.e., pennies or stickers) or reliable fixation on faces
Level 4	Reliable fixation on small objects; visual acuity 20/400 to 20/200
Level 5	Reliable visual acuity not better than 20/50 (both eyes open)

CVI=cerebral visual impairment

Appendix 2: Guidelines for refractive correction in infants and young children AAO guideline 2012[18]

Condition	Age <1 year	Age 1-2 years	Age 2-3 years
1. Isoametropia			
Myopia	–5.00 or more	–4.00 or more	–3.00 or more
Hypermetropia (no manifest deviation)	+6.00 or more	+5.00 or more	+4.50 or more
Hypermetropia with esotropia	+2.50 or more	+2.00 or more	+1.50 or more
Astigmatism	3.00 or more	2.50 or more	2.00 or more
2. Anisometropia (without strabismus)			
Myopia	–4.00 or more	–3.00 or more	–3.00 or more
Hypermetropia	+2.50 or more	+2.00 or more	+1.50 or more
Astigmatism	2.5 or more	2.00 or more	2.00 or more

AAO=American Academy of Ophthalmology

Appendix 3: Structural malformation

Risk Factors	Total Number (percent)	Vision Better Than and Equal to 20/200 Number (percent)	Vision Worse Than 20/200 Number (percent)	*P**
Corpus callosum	28 (8.7%)	5 (1.5)	23 (7.1)	0.981
Porencephaliccyst	4 (1.2)	0 (0.0)	4 (1.2)	0.346
Lissencephaly	5 (1.5)	0 (0.0)	5 (1.5)	0.291
Chiari malformation	2 (0.6)	1 (3.1)	1 (3.1)	0.238

**P* value calculated using Chi-square test