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Audio-Visual Assessment in Children with Cerebral Palsy

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KEYWORDS

CP, Auditory impairment, Language delay, Visual impairment

ABSTRACT:

Objectives: To find out the important co-morbidities such as Auditory & Visual impairment in children with Cerebral palsy and the common predisposing factors responsible for these salvageable maladies.

Methods: A cross-sectional observational study was carried out on consecutively enrolled children having clinical and imaging evidence of CP. Demographic, clinical, anthropometric, and justified investigations were done. Co-morbidities were assessed by Clinical psychologists, ENT surgeons and ophthalmologists, and Neurologists. The severity of type CP was compared with the type and degree of Audiovisual and language handicaps.

Results: Sixty-four under-five children with CP were analyzed for Demographic parameters, SEC, Anthropometry, Clinical types, and severity of CP. & Comorbidities. 62.5% of CP children did not have any perinatal high-risk factors. Of the CP children, 55% were delivered by Vaginal delivery; 68% were born Preterm, & 62% had delayed cries at birth.; 58% were Spastic Q-pelagic, 34% Spastic diplegic, 5% spastic hemiplegic, and 2 % each Hypotonic and extrapyramidal CPs. Among CP children 22 (34%) had hearing abnormality and speech delay was seen in 43 (67%), irrespective of hearing ability. 27/37 (73%) Spastic quadriplegic children had speech delay as compared to 14/22 (64%0 of diplegic CP children. Structural Eye abnormality was seen in 37/64 (58%) and Functional Eye abnormality in 43 (70%) of CP children.

Conclusions: The present study revealed a high association of comorbidities such as hearing impairment in 34 %, speech delay in 67 %, and visual impairment in as high as 70 % of Cerebral palsy children. The severity of CP was associated with increased association and severity of comorbidities. Early diagnosis & treatment of comorbidities can improve the quality of life.

1. Introduction

Cerebral Palsy is one of the common Non progressive, neurological entities caused by damage to the Brain by varied etio-pathological conditions occurring during the earliest rapid developing period. Moreover, evolving clinical presentations are limited to abnormality in tone, posture and execution of motor functions. This results in lifelong disability and compromised quality of life. The etiology of Brain damage could be varied, from developmental anomalies, Perinatal hypoxia, ischemia, metabolic abnormality such as hypoglycemia, Abnormal bilirubin, acidosis, electrolyte disturbances, infections, and Post natal acquired brain damage due to infections, shock, hemorrhages, stroke, trauma during early rapid developing phase of Brain. CP is being classified on 1) Anatomical, 2) Pathological, 3) Clinical subtypes. and 4) Severity wise based on Functional capacity of these patients.

Brain damage often affects not just the motor system but also adjacent brain regions responsible for hearing, language, vision, and cognition. Hearing

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impairment occurs in 10–20% of these children, with around 5% being deaf. Early diagnosis by 3 months and treatment by 6 months are crucial for learning and language development. Additionally, 40–60% of children with cerebral palsy have speech and language disorders, and about 25% are entirely nonverbal.CP also may be associated with recurrent seizures, Behavior problems, gastrointestinal disturbances, somatic growth abnormalities as well. All these comorbidities act as a "Fuel for Fire" in impeding the acquisition of Optimum Growth & Developmental potential of CP children; moreover if they are neglected and reared up in non-stimulating, non-promoting and ignorant environment.

Hence this study was planned to reveal the magnitude of association of Auditory, language and visual handicap with children having CP, so as to guide for early detection and anticipatory remedial measures to be taken so as to accomplish maximum developmental potential of these children.

2. Objectives

Materials & Methods: A cross-sectional study was conducted at a tertiary care hospital from October 2016 to September 2018, with approval from the Institutional Ethical Committee obtained prior to commencement**.** Written permission was obtained from the relatives of these CP children.

Inclusion Criteria: 1) Children with cerebral palsy disorder attending our tertiary care center

Exclusion Criteria: 1) Children with progressive neurological disorder, 2) Children with clinical symptoms and signs suggestive of lower motor neuron disease.

Study population: All consecutive children suffering from cerebral palsy who fulfilled the selection criteria have been included in the study.

Sample size: With a minimum of 10% CP children having these handicaps, confidence level of 95 %, margin of error 5%, and sample size was estimated to be 139. Target sample size was planned to 150, but due to drop outs from the study; and due to time constraints, single center study, we have analyzed 64 study subjects in our study.

Data collection: Data has been collected using a pretested and predesigned case record form. After thorough review of literature, the case record form was prepared and finalized. The various parameters such as bio-data, demographic, perinatal, developmental, Anthropometric data of these children were studied.

The detailed assessment of development, comorbidities such as seizures, visual, hearing and language development was recorded from the close relatives.

These CP children were classified, using the Swedish classification, into **Spastic** (Quadriplegic, Diplegic, Hemiplegic), **Ataxic,** and **Dyskinetic** (including athetosis, dystonia) and **Mixed.** The etiological classification was also used as **1) Congenital** (Prenatal, Perinatal, mixed) and **2) Acquired CP** if a child was normal at birth but developed clinical symptoms and signs of CP due to identifiable factors after birth.

Vision: assessment for structural and functional abnormality was done by senior ophthalmologist of the institute.

Hearing assessment was done by ENT surgeon well versed with pediatric patients and the requisite machines including BERA machine.

Privacy and confidentiality of the mother and her child was maintained throughout the study.

Statistical analysis: Data was collected, tabulated in MS-Excel work sheet, compiled, and analyzed using SPSS 20. The qualitative data was expressed using percentages. The quantitative data was expressed in terms of mean and standard deviation. The difference between the two proportions (bivariate) was analyzed using fisher's exact/ chi square test. The difference between two means has been analyzed using unpaired t test. For testing the difference between the means in more than 2 groups, we used two way analysis of variance test. The strength of association was expressed in terms of odds ratio. For assessing the trend of variables chi square for trend was applied. All analysis was one tailed for unidirectional and two tailed for bidirectional evaluation; and the significance level was set at 0.05.

3. Results

Maximum (67%) of CP children were in the age group of 13 to 60 months; with minimum and maximum age between 7 to 144 months. Male children were 57%; mean age was 29.21 ± 19.06 in male and 31.58 ± 28.06 in female CP children.

Out of total 59 Di-Plegic and Q-Plegics CP children; 27 (46%) were born by LSCS as against 32 (54%) by vaginal delivery. Odds ratio = 0.98; (95% C.I. 0.34 – 2.82); suggesting lower incidence of cerebral palsy with LSCS delivery; however statistically not significant.

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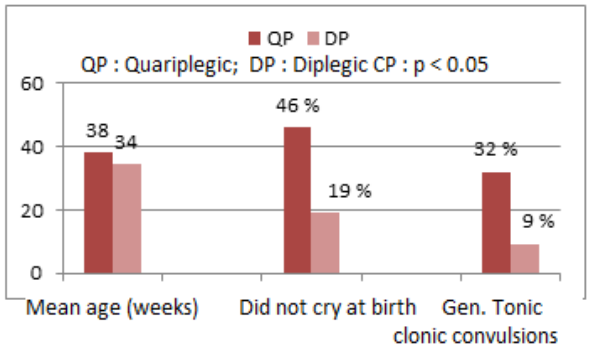
Mean gestational age was 34.32 ± 2.17 weeks in Di-Plegic CP as compared to 38.05 ± 0.57 weeks in Q-plegic CP children. The mean difference was of 3.73 weeks; $p < 0.05$.

Out of 37 Q-plegic children, 27 (46%) did not cry after birth as against 11 (19%) out of 22 Di-plegic children. Odds ratio was 2.7, 95% CI 0.89 - 8.16, suggesting increased risk of quadriplegia in babies who did not cry at birth. However Chi sq. was 3.17 & $p = 0.07$, Df = 1.

Involuntary movements seen in study subjects were, **:** Gen. Tonic Clonic 19 (30%), Focal seizure in 1. 12 /37 (32%), GTC + Choreoathetosis in 1, Choreoathetosis in 1,

Generalized tonic clonic seizures were seen in 12 /37 (32%) Spastic Q-plegic ; while in only 2/22 (9%) Spastic Di-plegic children

Graph-1: Mean age, Cry Status at birth & generalized tonic clonic Convulsions
In Quadriplegic vs. Diplegic Cerebral Palsy Children.



Developmental Delay was detected as follows; General motor 100%, Fine motor 95%, Language 78%, Psychosocial 93% of CP children.

Out of total 64 CP children, 62 (97%) were having spasticity. Quadriplegic CP in 37 (60%); Diplegic 22 (35%); and Hemiplegic 3 (5%); Floppy 1, Rigidity + Dystonic in 1 child was seen Weakness 62 (96.88%), Involuntary movements 25 (39.06%), Speech delay 48 (75%), . Feeding difficulty 30 (46.88 %] ,

Similarly **of Psychosocial delay was seen in 100% of Spastic quadriplegic CP; as compared to only 82 % in Spastic Diplegic CP; and was statistically significant p < 0.007.**

Antenatal history revealed risk factors in 24 /64 (38%) of CP children. PIH in 8 (12%), Oligohydramnios 6 (9%), Twin/triplets 4 (6%), GDM 2 (3%), IUGR 2 (3%), Threatened Abortions 2 (3%) were detected as Antenatal risk factors in these CP children.

Amongst Eye abnormalities seen in 37/ 64 (58%) CP children; Micropthalmia was seen in 2, Micro cornea in 1, Eye placement-Abnormality in 7, Eye slant-upward in 1, Coloboma in 1, Cataract in 4 (6%), Nystagmus in 1, Squint in 20 (31%) out of 37 children.

Of the total 64 CP children, 43 (70%) had structural and functional visual abnormalities. Amongst them children having, Vision-Abnormality was seen in 26 / 43 (40.63%), Myopia 5 (8.77%), Hypermeteropia 1 (1.75%), Severe visual tract dysfunction 3 (4.70%), Optic atrophy 8 (14.04).

Structural eye abnormality was seen in 16 (43%) of Spastic Q-plegic vs. 9 (41%) of Di-plegic CP children. Similarly Vision abnormality was seen 15 (40%) of Q-Plegic and 9 (41%) of Di-plegic CP children. Difference was not statistically significant (Chi sq. = 0.013, p = 0.91, df = 1).

Table -1: Distribution of study subjects based on Hearing deficit and Speech delay

Hearing n (%)	**Speech delay (n)**	**Speech Normal (n)**
Deficit 22 (34%)	**22 (100%)**	0
Normal 42 (66%)	**26 (62%)**	**16 (38%)**
Total 64	**48 (75%)**	**16 (25%)**
	Chi-Sq = 11.1; DF = 1, p = 0.001; Odds ratio 13.53.	

Among total 64 CP subjects, 22 (34%) had hearing deficit & 42 (66%) had normal hearing status. Similarly Speech delay was found in 22 (100%) of hearing deficit subjects and in 26 (62 %) of subjects having normal hearing. This difference was statistically significant; Chi sq. = 11.1; $p < 0.001$; Odds ratio 13.53.

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Table -2: Distribution of Hearing Abnormality in different types of CP

CP Types (n)	Hearing Abnormality +ve	Normal Hearing	
	n (%)	n (%)	P
Spastic Q-plegic (37)	**13 (**35)	**24(**65)	**Chi-Sq=0.99, df = 1, p=0.459 Odds ratio 1.84.**
Spastic Di-plegic (22)	**5 (**22)	**17 (**78)	
Spastic Hemi-plegic (3)	**2 (**66)	**1 (**34)	
Hypotonic (1)	**1 (**100)	**0**	
Extra pyramidal (1)	**1 (**100)	**0**	
Total (64)	**22 (**34)	**42 (**66)	

13 out of 35 (35%) Spastic quadriplegic CP subjects as compared to 5/22 (22%) Spastic di-plegic subjects had hearing abnormality. The difference is not statistically significant p > 0.05; Odds ratio between Normal hearing vs Abnormal hearing 1.84 in these CP children.

Table -3: Speech status in different types of Cerebral palsy children

CP Types (n)	Speech Delay	Normal speech	
	n (%)	n (%)	P
Spastic Q-plegic (37)	**27 (73)**	**10 (**27)	**Chi-Sq = 0.56, df = 1, p = 0.45 Odds ratio= 1.54**
Spastic Di-plegic (22)	**14 (64)**	8 (36)	
Spastic Hemi-plegic (3)	**2 (**66)	1 (34)	
Rigidity (1)	**1 (100)**	0	
Hypotonic (1)	**1 (**100)	0	
Total (64)	46 (71)	19 (29)	

Speech delay was more in 27/37 (73%) of Spastic quadriplegic children as compared to that in 14/22 (64 %) of Spastic di-plegic subjects. The difference is not statistically significant; chi square = 0.56; p = 0.45.; However Odds ratio between spastic quadriplegic & Spastic diplegic children's speech delay is 1.5;

Graph - 2: Speech status (%age) in Q-plegic and Di-plegic CP children

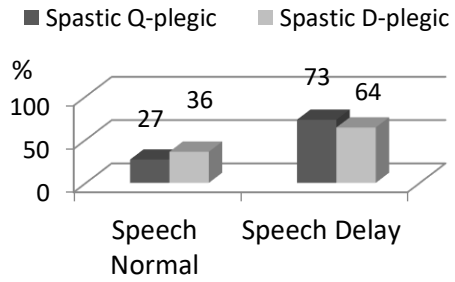

Table - 4: Distribution of study subjects based on BERA assessment

BERA	Frequency	Percentage
1. Hearing loss		
Present	27	42.19
Absent	37	57.81
2. Laterality		
Bilateral	25	92.59
Right side	1	3.70
Left side	1	3.70
3. Severity		
Minor	13	48.15
Moderate	7	25.93
Severe	7	25.39

On BERA testing Hearing loss was seen in 27 (42 %) of CP children; Majority 25/27 (92%) had bilateral hearing loss. Minor hearing loss was seen in 13 (48 %), moderate and severe hearing loss was seen in each 7/27 (26%) of CP children

Table -5: Association of severity of hearing loss in different types of CP subjects

Severty of hearing loss	Quadriplegia	Di-plegia	Hemi plegia	Hypotonia	Rigidity	All n (%)
Normal 10-20 dB)	16	14	2	0	0	32 (50%)

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Mild + Mod. (30-70 dB)	13	6	1	0	1	21 (32%)
Severe to Profound (70-120 dB)	8	2	0	1	0	11 (17%)
Total	37	22	3	1	1	64 (100%)
Chi Sq. = 10.233; df = 8, P < 0.05;						

Quadriplegic CP patients had statistically significant more severe degree of hearing loss than that of diplegic and other groups of CP children.

Table - 6: Association of severity of hearing loss and GMFCS scores (n=27)

Severity of hearing loss	**GMFCS**			
	II	**III**	**IV**	**Total**
	n (%)	**n (%)**	**n (%)**	**n (%)**
Mild	1 (8)	5 (38)	7 (54)	13 ***(48)***
Moderate	0	3 (43)	4 (57)	7 ***(26)***
Severe	0	4 (57)	3 (43)	7 ***(26)***
Total	1 (4)	12 (44)	14 (52)	27 ***(100)***

As shown in the table no significant association of GMFCS score was observed with severity of hearing loss.

4. Discussion

Cerebral palsy is a common group of Non-progressive Central Motor disorder of movement and posture, originating early in the life of the child due to various risk factors resulting in insult to the developing brain. Apart from motor deficits, sensory impairments like hearing and vision abnormality are equally responsible in catalyzing the severe morbidity of the Handicap and deteriorating the quality of life.

Mean age of total CP children was 30.3mths ± 23.5; while those of male & female children was 29.21 ± 19.06, & 31.58 ± 28.06, p = 0.69. Indian study (2002) reported by Pratibha D. Singhi et.al[1] from PGIMER, shown the Age at the time of presentation ranging from 2 mths. to 16 years, with mean age of 36.4 ± 31.9 mths. Similar observations made by Lanzi G, Fazzi E, Ugetti C[3] et al (mean age of 30.68 ± 2.52 months), Costa MF et al included (28 ± 13 months). The present study and all the hereto mentioned studies indicate that the maximum cases of CP are detected at toddler and preschool age; by this time critical period for development of brain is already over and hence fruitful modalities of management have minimal perceptible effect on the targeted goal of therapy.

In the present study; Spastic CP was seen in 97%; (Quadri-plegic 60%, Di-plegic 35%, Hemiplegic 5%; and only one child each was seen having Floppy and extrapyramidal (Rigidity + Dystonia) CP. Similar observations made by Pratibha D. Singhi et.al[1] in her cohort study of 1000 CP subjects., (Quadriplegia in 61 %, Diplegia in 22 %, Hemiplegia in 17%, Dyskinetic/ athetoid in 8.4 %, and hypotonic / ataxic in 7.7 %.). Katoch S et al[2] reported that 91.5% had spastic type and rest were either athetoid, mixed type or extra pyramidal types Park MJ et al inferred that 44.8% were Diplegic, 25.7% were hemiplegic, 16.2% were tetraplegic and 12.3% were unclassified in their study. They had included 105 cerebral palsy subjects in their cohort.. Similar observations made by Minocha P et al (Spastic CP 84.4%), and Fazzi E et al[3], Gedam DS et al (90% had spastic CP; and 45% had quadriplegic type), Preponderance of spastic CP cases in our study is similar to that reported by others. However, distribution of the clinical subtypes of spastic cases is different from that reported by western studies. In western studies Spastic diplegia is the commonest variety, but in our and other studies from developing world, the commonest variety is spastic quadriplegia; due to obvious reasons of surviving severe premature and VLBW babies in developed countries though with residual morbidity such as Spastic Diplegic CP.

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Various likely risk factors in our scenario studied were analyzed and found that the Majority 67% of mothers of CP children did not have any significant antenatal high risk factors detected on ANC checkup. Of the high risk factors 12 % were PIH, 9% were Oligohydramnios, 6 % were Twins / Triplets, 3% each were seen as Gestational Diabetes in Mother (GDM), IUGR & Threatened abortion. Majority of them delivered by normal vaginal delivery (54.69%) and were preterm (38%) and 62% had delayed cry at birth. The risk of development of spastic quadriplegic CP was 2.7 times higher in asphyxiated children, as compared to those who cried immediately after birth. Contrary to expectation, the present study also highlighted that the CP frequency was significantly more in Full term, and babies weighing more than 2.5 kg than in Preterm and Low birth weight babies. The probable reason being the Preterm and LBW babies with perinatal complications did not survive for lack of state of the art Perinatal Infrastructure in many of the Rural as well as Urban health facilities.

A study by Rajesh R et al. found that natal causes were the leading etiological factor for cerebral palsy, accounting for 52% of cases. Unknown causes were identified in 14.67% of cases, making it the second most common category. Bangash AS et al. found a positive family history of cerebral palsy in 4 cases (20%). Among mothers of children with cerebral palsy, 14 (70%) had received antenatal care, but only 5 (25%) children were born in hospitals or maternity centers, with the rest being delivered at home or by traditional birth attendants (Dais). Additionally, the study identified several risk factors based on interviews with these mothers, categorizing them into prenatal, natal, and postnatal factors. The most common risk factors included home and Dai-assisted deliveries (75%), consanguinity (50%), neonatal seizures (50%), infections during pregnancy (40%), and lack of antenatal care (30%).

Minocha P et al. found that 47.7% of mothers of children with cerebral palsy were primigravida, and 17.7% had anemia during pregnancy. Other prenatal risk factors included antepartum hemorrhage (5.5%), pregnancy-induced hypertension (4.4%), and infections (3.3%). Among the natal factors, asphyxia was the leading cause, accounting for 45.5% of cases. Additionally, 41.1% of children with cerebral palsy were delivered via induced delivery, and 13.3% via assisted delivery. Small for gestational age (25.5%) and prematurity (17.7%) were also significant risk factors.

Oztturk AT et al. identified the following etiological factors among their patients: asphyxia in 60.8% (118 patients), prematurity in 54.1% (105 patients), early membrane rupture in 2.6% (5 patients), intrauterine infection in 2.1% (4 patients), imminent abortus in 1.5% (3 patients), kernicterus in 1.0% (2 patients), and febrile convulsion in 1.0% (2 patients). Additionally, 4.6% (9 patients) exhibited varying degrees of cortical dysplasia. A single etiological factor was identified in 74.2% (144 patients) of the study population, while 25.8% (50 patients) had two or more factors contributing to the development of cerebral palsy.

Jain V et al found that 16 (5.2%) mothers were hypertensive whereas 25 (8.1%) were anemic even before pregnancy. This problem was aggravated during pregnancy, and we found anemia in 53 (17.3%) and hypertension in 26 (8.5%) mothers during pregnancy. Out of the total, 22 (7.2%) gave a history of bleeding and 25 (8.2%) had a history of either oligo-hydroamnios or leakage of liquor amnii during pregnancy. Further, 33 (10.7%) mothers gave a history *of spontaneous abortion. Totally 19 (6.2%) mothers also gave a history of infections or fever during pregnancy. History of delayed cry at birth was found in 208 (67.8%) children, indicating a hypoxic condition.*

Present study also revealed psychosocial delay in all type of cerebral palsy. Among the spastic quadriplegic 100% cases had the delay, in diplegic cases 82% of the cases had delay and in hypotonic, hemiplegic and extra pyramidal 100% cases had psychosocial delay. The risk of Psychosocial delay was significantly more in Quadriplegic CP as compared Minocha to that in diplegic CP; $\chi^2 = 7.2$, $p = 0.007$.

We further did not find any association between the speech delay and type of cerebral palsy.

Minocha P et al inferred that intellectual disability (47.7%) followed by epilepsy (41.6%) was the most common comorbidity. Speech delay, hearing defects, and visual impairment were present in 27.7%, 10%, and 10% cases, respectively. Zhang JY et al inferred that of the 297 children who had communication impairment, 96 children were completely unable to communicate verbally (32.3%), 195 had some verbal communication capabilities (65.7%), and 6 were left unspecified (2%). Further, among children who had a communication impairment, those unable to verbally communicate were more likely to also have cognitive impairment, cortical visual impairment, sensorineural auditory impairment,

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and feeding impairment. Gedam DS et al reported that delayed milestone is most common presenting symptoms in 98% cases, followed by feeding difficulty, convulsion and speech defect in 38%, 36% and 34% cases. Pratibha D. Singhi et.al1 reported Mental retardation in 64.6% of Quadriplegic, 61.7% of Diplegic, 61 % of Hemiplegic,75% of Hypotonic and 100% Athetoid Cerebral palsy subjects..

In the current study, **Amongst Eye abnormalities seen in 37/ 64 (58%) CP children;** Micropthalmia was seen in 2, Micro cornea in 1, Eye placement-Abnormality in 7, Eye slant-upward in 1, Coloboma in 1, Cataract in 4 (6%), Nystagmus in 1, Squint in 20 (31%) out of 37 children.

Of the total 64 CP children, 43 (70%) had structural and functional visual abnormalities. Amongst them children having, Vision-Abnormality was seen in 26 / 43 (40.63%), Myopia in 5 (8.77%), Hypermeteropia in 1 (1.75%), Severe visual tract dysfunction 3 (4.70%), Optic atrophy in 8 (14.04).

Vision abnormality was present in 40% in quadriplegic cases, 41% in Diplegic cases, 33% in hemiplegic cases and 100% in hypotonic and extrapyramidal cases and in total it was present in 35.94% cases. Amongst the vision abnormality, Central/Cortical was in 34 %, Optic atrophy in 31 %, Myopia in 19%, severe visual tract dysfunction in 12 % and Hypermetropia in 4 % of CP subjects.
Pratibha D. Singhi et.al[1] reported Refractive errors in 48%, Squint in 24.5 % , Myopia in % Optic atrophy in 10.8% , Cataract in 3.9% and Nystagmus in 4 %, Chorioretinitis and cortical blindness in 2-4 % of CP subjects.

Katoch S et al. reported that 13.5% of children had myopic refractive error, while 20% had hypermetropic refractive error. Normal pupillary reactions were observed in 94% (188 patients). Eleven patients with diffuse disc pallor exhibited sluggish pupillary reactions, and one patient with congenital glaucoma had dilated, non-reactive pupils. Developmental cataract was found in 2.5% (five patients). Normal fundi were observed in 81% (62 patients). Temporal disc pallor was noted in 11% (22 patients), and diffuse disc pallor in 5.5% (11 patients). One patient showed advanced glaucomatous cupping in both eyes, another had a myopic fundus, and one patient each exhibited macular choroiditis, fundal coloboma, and a salt-and-pepper fundus associated with deafness. Three out of five patients with cataract underwent surgery. Horizontal, jerky nystagmus was identified in 5.5% (11 patients); within this group, one had bilateral cataract, two had disc pallor, one had fundal coloboma, and one had more than -5D myopia. Elevated intraocular pressure (IOP) over 21 mmHg was found in three patients, with one showing advanced glaucomatous cupping and two having a cup-to-disc ratio of 0.5:1. Horizontal corneal diameters greater than 13 mm were observed in 4% (eight patients), with only one having congenital glaucoma; the remaining had normal IOP and fundi. A horizontal corneal diameter less than 10 mm was found in one patient, who also had a fundal coloboma with nystagmus.

Park MJ et al. discovered that strabismus affected 74 patients (70.5%), with 46 patients having exodeviation, 27 with esodeviation, and 1 with dissociated vertical deviation. Significant refractive errors were identified in 56 patients (53.3%), including 19 with hyperopia $\geq +1.50$ diopters (average $+2.86 \pm 1.30$ diopters), seven with myopia ≥ -1.50 diopters (average -3.78 ± 2.24 diopters), 21 with hyperopic astigmatism $\geq +1.50$ diopters (average $+2.46 \pm 1.90$ diopters), and nine with myopic astigmatism ≥ -1.50 diopters (average -1.68 ± 0.91 diopters). Anisometropia ≥ 1.00 diopter was found in 24 patients (average 2.36 ± 2.49 diopters). Six patients showed corneal erosions, four had papillary hypertrophy, one had mild lens opacity, and no abnormalities were observed in the remaining patients during slit lamp examination.

Reid SM et al. conducted a systematic review and meta-analysis indicating that visual loss occurs in 2 to 12% of cerebral palsy cases. Woo SJ et al. found ocular abnormalities in 89% of 78 patients studied, with refractive errors in 53 patients (60%) and strabismus (exotropia, esotropia, hypertropia) in 52 patients (59%). Posterior segment abnormalities were noted in 22% of patients, while corneal and lens abnormalities were found in 9%, and epiblepharon in 6%. Da Cunha Matta AP et al. reported that 38 patients had strabismus, including 25 with convergent (esotropia) and 11 with divergent (exotropia).

Similar findings were reported by Fazzi et al., Rajesh R et al., and Kaur G et al., who collectively found ocular morbidities in 69.3% of cases, with hypermetropia in 26.5%, myopia in 6.1%, and astigmatism in 4.1%. Venkateswaran S et al. identified cortical visual blindness in ten patients, optic atrophy in six, and severe retinal disease from cytomegalovirus (CMV) in one patient with substantial visual impairment.

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All these studies indicate that majority of CP subjects has visual co-morbidities. Early detection and therapy thereof is warranted for correction of visual abnormality and achievement of optimum global development.

Hearing deficit and its impact on speech delay was assessed by clinical and BERA study. BERA study was abnormal in 50 % of CP patients; and more severe 8/37 (21%) in Qudriplegic CP as compared to 2/22 (9%) in Diplegic CP children. It was observed that out of 22 subjects having hearing deficit, 100% had Speech delay, as compared to 42 children having normal hearing, 62% only had speech delay in these CP children. The difference was statistically significant χ^2 = 11.1, P < 0.01; and Odds ratio of 11.53. Similarly it was found that 35% Spastic Quadriplegic & 22% Spastic Diplegic CP children had hearing abnormality, The Odds ratio was 1.84 ; though the difference was not statistically significant (χ^2 = 0.54, p = 0.45). Speech delay was seen in 73% Spastic Quadriplegic & 64% Spastic Diplegic CP children. The Odds ratio was 1.54; though the difference was not statistically significant (χ^2 = 0.56, p = 0.45).Though the present study lack statistical significance due to small sample size, it is quite obvious from this study that hearing deficit has close relation with speech delay. The chances of severe hearing and language impairment was seen proportional to the severity and extent of spasticity (more in quadriplegic as compared to that in diplegic CP).

Weir FW[7] and colleagues reported 39% of their study subjects had hearing loss and among them 65.7% were bilateral hearing loss.

Pratibha D. Singhi et.al[1] reported Hearing disorder in 14 % and Speech disorder in 7.8 % of CP subjects. Their study reported Hearing defect in 13.6 % Quadriplegic, 10.4% of Diplegic, 10.1% of Hemiplegic,15.6% of Hypotonic and 33.3% Athetoid Cerebral palsy subjects. There was no reported significant difference (ranging from 7.3 % in quadriplegic to 10.7 % in Athetoid) in speech defects in different types of CP subjects.

Venkateswaran S et al. reported that hearing abnormalities were present in 40.2% (37 out of 86) of children, epilepsy developed in 46.7% (43 out of 92) of children, and 46% (40 out of 87) had feeding difficulties. Additionally, 40.2% (37 out of 86) of the tested patients had some degree of hearing loss. Complete deafness was observed in 5.8% (five children), three of whom had a birth weight of less than 1000 grams. Hearing loss was noted in seven of the eleven patients with an infectious etiology (OR 2.6, 95% CI 0.7–9.7; p=0.150). The patient with kernicterus as the etiology had moderate hearing loss.

Reid SM[9] et al conducted a systemic review and meta-analysis of the prevalence of hearing loss and visual loss in cerebral palsy patients and found that hearing loss ranges from 4 to 13% of cases.

5. CONCLUSIONS

Cerebral palsy is a heterogeneous group of Non progressive Central motor disorder of Movement, Posture and Coordination, originating from prenatal, perinatal and early post-natal injury to developing brain. Non progressive varied etiology, comorbidities, and clinical course, are the hallmark of this life-long handicap.

Clinical spectrum of CP in children in developing countries including India, differ from that in developed countries. Associated problems are different, majority have Mental retardation, Visual and Hearing – speech deficit, and seizures. Comprehensive assessment and early is management is mandatory during the placid stage of brain development to achieve encouraging long term results.

The most frequently detected type of cerebral palsy was severe spastic (hypertonic) quadriplegia, suggesting a high incidence of severe brain injury among neonates in our region. This aligns with our findings that perinatal asphyxia and postnatal encephalitis/encephalopathy are the primary risk factors for cerebral palsy.

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The present study has highlighted the importance of early detection and prompt management of high risk factors during antenatal period such as Pregnancy induced hypertension, Oligohydramnios, Gestational diabetes in mother, and threatened abortion.

In addition, numerous Co-morbidities were observed among these in the present study. It highlighted the prevalence of visual abnormality in as high as 40 & 41% of quadriplegic & diplegic CP children respectively. Similarly Hearing impairment and speech delay was seen with highest percentage in 35 % and 73 % of spastic quadriplegic subjects respectively. BERA study was abnormal in 50 % of CP patients.

Prevention of Perinatal risk factors such as prematurity, birth asphyxia, severe unconjugated neonatal jaundice, and other neuro affective metabolic disorders can prevent cerebral palsy as well as its comorbidities such as audio visual & speech disorders! Early detection Audio-visual defects can have salvaging effect on quality of life

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