International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 13, No. 7, July 2024 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.7.2024.24

ORIGINAL RESEARCH

Correlation Between Clinical Features and Neuroimaging of Brain in Cerebral Palsy Children

[1]Singh D.K., [2]Gupt S. K., [3]Dhiman N., [4]Kumar B.K., [5]Kanaujia V.

[1]Assistant Professor, Department of Physical Medicine and Rehabilitation, Uttar Pradesh University of Medical Sciences, Saifai, Etawah, India
[2]Assistant Professor, Department of Physical Medicine and Rehabilitation, King George's Medical University, Lucknow, Uttar Pradesh, India
[3]Fellow Pain Medicine, Department of Anaesthesiology, Pain Medicine and Critical Care, AIIMS, New Delhi, India
[4]Assistant Professor, Department of Physical Medicine and Rehabilitation, AIIMS, Mangalagiri, Guntur, Andhra Pradesh, India
[5]Assistant Professor, Department of Physical Medicine and Rehabilitation, Uttar Pradesh University of Medical Sciences, Saifai, Etawah, India

Corresponding Author
Kumar B.K.
Assistant Professor, Department of Physical Medicine and Rehabilitation, AIIMS, Mangalagiri, Guntur, Andhra Pradesh, India
Email: kyron143@gmail.com

Received Date: 20 May, 2024 Acceptance Date: 25 June, 2024

ABSTRACT

Introduction: Cerebral Palsy (CP) is the most common disorder causing disability in children resulting from injury to the developing brain. Neurological imaging techniques like magnetic resonance imaging (MRI) facilitates identification of location and extent of brain injury in CP and helps to correlate with motor function, type of cerebral palsy and functional outcome. **Aim:** To correlate the brain MRI abnormality with clinical feature. **Methodology:** Cross sectional study was done on cerebral palsy diagnosed children aged 2-12 years, MRI of brain was performed to determine the type of lesion and assigned a grade based on the scoring system by expert radiologist. **Results:** In children with Cerebral Palsy abnormal neuroanatomical findings was found in 46 of the 50 cases with help of MRI. Type E (Periventricular leukomalacia) brain lesion was most common seen in all types of CP. Type D (Enlargement of lateral ventricle), type H (Thin corpus callosum) and type G (Border zone infarction) were next most common. Eleven (100%) patients in GMFCS level 1 had brain MRI grade 1(normal) (p=0.003). In GMFCS level 2 and 3 predominant MRI grade was 1. In GMFCS 4 and 5 predominant MRI grade was 2 (mild) (p=0.013) and (p=0.147) respectively. **Conclusion:** Majority of CP cases have neuroimaging finding on MRI and very few have no MRI finding. There was correlation between motor function and type of CP with severity of lesion in MRI and type of brain lesion.

Keywords: Cerebral Palsy, Neuroimaging, Periventricular Leukomalacia, Spasticity

INTRODUCTION

Cerebral palsy (CP) is the most common cause of disability in children and has a high prevalence of 2 to 2.5 per 1,000 live births.[1] CP is classified based on tone pattern as spastic, dyskinetic, hypotonic or mixed and based on limb involvement as diplegia (lower limbs affected more than upper limbs), hemiplegia (upper limb frequently more affected than lower limb), and quadriplegia.[2] Most children with CP face limitation in ambulation which hinders participation in physical, recreational, and social activities thus considerably affecting the quality of life. The ambulatory potential varies significantly among different subtypes of CP. Thus, achieving ambulation is a key goal in the rehabilitation management of children with CP. [3]

Neuroimaging is an important aspect in the diagnostic evaluation of CP and helps in providing understanding into the etiology, management and neurodevelopmental outcome.[4] Neuroimaging through the use of an MRI or computed tomography detects abnormal neuroradiological findings in 80 to 83% of

Online ISSN: 2250-3137
Print ISSN: 2977-0122
DOI: 10.69605/ijlbpr_13.7.2024.24

children with CP.[5] MRI has been shown to have a high yield in identifying abnormalities in children with CP.[6,7] Most common causes of CP are structural brain lesions on neuroimaging [8-12] especially prematurity-related injuries, and malformations of brain development [3,13,14]. Significant associations have been found between cerebral white matter and gray matter abnormalities on MRI and neurodevelopmental impairment.[15] Strong recommendations have been established for the use of neuroimaging at an early stage in children with a suspected diagnosis of CP.[16]

Improved neuroimaging has provided an opportunity to relate various CP types to the localization and type of brain lesion. This assists in evaluating the severity of damage and early identification of factors that could help in predicting long term functional outcome, prognostication and planning of realistic goals for the rehabilitation management.

MATERIALS AND METHODS

This was a cross-sectional observational study conducted at a tertiary care hospital in northern India after taking approval from Institutional Ethical Committee. The diagnosis of CP was made according to the standard clinical definition stated by Rosenbaum et al.[17] Patients were screened and detailed history were recorded following which physical and neurological examination was carried out by a physiatrist. Cases were enrolled in the study after satisfying the inclusion and exclusion criteria. Diagnosed cases of CP in the age group of 2-12 years were included whereas children with congenital anomalies of upper and lower limb and those with contraindication for MRI were excluded from this study. Informed consent was taken from parents or guardians and selected patients were advised neuroimaging (MRI). Magnetic Resonance Imaging (MRI) using a 1.5 T Philips Intera Achieva MR scanner, and obtained T1weighted, T2 weighted, and T2 Fluid Attenuation Inversion Recovery (FLAIR) sequences in the axial plane. Additionally, T2 weighted imaging was performed in the sagittal and coronal planes. The expert radiologist conducted an MRI analysis to determine the type of lesion and assigned a grade based on the scoring system.

The lesions were described as Hypomyelination (A), Cerebral atrophy (B), Malformation (C), Enlargement of lateral ventricle (D), Periventricular leukomalacia (E), Porencephaly (F), Border zone infarction (G), Thin corpus callosum (H) and Cerebellar atrophy (I).

A three-grade scoring system on MRI was used [12] of sub-components including Size of lateral ventricles, White Matter (WM) abnormal signal intensity, WM reduction, Cysts, Size of subarachnoid space, Corpus callosum and Cortical grey matter with score 3 indicating most severe abnormalities. [Grade1: 7-11 (normal), grade2: 12-16 (Mild) and grade3: 17-21 (Severe)].

Sample size calculation: Based on previous study, prevalence of CP was 2 to 2.5 per 1000 live birth.[1] Taking this value as reference, total sample size taken was 50 after reducing the margin of error.

The data were entered in MS EXCEL spreadsheet and analysis was done using Statistical Package for Social Sciences (SPSS) version 21.0. Quantitative variables were compared using unpaired t-test/Mann-Whitney Test. Qualitative variables were correlated using Chi-Square test /Fisher 's exact test. Pearson correlation coefficient/Spearsman rank correlation coefficient (for non-parametric data) were used to correlate quantitative variables with each other. Linear regression analysis was performed to measure the relationship between MRI-Score and other variables.

RESULTS

The mean age of patients in this study was 5.43±2.58 years with male predominance (64%). Maximum patients were in the age group of 2-5 years (60%). Based on the tone pattern 90% of the cases were of spastic CP and based on topographical distribution maximum had diplegia (Table 1).

Out of 50 patients most common type of brain lesion was periventricular leukomalacia (E) in 45 cases whereas 4 cases enrolled in the study had no brain lesion on MRI. Enlargement of lateral ventricle (D), periventricular leukomalacia (E), border zone infarction (G) and thin corpus callosum (H) were the findings observed on MRI most commonly in majority of the patients with spastic and mixed type of CP (Table 2). Out of 50 patients type D (Enlargement of lateral ventricle), E (Periventricular leukomalacia), G (Border zone infarction) and H (Thin corpus callosum) brain lesion found in all topographic type of cerebral palsy patients of which type E lesion was most common. Type A (Hypomyelination) and C (Malformation) brain lesion were not found in any type of CP. No brain lesion was observed in 4 (15.38%) cases of diplegia (Table 3).

On correlating MRI grading with the type of CP (topographical) all quadriplegic patients and a few patients with diplegia (19%) had Grade 2 on MRI and all patients with hemiplegia had Garde 1 on MRI. Patients with mixed, dyskinetic and spastic quadriplegia CP had grade 2 MRI findings and those with spastic hemiplegia and diplegia had grade 1 findings (Table 4).

Table 1: Type of cerebral palsy based on tone pattern and topographical distribution.

Type of cerebral palsy (tone pattern)		
Spastic	45	90%
Mixed	4	8%
Dyskinetic	1	2%
Topographical distribution		

DOI: 10.69605/ijlbpr_13.7.2024.24

Diplegia	26	52%
Quadriplegia	14	28%
Hemiplegia	10	20%

Table 2: Correlation of type of brain lesion with type of cerebral palsy (tone pattern)

Type of brain lesion	Type of CP (tone pattern)			p value
	Dyskinetic (1)	Mixed (4)	Spastic (45)	
A	0	0	0	-
B	1	1	2	0.001
C	0	0	0	-
D	1	4	29	0.271
E	1	4	40	0.734
F	1	1	4	0.015
G	1	4	19	0.049
H	1	3	27	0.614
I	0	1	0	0.003
No Lesion	0	0	4	0.785

A: Hypomyelination, B: Cerebral atrophy, C: Malformation,
D: Enlargement of lateral ventricle E: Periventricular leukomalacia,
F: Porencephaly), G: Border zone infarction, H: Thin corpus callosum
I: Cerebellar atrophy

Table 3: Correlation of type of brain lesion with type of cerebral palsy (topographical)

Type of brain lesion	Type of CP (topographical)			p value
	Diplegia (26)	Hemiplegia (10)	Quadriplegia (14)	
A	0	0	0	-
B	0	1 (10%)	3 (21.43%)	0.084
C	0	0	0	-
D	16 (61.54%)	7 (70%)	11 (78.57%)	0.726
E	21 (80.77%)	10 (100%)	14 (100.00%)	0.163
F	1 (3.85%)	0	5 (35.71%)	0.015
G	9 (34.62%)	4 (40%)	11 (78.57%)	0.054
H	14 (53.85%)	4 (40%)	13 (92.86%)	0.028
I	0	0	1 (7.14%)	0.453
No lesion	4 (15.38%)	0	0	0.260

A: Hypomyelination, B: Cerebral atrophy, C: Malformation,
D: Enlargement of lateral ventricle E: Periventricular leukomalacia,
F: Porencephaly), G: Border zone infarction, H: Thin corpus callosum
I: Cerebellar atrophy

Table 4: Correlation of Type of CP (tone pattern and topographical) with MRI grading.

Type of CP (tone pattern)	MRI Grade 1	MRI Grade 2	
Dyskinetic (1)	0	1	0.011
Mixed (4)	0	4	
Spastic (45)	31	14	
Type of CP (topographical)			
Diplegia (26)	21 (80%)	5 (19%)	<0.0001
Hemiplegia (10)	10 (100%)	0 (0%)	
Quadriplegia (14)	0 (0%)	14 (100%)	

DISCUSSION

A lifelong condition known as cerebral palsy is primarily identified by a motor disorder that results in physical impairment during human development, particularly affecting body movement and posture. Age distribution of patients in study population was in range of 2-12 years, with mean age of 5.43 (±2.58) years. Both the groups were comparable in age distribution of patients. The age group of 2-5 years had the highest number of individuals. In the study population, the observed sex ratio was 1.78:1 (M: F), indicating a higher number of males, which was similar to the findings of Pharaoh et al.[18] In the present study, diplegic 26 (52%) cases was most common, quadriplegic 14 (28%) cases were next and hemiplegic 10 (20%) cases were least common. Similar observation was made in studies by Pharoah et al.[18] and Erkin et al.[19] Spastic CP in 45 (90%) cases

Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.7.2024.24

was most common, mixed was with 4 (8%) cases. Only 1(2%) case was of dyskinetic type.

In our study we observed almost even distribution of patients in GMFCS level 1, 2, 3 and 4, whereas other studies [20,21]had maximum patients in GMFCS level 1.

Type E (Periventricular leukomalacia) brain lesion was most common seen in all types of CP (100% of dyskinetic, 100% of mixed and 88.89% of spastic). Type D (Enlargement of lateral ventricle), type H (Thin corpus callosum) and type G (Border zone infarction) were next most common and were present in all type of CP. Type I (Cerebellar atrophy) brain lesion was seen in only 1 (25%) case of mixed type of CP (p=0.003). Type B (Cerebral atrophy) brain lesion was seen in 100% of dyskinetic, 25% of mixed and 4.44% of spastic CP (p=0.001). Type A (Hypomyelination) and type C (Malformation) brain lesion were not seen in any type of CP. 4 (8.89%) cases of spastic type of CP did not have any type of lesion on MRI brain.

Type E (Periventricular leukomalacia) brain lesion was also most common lesion seen 21 (80.77%) of diplegia, 14 (100%) of quadriplegia and 10 (100%) of hemiplegia type of CP. Type D (Enlargement of lateral ventricle), type H (Thin corpus callosum), and type G (Border zone infarction) were next most common lesion seen in cerebral palsy with (diplegia, quadriplegia and hemiplegia). Type B (Cerebral atrophy) brain lesion was seen in 3 (21.43%) of quadriplegia and 1 (16.67%) of right hemiplegic cases. Type F (Porencephaly) was seen in 5 (35.71%) of quadriplegic and 1 (3.85%) case of diplegic type of CP (p=0.015). Type I (Cerebellar atrophy) brain lesion was seen in only 1 (7.14%) case of quadriplegic type of CP. 4 (15.38%) cases of diplegic type of CP does not have any type of brain lesion on MRI. Similar findings were reported by Okumura et al.[22]

Although Type E brain lesion was observed in most of the children with CP, there was no specific correlation with any tone type or topographical classification of CP.

In children with CP abnormal neuroanatomical findings were found in 80% to 90% of cases with help of MRI. Brain MRI in CP shows area and extent of lesion in majority of cases which is helpful in correlating with functional outcome.

CONCLUSION

The type and extent of the brain lesion aid in the clinician's comprehension of the functional and motor outcomes as well as the application of rehabilitation techniques in the treatment of cerebral palsy. In certain instances, though, an MRI does not reveal a brain lesion. Therefore, clinical assessment and analysis continue to be crucial for diagnosing cerebral palsy and developing the best rehabilitation strategies.

Conflicting Interest: Nil
Funding: Nil

REFERENCES

1. Stanley F, Blair E, Alberman E. Cerebral Palsies: Epidemiology and Casual Pathways. London: Mac Keith Press; 2000.
2. Gainsborough M, Surman G, Maestri G. Validity, and reliability of the guidelines of the surveillance of cerebral palsy in Europe for the classification of cerebral palsy. Dev Med Child Neurol 2008; 50:828-831.
3. Barkovich AJ, Kuzniecky RI, Jackson GD, Guerrini R, Dobyns WB. Classification system for malformations of cortical development. Neurology. 2001; 57:2168-10.
4. Mukharjee S, Garbler-Spira DJ. Cerebral Palsy. Braddom R L. Physical Medicine and Rehabilitation: New Delhi. Elsevier 2008:1243-67.
5. Cioni G, Di Paco C, Bertuccelli C, Paolicelli PB, Canapicchi R. MRI findings and sensorimotor development in infants with bilateral spastic cerebral palsy. Brain Dev 1997; 19:245–53.
6. Ashwal S, Russman BS, Blasco PA. Practice parameter: diagnostic assessment of the child with cerebral palsy: report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology 2004; 62:851-63.
7. Robinson MN, Peake LJ, Ditchfield MR. Magnetic resonance imaging findings in a population-based cohort of children with cerebral palsy. Dev Med Child Neurol 2009; 51:39-45.
8. Krageloh-Mann I, Horber V. The role of magnetic resonance imaging in elucidating the pathogenesis of cerebral palsy: a systematic review. Dev Med Child Neurol 2007; 49:144-51.
9. Staudt M, Krageloh-Mann I, Grodd W. Ipsilateral corticospinal pathways in congenital hemiparesis on routine magnetic resonance imaging. Pediatr Neurol 2005; 32:37-39.
10. Wilke M, Staudt M, Juenger H, Grodd W, Braun C, Krägeloh-Mann I. Somatosensory system in two types of motor reorganization in congenital hemiparesis. Topography and function. Hum Brain Mapp 2009; 30:776-88.
11. Cioni G, Sales B, Paolicelli PB, Petacchi E, Scusa MF, Canapicchi R. MRI and clinical characteristics of children with hemiplegic cerebral palsy. Neuropediatrics 1999;30: 249-55.
12. Okumura A, Kato T, Kuno K, Hayakawa F. MRI findings in patients with spastic cerebral palsy. Correlation with type of cerebral palsy. Dev Med Child Neurol 1997; 39:369-72.
13. Leventer RJ, Phelan EM, Coleman LT, Kean MJ, Jackson GD, Harvey AS. Clinical and imaging features of cortical malformations in childhood. Neurology 1999; 53:715-22.
14. Sugimoto T, Woo M, Nishida N, Araki A, Hara T, Yasuhara A, Kobayashi Y, Yamanouchi Y. When do brain abnormalities in cerebral palsy occur? An MRI study. Dev Med Child Neurol 1995; 37:285-92
15. Krageloh-Mann I. Imaging of early brain injury and cortical plasticity. Exp Neurol 2004; 190:84–90.
16. Ashwal S, Russman BS, Blasco PA, Miller G, Sandler A, Shevell M et al. Practice parameter. Diagnostic assessment of the child with cerebral palsy. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology 2004; 62:851-10.

Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.7.2024.24

17. Rosenbaum P, Paneth N, Leviton A, Goldstein M, Bax M. A report: The definition and classification of cerebral palsy April 2006. Dev Med Child Neurol 007; 49:8-14.
18. Erkin G, Delialioglu SU, Ozel S. Risk factors and clinical profiles in Turkish children with cerebral palsy: analysis of 625 cases. Int J Rehabil Res 2008; 31:89-91.
19. Benedict RE, Patz J, Maenner MJ. Feasibility and reliability of classifying gross motor function among children with cerebral palsy using population-based record surveillance. Paediatric and Perinatal Epidemiology 2010; 25:88-96.
20. Andersen GL, Irgens LM, Haagaas I. Cerebral palsy in Norway:prevalence, subtypes and severity. European Journal of Paediatric Neurology 2008; 12:4-13.
21. Himmelmann K, and Uverbrant P. Function and neuroimaging in cerebral palsy: a populationbased study. Developmental Medicine & Child Neurology 2011; 53:516–21.
22. Okumura A, Kato T, Kuno K. MRI findings in patients with spastic cerebral palsy. 1: correlation with type of cerebral palsy. Dev Med Child Neurol 1997; 39:369-72.