Clinico-etiological and radiological evaluation of dyskinetic cerebral palsy in a tertiary care centre

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Abstract

Background and Objectives:

Cerebral palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. Dyskinetic cerebral palsy is characterized by abnormal postures or movements with impaired muscle tone regulation, movement control, and coordination, comprising two major movement disorder patterns: dystonia and choreoathetosis. This study was conducted to study the etiological risk factors, clinical and radiological features of dyskinetic cerebral palsy.

Methods: Children aged between 1 month and 18 years diagnosed with dyskinetic cerebral palsy, who presented to the tertiary care center, Bangalore in between November 2019 and May 2021, were studied. Clinical history, examination, and radiological characteristics were analyzed and interpreted.

Results: Dyskinetic CP had a slight male predominance, with a higher incidence in children born at term gestation. The development of motor milestones was more affected than cognition. Perinatal asphyxia (66.6%) as a cause of CP was more common than bilirubin-induced neurologic dysfunction (BIND) (34%). Children with BIND showed a higher incidence of hearing impairment (13.7%), while visual impairment was seen more commonly with perinatal asphyxia. Both dystonia and spasticity were commonly seen in these children. In the MRI, changes involving the globus pallidus were seen in children with BIND, while posterior putamen and thalamus and cortical changes were associated with perinatal asphyxia.

Conclusion: In Dyskinetic CP, perinatal asphyxia is more common than BIND. The history of birth asphyxia, neonatal hyperbilirubinemia, visual, and hearing impairment is helpful to differentiate these subcategories. MRI shows globus pallidus involvement in all children with BIND and posterior putamen and thalamus involvement in all cases of birth asphyxia, thus confirming dyskinetic CP due to BIND or birth asphyxia.

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Keywords:

- BIND
- Dyskinetic CP
- Choreoathetosis
- Globus pallidus

Introduction

Cerebral palsy is a group of permanent disorders affecting the development of movement and causing a limitation of activity.[1] Non-progressive disturbances that manifest in the developing fetal or infant brain led to cerebral palsy. It is the most common cause of childhood disability. The degree and type of motor impairment and functional capabilities vary depending on the etiology. Cerebral palsy may have several associated comorbidities, including epilepsy, musculoskeletal problems, intellectual disability, feeding difficulties, visual abnormalities, hearing abnormalities, and communication difficulties.[2] Cerebral palsy (CP) is a neurodevelopmental condition that affects muscle tone, movement, and motor skills. This is not a single disease but rather a heterogeneous clinical syndrome resulting from injury to the developing brain. Although the disorder itself is non-progressive, the clinical expression changes over time as the brain matures.[3]

The present definition of CP was adopted by the international consensus in 2005, which states: 'Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain'.[4] The motor disorders of CP are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy and by secondary musculoskeletal problems.[5]

Dyskinetic cerebral palsy (DCP) is the second most common type of cerebral palsy after spastic forms. DCP is typically caused by non-progressive lesions to the basal ganglia or thalamus, or both, and is characterised by abnormal postures or movements associated with impaired tone regulation or movement coordination.[6] In DCP, two major movement disorders, dystonia and choreoathetosis, are present together most of the time. Dystonia is often more pronounced and severe than chorea-athetosis, with a major effect on daily activity, quality of life, and societal participation.[7]

Many different hypotheses suggest an imbalance between indirect and direct basal ganglia pathways, disturbed sensory processing, and impaired plasticity in the basal ganglia.[7] Rehabilitation strategies are typically multidisciplinary. Use of oral drugs to provide symptomatic relief of the movement disorders is limited by adverse effects and the scarcity of evidence that the drugs are effective. Neuromodulation interventions, such as intrathecal baclofen and deep brain stimulation, are promising options.[8] In India, the pooled prevalence of cerebral palsy is 2.95/1000 children. Based on the evidence suggesting the involvement of the basal ganglion as the major pathology, an MRI brain should be suggested among all the cerebral palsy children to rule out the metabolic complications. Also, thorough ophthalmological examination, audiometry, and brain evoked response audiometry (BERA) should also be suggested.

Hyperbilirubinemia is known to increase the risk of developing CP. Neonates exposed to high levels of bilirubin continue to experience severe motor symptoms and cerebral palsy. Exposure to moderate levels of unconjugated bilirubin may also cause damage to the developing central nervous system, specifically the basal ganglia and cerebellum. Advances in the care of neonatal hyperbilirubinemia have decreased the incidence of kernicterus.[9] These brain lesions can be identified using MRI, following extreme hyperbilirubinemia has been linked to dyskinetic CP. Advanced imaging techniques, such as diffusion tensor imaging or single-photon emission computed tomography (CT) allow quantification of more subtle white matter injury following presumed exposure to unbound bilirubin and might explain more subtle movement disorders. New categories of bilirubin-induced neurologic dysfunction, either independently or characterized by subtle bilirubin encephalopathy, following moderate hyperbilirubinemia, have been implicated in long-term motor function. Global incidence of kernicterus is estimated to be 1 in 40,000 births, about 1 in 650 to 1000 neonates born >35 weeks postmenstrual age (PMA) experiencing transient hyperbilirubinemia with >25 mg/dL.[10] But few epidemiological surveys have noted the gradual increase in these incidences, irrespective of the improved care of the CP children. This has become one of the challenging scenarios in early detection and treatment. Guidelines for the

management of CP children have liberalised the investigation and phototherapy of the neonates to early detection and treatment.[11] Hence, we conducted a study to analyse clinic-etiological and radiological evaluation of dyskinetic cerebral palsy at our tertiary care centre. The objectives are to study the etiological risk factors, clinical and radiological features of dyskinetic cerebral palsy

Methodology:

Children aged between 1 month and 18 years diagnosed with dyskinetic cerebral palsy, who presented to the outpatient and inpatient department in a tertiary care center, Bangalore, were enrolled for the study. This Observational study was conducted from November 2019 – May 2021. All those children aged between 1 month and 18 years diagnosed and clinically classified as Dyskinetic Cerebral palsy and mixed Cerebral Palsy [Dystonic and Spastic type] were included and other types of cerebral palsy, all those children with dyskinetic cerebral palsy who already underwent orthopaedic or neurological treatment and dyskinesia secondary to the progressive neurological disorder were excluded from this study. A predesigned and pre-structured proforma was used for details of history, physical examination, and MRI of the brain and other relevant investigations. Informed Consent was taken from the parents of children before undertaking this study. Children were subjected to an MRI. Data was collected and entered into a Microsoft Excel sheet and was analysed. Based on published literature,[12] the sample size was 50. Sample Size = $\{Z2^*(p)^*(q)\}/\Delta 2$ where, P: Prevalence of dyskinetic CP 0.15; 1-α: Confidence level 0.95; Z: Z value associated with confidence 1.96; Δ: Absolute precision 0.1; N: Minimum sample size 49.

Results

A total of 51 children diagnosed with dyskinetic CP were enrolled in the study. Table 1 shows various details of the study population. Statistical tests applied included the Chi-square test; HS – Highly significant at $p<0.01$; S–Significant at $p<0.05$.

Table 1 shows various demographic details, clinical features, and laboratory details.

Parameter	**Sub-Parameter**	**BIND=17 (33.33%)**	**HIE=34 (66.66%)**	**P- value**
Gender	Male	11 (64.7)	15 (44.1)	0.16
	Female	6 (35.7)	19 (55.8)	
Gestational age	Term	12 (70.5)	29 (85.2)	0.46
	Preterm	4 (23.5)	4 (11.7)	
	Post-term	1 (5.8)	1 (2.9)	
Birth history	HIE	0(0)	34 (100)	0.0000
	Jaundice	17 (100)	0(0)	
Birth weight	Low birth weight	3 (17.6)	5 (14.7)	0.33
	Large baby	0 (0)	4 (11.7)	
	Normal	14 (82.3)	25 (73.5)	
Development	Global developmental delay	9 (52.9)	34 (100)	0.000
	Predominant Motor delay	8 (47)	0 (0)	HS
	Seizures	2(11.7)	2(5.8)	
	Developmental delay plus seizures	0(0)	1(2.9)	

Anthropometry	Wasted	0 (0)	1 (2.9)	0.47
	Stunted	1 (5.8)	0 (0)	
	Wasted and stunted	15 (88.2)	33 (97)	
	Normal	1 (5.8)	0(0)	
Head circumference	Normal	3 (47)	3(8.1)	1
	Microcephaly	9 (52.9)	31(91.1)	
Tone	Variable	6 (35.2)	4 (11.7)	
	Spasticity	9 (52.9)	26 (76.4)	
	Hypotonia	2 (11.7)	4 (11.7)	
Extrapyramidal symptoms	Dystonia	6 (29.4)	4 (14.7)	0.5
	Chorea	1 (5.8)	1 (2.9)	
	Athetosis	0 (0)	1 (2.9)	
	Dyskinesia and spasticity	11 (64.7)	27 (79.4)	
Hearing	Impaired	6 (85.7)	1 (14.3)	0.002
Vision	Impaired	4 (30.7)	9 (69.3)	0.012
MRI findings	Putamen + Thalamus	3 (17.6)	10 (29.4)	0.000
	Globus pallidus	13 (76.4)	3 (8.8)	

HIE-hypoxic-ischemic encephalopathy, BIND-bilirubin-induced neurological dysfunction, MRI-Magnetic Resonance Imaging.

Children with BIND most commonly had globus pallidus involvement, while those with perinatal asphyxia more commonly had involvement of putamen+thalamus and periventricular leucomalacia changes. Figures 1 and 2 depict salient clinical and neuroimaging features seen in children with dyskinetic cerebral palsy.

Figure 1 shows various clinical and Magnetic Resonance Imaging (MRI) features of dyskinetic cerebral palsy due to bilirubin-induced neurological dysfunction (BIND). Image 1a shows a clinical photograph of a child with dystonia, hypoplastic upper incisor teeth. 1b shows icterus in the conjunctiva and upward gaze weakness, 1c shows protrusion of the tongue due to tongue dystonia, 1d shows hypoplastic upper incisors, and 1e shows T2W MRI of the brain with symmetrical hyperintensities in the globus pallidus.

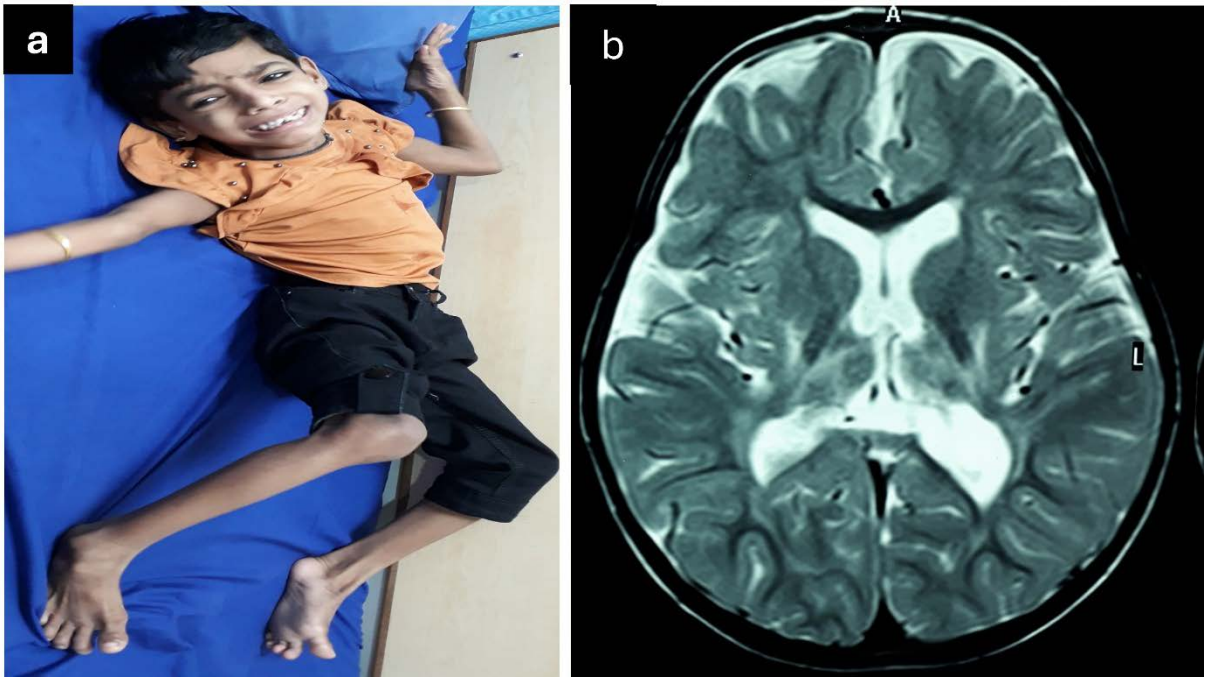

Figure 2 shows clinical and Magnetic Resonance Imaging (MRI) images of a child with dyskinetic cerebral palsy due to hypoxic ischemic encephalopathy. Image 2a shows a clinical photograph of a child with severe generalized dystonia, image 2b shows an MRI of the brain, T2W hyperintensities in the posterior putamen and thalamus.

Discussion

This was a prospective, observational study conducted at a tertiary care center from November 2019 to May 2021, in which 51 children diagnosed with Dyskinetic Cerebral Palsy aged between 1 month and 18 years were studied. Dyskinetic CP had a slight male predominance, with a higher incidence in children born at term gestation and those with normal birth weight. All of them presented with delayed attainment of milestones, motor more than cognitive. Perinatal asphyxia 34/51(67%) as a cause of CP was more common than bilirubin-induced neurological dysfunction 17/51(34%). While global developmental delay was seen in children with perinatal asphyxia, predominant motor delay was commonly seen in children with BIND. Malnutrition was commonly seen in children with Dyskinetic CP, with a large majority of children being both stunted and wasted. Microcephaly was more commonly seen when the cause of CP was perinatal asphyxia. Children with BIND showed a higher incidence of hearing impairment, while visual impairment was seen more commonly with perinatal asphyxia. Both dystonia and spasticity were commonly seen in these children. The presence of spasticity should not mislead the diagnosis of dyskinetic CP as spastic CP. In the MRI, changes involving the globus pallidus were seen in children with BIND, while mixed lesions involving basal ganglia, cortico-subcortical, and white matter were associated with perinatal asphyxia. Among the children diagnosed with Dyskinetic Cerebral Palsy, in our study 50.9% were males and 49% were females. In the study by Sun et [al], males comprised 65.6% and in the study by van Toorn et al, males made up to 76.6% of the study population, as shown in table 2.[13,14]

In our study, 80.3% were term, 15.6% preterm, and 3.9% post-term gestation, which is comparable to previous studies. Sun et al, 63.4% were term, 35.5% were preterm, and 1.1% were post-term, and van Toorn et al, 70% were term and 30% were preterm. [13,14] This shows a higher incidence of dyskinetic CP in term babies compared to preterm and post-term babies.

In our study, 74.5% had dyskinesia as the predominant clinical manifestation. In the study

by van Toorn et al, 86% had dyskinesia.[14] In the study by Sun et al, dystonia was present in 26% and choreoathetosis in 26%.[13] In our study, dystonia was more frequently encountered (19.6%) compared to chorea (3.9%) and athetosis (1.9%). Spasticity is also another clinical manifestation that is frequently encountered, amounting to 41% in the study by Sun et al, 63% in the study by van Toorn et al, and 74.5% in our study.[13,14] This high occurrence of spasticity is one of the common reasons for misdiagnosing Dyskinetic CP as Spastic CP. This is very important in terms of management as spastic CP may benefit from Botulinum toxin, muscle relaxants, and surgery; however, these interventions may worsen in dyskinetic CP. Incidence of epilepsy was found to be 9.8% in our study, in contrast to 22.5% in the study by Sun et al and 20% in the study by van Toorn et al.[13,14] This is probably due to the difference in the demographic characteristics of the children included in the study.

Incidence of impaired vision was 25.4% in our study, 12.9% in the study by Sun et al, and 46.6% in the study by van Toorn et al.[13,14] Visual impairment was more common with perinatal asphyxia compared to BIND. Incidence of hearing impairment was 13.7% in our study, 10.7% in the study by Sun et al, and 90% in the study by van Toorn et al.[13,14] This higher incidence of hearing impairment in the study by van Toorn et al is probably due to the inclusion of only children diagnosed with BIND as their study population.[14] Even in our study, 85.7% of the children with hearing impairment had BIND as the etiology of Dyskinetic CP. As per our study, visual impairment is the most common comorbidity in children with Dyskinetic CP due to perinatal asphyxia, followed by hearing impairment and epilepsy in the order of their incidence.

In our study, perinatal asphyxia accounted for 67.6% and neonatal hyperbilirubinemia accounted for 33.3% of the causes of Dyskinetic CP. In the study by Sun et al, neonatal hyperbilirubinemia accounted for 32.3% of the cases, HIE 34.3%, and NNHB+HIE 8.6% of the cases.[13] Van Toorn et al included only children with a history of NNHB in their study.[14] According to our study and the study by Sun et al, asphyxia and HIE are a more common cause of Dyskinetic CP compared to NNHB.[13] In our study, the incidence of dyskinesia+spasticity was found to be more common among children with perinatal asphyxia as a cause for CP compared to BIND, while the incidence of dystonia was equal in both perinatal asphyxia and BIND groups. In the study by Sun et al, the most common MRI finding in a child with dyskinetic CP was basal ganglia abnormalities, followed by white matter lesions.[13] Normal MRI was found in 10.7% of the children. In the study by van Toorn et al, all children included in the study had BIND as the cause for CP, and basal ganglia abnormalities on MRI were noticed in 80% of the MRIs done.[14] In our study, pure basal ganglia changes were seen in 56.8% of the children, basal ganglia with white matter involvement were seen in 14.7% of them, and basal ganglia + cortico-subcortical + white matter changes were seen in 33.3% of the subjects. 94% of children with BIND had an MRI showing basal ganglia abnormalities, while children with perinatal asphyxia showed mixed lesions involving basal ganglia, cortico-subcortical, and white matter more commonly.

Table 2 Comparison of various parameters with other studies.

Parameter n(%)	**Sun et al 93 (%) [13]**	**van Toorn et al 30 (%) [14]**	**Present study 51(%)**
Gender			
Male	65.5	76.6	50.6
Female	34.4	23.3	49
Age group	4-13 years	14 months- 12.5 years	1 month-18 years
Gestations			
Term	63.4	70	80.3
Preterm	35.5	30	15.6
Post-term	1.1	NR	3.9

Birth History			
Hypoglycaemia	32.2	100	33.3
HIE	34.3	NR	66.7
Hypoglycaemia and HIE	8.6	NR	
Clinical features			
Dystonia	26	NR	19.6
Chorea	26	NR	3.9
Athetosis	NR	NR	1.9
Spasticity	41	63	68.6
Dyskinesia		86	
Co- morbidities			
Microcephaly	12.9	NR	78.4
Epilepsy	22.5	20	9.8
Visual impairment	12.9	46.6	25.4
Hearing impairment	10.7	90	13.7
Duration	10 years	5 years	1.5 year
Etiology			
Hypoglycaemia	32.3	100	33.3
HIE	34.3	NR	66.7
HIE and hypoglycaemia	8.6	NR	
MRI changes			
Basal ganglia only	28 (30.1)	20 (80)	29 (56.8)
White matter only	22(23.6)	NR	0
Basal ganglia and white matter	22 (23.6)	NR	5 (14.7)
Normal	10 (10.7)	5 (20)	0

HIE-hypoxic-ischemic encephalopathy, MRI-Magnetic Resonance Imaging, NR-Not reported

Conclusion

In Dyskinetic CP, perinatal asphyxia is more common than BIND. The history of birth asphyxia, neonatal hyperbilirubinemia, visual, and hearing impairment is helpful to differentiate these subcategories. MRI shows globus pallidus involvement in all children with BIND and posterior putamen and thalamus involvement in all cases of birth asphyxia, thus confirming dyskinetic CP due to BIND or birth asphyxia.

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