Clinical Presentations and CT Scan Findings in Children with Cerebral Palsy

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Abstract

Background: Cerebral palsy is non- progressive disorder of posture or movement due to a lesion of the developing brain. It is the commonest physical disability in childhood.

Objective: To study the clinical, neurological abnormalities, prevalence of convulsion (epilepsy) & to asses the value of CT scans of brain in patients with cerebral palsy.

Patients& Methods: It is a cross-sectional hospital based study, carried at Al-Kindy Teaching hospital out-patient pediatrics unites for the period from first of January through June 2005.

A total of 91 cases (52 boys, 39 girls) with over all mean age 25.6-month range (1 month-84 month) were collected. Data were collected from their parents about age, sex, main clinical presentation, and prenatal, perinatal, postnatal history, history of convulsion. They all had clinical examination.

All patients were sent to radiology department at same hospital for CT scan of brain.

Results: The study revealed that boys affected more than girls (52 boys, 39 girls) with male to female ratio 1.33:1,thecommonest age group affected between 7 month-12 month (30.8%), followed by the age group between 13-24 month (20.9%). delayed milestone with hypertonia & convulsion was the commonest clinical presentation, each represent (48.3%) followed by delayed milestone with hypotonia (34.0 %), microcephaly (29.6%) & speech delay (27.4%). The commonest type of cerebral palsy was spastic form (53. 9%), followed by hypotonic form (34.0%). Convulsion was predominant in quadriplegic cerebral palsy (50%). cortical brain atrophy was commonest CT scan finding (41.7%), followed by sub cortical brain atrophy (28.6%). Cortical brain atrophy was predominant in quadriplegic cerebral palsy (27.5%).CT scan was normal in (19.8%), predominantly in hypotonic cerebral palsy (13.2%).

Conclusion: The pattern of clinical presentation & prevalence of convulsion in patients with cerebral palsy in the present study are comparable to the result from studies in other clinical settings with slight variation. CT scan of brain is highly valuable in patients with cerebral palsy.

Key Words: Cerebral palsy, Convulsion, CT scan

Introduction:

Cerebral palsy is diagnostic term used to describe a group of motor syndrome resulting from disorder of early brain development.[1]

It s a heterogeneous group of persistent disorder of movement and posture caused by non-progressive defects or lesions of immature brain with different etiologies in the pre-, peri- or postnatal period. [2, 3, 4]

The most important of these diseases is cystic per ventricular leukomalacia, followed by intra- and periventricular leukomalacia, hypoxic-ischemic encephalopathy, vascular disorders, infection or brain malformation. [3]

It' s the most common cause of severe chronic disability and suffering in child, occurring in about 2-2.5\1000 live birth. [1, 2, 5]

Cerebral palsy can be classified according to pathology of brain injury e.g. vascular, infective, inflammatory, or traumatic or according to the timing of brain injury such as prenatal, perinatal or postnatal. [1, 2]

The modified classification is widely used classification. This is based on the tone, number & distribution of affected limbs. The changing nature of symptoms& signs make the clinical classification difficult in the first year of life as pattern of movement & tone may changes completely.

The revised classification now in use define 3 main categories of motor disorders, spastic (70-80%), dyskinetic (10-15%), ataxic (< 5%) [1, 2, 3].

The motor disorders of cerebral palsy are often accompanied by disturbance of sensation, cognition, communication, perception and\or behavio- or, and\or a seizure disorder [5].

The presentation of cerebral palsy can be global mental and physical dysfunction or isolated disturbance in gait, cognition [6, 7].

The diagnosis of cerebral palsy is made purely on clinical grounds and is based upon a history of abnormal motor development that is non progressive coupled with an examination (e.g. hyper tonicity, increased reflex, Clonus) placing the lesion in the brain [2, 8] .

In order to establish that a brain abnormality exists in children with cerebral palsy that may, in turn, suggest an etiology and prognosis, neuroimaging is recommended [8, 9] .

The aim of this study is to identify clinical and neurological abnormalities in children with cerebral palsy, the prevalence of convulsion and to asses the value of CT scan in patients with cerebral palsy.

Patients & Methods:

It's a cross sectional descriptive hospital-based study carried at AL-Kindy Teaching Hospital outpatient pediatrics unit, from the first of January through June 2005.

A total of 91 cases (52 boys, 39 girls) were collected during this period, sociodemographic information about age, sex, history of main clinical presentation, convulsion, prenatal, perinatal, postnatal histories were taken from their parents.

They all had full clinical & neurological examination; head circumference was measured by tape measure. All patients were sent to radiology department for CT scan of brain.

Data were analyzed using percentage, chi-square, fisher's test & p value. The value less 0.05 is considered significant.

Results:

The total number of patients was 91(52 boys and 39 girls) with male to female ratio 1.33: 1. Their ages ranged between 1 month and 84 months with a mean age 25.6 month.

Table 1 shows the distribution of cases of cerebral palsy according their age ,the age group (7-12month) was mostly involved in 28 cases (30.8%) followed by the age group (13-24month) in 19 cases (20.9%).

Table 2 shows the clinical presentation of cases of cerebral palsy, 44 cases (48.3%) presented with delayed milestone and hypertonia, 44 cases (48.3%) presented with convulsion, 31cases (34.0%) presented with delayed milestone and hypotonia. 27 cases (29.6%) presented with small head (micro-cephaly), 25 cases (27.4%) presented with speech delay.

Table (1) Distribution of cases of cerebral palsy according Age

Age	No.	%
1-6months	14	15.4
7-12 month	28	30.8
13-24 month	19	20.9
25-36 month	7	7.7
37-48 month	9	9.9
49-60 month	6	6.6
61-72 month	4	4.4
73-84 month	4	4.4
Total	91	100%

Table (2) main clinical presentation of cases of cerebral palsy

main clinical presentation	No.	%
delayed milestone +hypertonea	44	48.3
convulsion	44	48.3
delayed milestone +hypotonea	31	34
small head	27	29.6
delayed speech	25	27.4
large head	9	9.9
squint	7	7.7
hemiplegics gait	5	5.5
Nystagmus + tremor	3	3.3
Dysmorphic Feature	3	3.3
Hepato-spleenomegaly	2	2.2
bilateral cataract	1	1.1

Table 3 shows that spastic cerebral palsy was common type in present study. Quadriplegic type was found in 36 cases (39.6%), diplegic 8 cases (8.8%), hemiplegics 5 cases (5.5). totally they represent 49 cases (53.9%). Followed by hypotonic cerebral palsy 31 cases (34.0%), extra-pyramidal &ataxic cerebral palsy was found in 9 cases (9.9%) & 2 cases (2.2%) respectively.

The distribution of cases of convulsion in different types of cerebral palsy is shown in table 4. Twenty two cases of convulsion (50%) accompanied quadriplegic type, Followed by 10 cases (22.7%) in hypotonic cerebral palsy. 8 cases (18.2%) in diplegic cerebral palsy, two cases (4.5%) in hemiplegics & one case (2.3%) in extra pyramidal & ataxic cerebral palsy respectively.

Table (3) Types of Cerebral Palsy cases

Types spastic	No.	%
Quadriplegia	36	39.6
Diplegia	8	8.8
Hemiplegia	5	5.5
extrapyramidal	9	9.9
hypotonic	31	34
Ataxic	2	2.2
Total	91	100%

Table (4) Distribution of cases of convulsion according to types of Cerebral Palsy.

Types of C.P	No. of convulsion	%
Quadriplegic	22	50
Diplegic	8	18.2
Hemiplegic	2	4.5
extrapyramidal	1	2.3
hypotonic	10	22.7
Ataxic	1	2.3
Total	44	100%

CT scan changes in patients with cerebral palsy were shown in table 5.

The commonest CT scan changes was brain atrophy which was presented totally in 66 cases (72.5%), of which 36 cases (41.7%) had cortical brain atrophy with dilatation of ventricles,26 cases (28.6%) had sub cortical brain atrophy & 2 cases (2.2%) had hemi-atrophy. Encephalitis was presented in 5 cases (5.5%), communicating hydrocephaly was presented in two cases (2.2%), CT scan was normal in 8 cases (19.8%).

CT scan changes in different type of cerebral palsy was shown in table 6 Cortical brain atrophy with dilatation of ventricles which was the commonest CT changes presented in 38 cases (41.7%), of which 25 cases (27.5%) was seen in quadriplegic type, 11cases (12.%) in hypotonic type, two cases (2.2%) in extra-pyramidal type.

Sub cortical brain atrophy was presented in 26 cases (28.6%), of which 7 cases (7.7%) were seen in diplagic type, 6 cases (6.6%) in hypotonic type, 5 cases (5.5%) in extra pyramidal type, 3 cases (3.3%)in quadriplegic type, three cases (3.3) in hemiplegics, &two cases (2.2%) in ataxic type of cerebral palsy.

Hemi-atrophy presented in 2 cases (2.2%) of which 2 cases (2.2%) was seen in hemiplegics cerebral palsy.

Encephalitis presented in 5cases (5.5%), of which 4cases (4.4%) was seen in quadriplegic type, one case (1.1%) in hypotonic cerebral palsy.

Communicating hydrocephaly was presented in 2 cases (2.2%), of which one case (1.1%) was in quadriplegic type & one case (1.1%) was seen in hypotonic cerebral palsy.

Normal CT scan was presented in 18 cases (19.8%), of which 12 cases(13.2%) of normal CT was found in hypotonic type, three cases (3.3%) in quadriplegic type, two cases (2.2%) in extra pyramidal, &one case (1.1%) in diplegic type of cerebral palsy.

Table (5) CT scan changes

CT scan changes	No.	%
Brain atrophy		
cortical +ventriculomegaly	38	41.7
sub cortical	26	28.6
hemi atrophy	2	2.2
Encephalitis	5	5.5
communicating hydrocephaly	2	2.2
Normal	18	19.8
Total	91	100%

Table (6) CT Scan changes in different types of cerebral palsy

changes of CT	Quadriplegic	Diplegic	hemiplegic	extrapyramidal	hypotonic	Ataxic	Total
Brain atrophy							
cortical	25 (27.5%)	-	-	2(2.2%)	11(12.0%)	-	38(41.7%)
sub cortical	3(3.3%)	7(7.7%)	3(3.3%)	5(5.5%)	6(6.6%)	2(2.2%)	26(28.6%)
hemi atrophy	-	-	2(2.2%)		-	-	2(2.2%)
Encephalitis	4(4.4%)	-	-	-	1(1.1%)	-	5(5.5%)
communicating hydrocephaly	1(1.1%)	-	-	-	1(1.1%)	-	2(2.2%)
Normal	3(3.3%)	1(1.1%)	-	2(2.2%)	12(13.2%)	-	18(19.8%)
Total	36	8	5	9	31	2	91(100%)

Table 7 shows significant association between convulsion & quadriplegic cerebral palsy, where convulsion was presented in 22 cases (50%).

Table 8 shows highly significant association between convulsion & neonatal convulsion, where onset of convulsion started early in neonatal period in (52.3%).

Table 9 shows highly significant association between cortical brain atrophy & quadriplegic cerebral palsy, where cortical brain atrophy was presented in 25 cases (27.5%).

Table 10 shows highly significant association between sub cortical brain atrophy & quadriplegic cerebral palsy, where sub cortical brain atrophy was presented only in three cases (3.3%) only.

Table (7) association between Quadriplegia & convulsion

Quadriplegia Convulsion	non Quadriplegic	Quadriplegic	Total
no Convulsion	33	14	47
Convulsion	22	22	44
Total	55	36	91

$\chi 2=3.88$, df=1, p= 0.048 significant

Table (8) association between neonatal convulsion&convulsion

Neonatal Convulsion Convulsion	no Neonatal Convulsion	Neonatal Convulsion	Total
no Convulsion	41	6	47
Convulsion	27	17	44
Total	68	23	91

$\chi 2=6.71$, df=1, p=0.0009 highly significant

Table (9) association between Quadriplegia & cortical brain atrophy

Quadriplegia Cortical	non Quadriplegic	Quadriplegic	Total
no Cortical	42	11	53
Cortical	13	25	38
Total	55	36	91

$\chi 2=16.94$, df=1, p=0.000036 highly significant

Table (10) association between Quadriplegia & subcortical brain atrophy

Quadriplegia Subcortical	non Quadriplegic	Quadriplegic	Total
No Subcortical	32	33	65
Subcortical	23	3	26
Total	55	36	91

p=0.001 highly significant

Table 11 shows significant value in which cortical brain atrophy was not seen in diplegic cerebral palsy.

Table 12 shows highly significant association between sub cortical brain atrophy& diplegic cerebral palsy, where sub cortical brain atrophy was presented in 7 cases (7.7%).

Table 13 shows highly significant association between hemi atrophy& hemiplegics cerebral palsy where hemi atrophy was presented only in hemiplegics cerebral palsy.

Table 14 shows no significant association between cortical brain atrophy & hemiplegics cerebral palsy.

Table (11) association between Diplegia & cortical brain atrophy

Diplegia Cortical	**non diplegic**	**diplegic**	**Total**
no Cortical	45	8	53
Cortical	38	0	38
Total	83	8	91

P= 0.0188 **highly significant**

Table (12) association between Diplegia & subcortical brain atrophy

Diplegia Subcortical	**non diplegic**	**diplegic**	**Total**
no Subcortical	64	1	65
Subcortical	19	7	26
Total	83	8	91

P= 0.000521 **highly significant**

Table (13) association between Hemiplegia & Hemiatrophy

hemiplegia hemi atrophy	**non hemiplegic**	**hemiplegic**	**Total**
no hemi atrophy	86	3	89
hemi atrophy	0	2	2
Total	86	5	91

P= 0.00244 **highly significant**

Table (14) association between Hemiplegia & cortical brain atrophy

hemiplegia Cortical	**non hemiplegic**	**hemiplegic**	**Total**
no Cortical	48	5	53
Cortical	38	0	38
Total	86	5	91

P= 0.072 **not significant**

Discussion:

The presented study revealed that males affected are more than females; this finding was similar to other studies [10, 11, 12]. Male gender considered risk factor in cerebral palsy patients [2].

Prior to 1 year of age, a definite diagnosis of CP is extremely difficult to make, and it should only be made with hard evidence [13]. In the present study the majority of patients were 7month-12 month, followed by the age group 13-24 month.

Clinical examination of these patients revealed obvious gross motor delay, poor head control, spasticity, exaggerated tendon reflex, others presented with hypotonia, diminished reflex.

The main clinical presentation in the present study was delayed motor milestone with hypertonia & convulsion followed by delayed motor milestone with hypotonia. This result similar to study in Saudi children, hypotonia was presented in 41%, hypertonia in 38% [14].

Speech delay was presented in 25cases (27.4%), which is dissimilar to the study of Saudi children where speech delay was presented in 61 % [14].

One case was presented with hepatosplenomegaly& cataract, which raises the suspicion of congenital rubella infection.

The spastic CP was commonest type in the present study followed by hypotonic CP, extra pyramidal CP while ataxic Cp was an uncommon variety. This finding was comparable to the results studies conducted in Baghdad, Dakar, other studies [11, 15, 16]. Quadriplegic spastic CP was commonest type among spastic CP, followed by diplagic, hemiplegics CP also this finding was similar to other clinical studies [9, 11, 12, 15, 16, 17, 18].

The high percentage of cases of Hypotonic CP in this study may be explained by that those cases are either real cases of hypotonic CP, or hypotonic phase of spastic CP. Children with spastic CP are generally hypotonic in the first 6-9 month, and then gradually become hyper tonic [2].

Convulsion in more common in child with CP. (1, 2, 4) the study revealed that convulsion was presented in 44cases (48.3%). this finding was similar to studies in Saudia; Dakar &others with slight variations [2, 4, 10, 12, 14, 16].

Highly significant association between quadriplegic CP & convulsion was found in present study, similar result was found in other studies [2,19,20, 21]. This result could be explained that the brain lesion is more diffuse & extensive in quadriplegic CP.

Highly significant association between the onset of convulsion & convulsion, where in 52.3% of cases the convulsion started in neonatal period. This finding was comparable to the result from other studies [20, 21] .

Although the diagnosis of CP is made purely on clinical ground, but selected investigations may be required for ascertaining the cause [2] .Children with suspected CP should have early neuroimaging test to establish that a brain abnormality exists& to suggest an etiology and prognosis [9] .

The commonest CT finding was cortical brain atrophy with dilatation of ventricles followed by sub cortical brain atrophy& hemi-atrophy of brain. This result is comparable to studies done for Saudi children, Dakar & other studies [2, 12, 14, 16, 22].& dissimilar to a study done in Baghdad, where sub cortical brain atrophy was the commonest type [15] .

Highly significant association was found in the present study between cortical brain atrophy & quadriplegic CP. This result is comparable to studies done in Baghdad& Saudi Arabia [14, 15] .

Sub cortical brain atrophy was seen in all type of CP, but highly significant association was found with diplegic CP, followed by extra pyramidal CP. This is similar to result done in Saudia[15] .Highly significant correlation was found between hemi atrophy of brain & hemiplegics CP, in which hemi atrophy was seen only in hemiplegic CP. this finding, is similar to other studies [2, 8, 24] .

About 2\3 of patients with hemiplegia had unilateral lesions, which are rare in patients with diplagia & quadriplegia [2] .

Encephalitic changes were commonly seen in quadriplegic CP; encephalitic changes affect central white matter by edema early in the illness followed by atrophic changes &calcification later on. (22, 24)

Communicating hydrocephaly was presented in one case with quadriplegic CP & other case with hypotonic CP. similar result was revealed in other studies[12,14] .

CT scan in the present study was normal in 18cases (19.8%), the result was similar to study done in Saudi children, where CT was normal in 11.5%& similar to other clinical studies,[4,12,14,16,22,23] while it's not similar to Baghdad study [15] .High percentage of normal CT in hypotonic CP could be explained, that those cases were either real cases of hypotonic CP or a diagnostic confusion with other diseases like, neuromuscular diseases (myopathies),mental retardation, neurodegenerative disease (Tay-sach, Krabbe's disease, metachromatic leucodystrophy), other metabolic &genetic diseases which mimic hypotonic CP.

Patients who presented clinically with CP may have normal result on brain CT; other underlying metabolic & genetic disease should be excluded [25].

CT is highly valuable in the present study, the pattern of clinical types of CP & abnormalities in CT were comparable to the results from studies in other clinical environments.

We recommend that CT be mandatory in suspected cases of CP to establish any brain abnormalities, & to suggest an etiology & prognosis.

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References:

1-Michel V. Johston. Nervous System; cerebral Palsy, Nelson Text Book of Pediatrics, 17 editions, 2004 W.B. Saunders comp, P.2024-2025.

2-Aneja S. Evaluation of a Child With cerebral Palsy. Indian J Pediatr 2004; 71: 627-634.

3-Maurer U. Etiologies Of cerebral Palsy and Classical Treatment Possibility. Wien Med Wochenschr.2002; 152: 14-8.

4-Laura R .Ment. Toward a Better Understanding of Cerebral Palsy. Journal Watch Neurology.2004; 62: 851-63.

5-Michael V. Johnston; Donna M. Ferriero; Susan J. Vannunci. Models Of Cerebral Palsy: Which Ones Are Best? J Child Neuroi.2005; 20:984-987.

6-Krigger KW. Cerebral Palsy: An Overview. Am Fam Physician. 2006; 73:91-100.

7-Koman LA; Smith BP; Shilt JS. Cerebral Palsy. Lancet 2004; 363:1619-31.

8-Russman BS; Ashawi S. Evaluation of Child with Cerebral Palsy. Semin Pediatr Neurol.2004; 11: 47-57.

9-Lauric Barclay. New AAN Practice Guidelines for Evaluating Children with Cerebral Palsy. Neurology.2004; 62: 851-853.

10-Svraka E; Logas S. Cerebral Palsy & Epilepsy In children. Med Arch. 2005; 59(3): 188-90.

11-Shevell M; Majnemer A; Morin I. Etiological Yield of Cerebral Palsy: a contemporary case series. Pediatr Neurol.2003; 28(5): 352-9.

12-Rahman MM; Akhter S; Akhter MS; Karim BA. Epilepsy in Children With Cerebral Palsy. Mymensingh Med J.2004; 13(1): 67-70.

13-Lawrence T, Taft. Cerebral Palsy. Pediatric in Review.1984; 6(2) 35-37

14-Al-Sulaimann AA. Epilepsy in Saudi Children with Cerebral Palsy.
Saudi Med J.2001; 22(1): 19-21.

15-Al-Nadawi MN; Al-Obeidy L; Umran R. CT Scan Changes in Patient with Cerebral Palsy. J Fac Med (Baghdad). 2002; 44(3): 498-500.

16-Ndiaye M; Thiam A; Bagnou S; N dao AK. Cerebral Palsy in Dakar. Dakar Med. 2002; 47(10:77-80.

17-Himmelman K; Harberg G; Beckung E; Harberg B. The Changing Panorama of Cerebral Palsy in Sweden.ix. Prevalence and origin in the birth period 1995-1998. Acta paediatr. 2005; 94(3): 287-94.

18-Parkes J ; Dolk H; Hill N; Pattender S. Cerebral Palsy In Northern Ireland;1981-93 . Paediatr Perinat Epidemiol. 2001; 15(3): 278-86.

19-Senbil N; Sonel B; Aydin F; Gurer YK. Epileptic and Non Epileptic Cerebral Palsy. EEG & Cranial Imaging Finding. Brain Dev.2002; 24(3): 166-9.

20-Ela paucic Kirincic ; Zlata Modrusan ; Nada Sindicici. Epilepsy among Children With cerebral Palsy Born in RIJEK Between 1982-1992.Medcina. 2005; 41(10:31-36.

21-Bruck I; Antoniuk SA; Spessato A. Epilepsy in Children with Cerebral Palsy. Arq .Neuropsiquiatr. 2001; 59(1): 35-9.

22- Hoon AH. Neuroimaging In cerebral Palsy: patterns of brain dysgenesis and injury. J Child Neurol.2005; 20(12): 936-9.

23-Gururaj a; Szthriha L ; Dawodu A; Nath KR et. al. CT scan & MRI patterns of hypoxic brain damage following perinatal asphyxia. J Trop Pediatr.2002; 48(1): 5-9.

24-Iwabee; Piovesana AM. Comparative study of muscular tonus in spastic tetraplagic cerebral palsy in children with predominantly cortical & subcortical lesion in CT of skull. Arq.Neuropsiquiatr.2003; 61(3): 617-20.

25-Boosara Ratanawongsa. Cerebral Palsy Article by Boosara Ratanawongsa .WWW. e Medicine. Last updated August 9, 2005; 1-22.

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