Cerebral Palsy in Jordan: Clinical and Neuroimaging Characteristics

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Abstract

Objective: To evaluate the diagnostic findings of neuroimaging in patients with cerebral palsy and if there is any specific finding correlated to certain types of Cerebral Palsy.

Methods: Case records of 158 patients diagnosed to have cerebral palsy attending the pediatric neurology and neurodevelopmental clinics at King Hussein Medical Center and King Abdullah University Hospital over 2 years period, 2006 and 2007, were studied retrospectively with reference to their clinical characteristics and their correlation to the neuroimaging (MRI and CT scan) findings.

Results: A total of 158 cases with cerebral palsy were included in the study, 84 (53%) males, 74 (47%) females, 41 (26 %) preterm and 117 (74%) full- term babies. Spastic cerebral palsy was seen in 112(70.8%) with spastic quadriplegia being the commonest seen in 63(40%). Hypotonic ataxic type present in 22(14%), dyskinetic 15(9.4%) and mixed cerebral palsy in 9(5.6%). Abnormal neuroimaging findings were seen on MRI in 125(79%), while in CT scan in 113(71%). Specific neuroimaging findings were seen suggesting brain asphyxia in 40(25%), congenital brain anomaly in 22(14%), intracranial hemorrhage in 10(6%), vascular and infectious causes in 29(18.5%), unknown/isolated brain atrophy in 26(16.5%), and periventricular leukomalacia in 31(20%). The most common single etiology identified was birth asphyxia 40(25 %), and the second is periventricular leukomalacia which was identified in 31 patients (20%). Nonspecific brain atrophy was considered as nonspecific finding, that was found most often in patients with dyskinetic CP 5/15(33 %), and in patients with spastic quadriplegia 15/63(24 %) as compared to other groups.

Conclusion: The principle contribution of imaging is to the understanding of etiology and pathogenesis, including ruling in or out conditions that may have implicated a genetic counseling, such as malformations. MRI is a more sensitive test than CT in detecting brain abnormalities.

Keywords: Neuroimaging, Cerebral Palsy.

(J Med J 2008; Vol. 42 (3): 162-169)

Received	***Accepted***
July 17, 2006	*April 27, 2008*

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Introduction

Cerebral palsy is primarily a disorder of movement and posture. It is defined as a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development.[1] Its incidence is around 2.5:1000 live births.[2] Cerebral palsy is a clinical diagnosis made by an awareness of risk factors, regular developmental screening of all high risk babies and neurological examination.[3] The diagnosis of cerebral palsy is clinical but selected investigations may be required for ascertaining the cause. [4] Brain abnormality may be present in children with cerebral palsy that may suggest an etiology and prognosis. Therefore, neuroimaging is recommended with Magnetic Resonance Imaging, (MRI) is more sensitive to detect these abnormalities than Computerized Tomography (CT). [5] Many reports have highlighted the role of neuroimaging in infants or children who have been diagnosed or are suspected with having cerebral palsy in identifying the places, types and extent of lesions in the CNS. [5, 6]

The progress in understanding the origin of cerebral palsy is thought to be due to the increased access to neuroimaging technique with their improving capacity to show the type of brain pathology. Though not always diagnostic, neuroimaging provides useful diagnostic information to a high degree of cerebral palsy children when no other diagnostic clue is available. Neuroimaging has contributed to the recognition of the brain malformations that are frequently the cause of cerebral palsy.[7]

The purpose of this study is to determine the diagnostic benefit and the finding of magnetic resonance image and computerized tomography in children with cerebral palsy.

Methods

We studied the neuroradiological finding of 158 children diagnosed to have cerebral palsy, attending the pediatric neurology and neurodevelopment clinics in King Hussein Medical Center and King Abdullah University Hospital during 2006 and 2007. Cerebral palsy was defined as a syndrome of non-progressive disorder of movement and posture, secondary to lesions or anomalies of the brain arising in the early stages of its development.[1]

Thus, neurodegenerative, metabolic and neuplastic processes were excluded. Additionally disorders not traditionally considered cerebral palsy were excluded according to Badawi et al. [8] Cerebral palsy was classified topographically according to the extremities involved (monoplegic, hemiplegic, diplegic and quadriplegic) and the characteristics of the neurological dysfunction (spastic, ataxic, dyskinetic and mixed). [9] The medical files for all patients were reviewed and patients meeting the above definition and classification of cerebral palsy were included in this study.

The finding of neuroimaging (MRI/CT) was reviewed and detailed information about the neurological, neurodevelopmental, antenatal, perinatal, postnatal history was taken and plotted on a special data collection sheet developed for this study. Age, gender and gestational age (term, >37 weeks; preterm, <37 weeks) were documented. When the same Neuroimaging method was performed more than once, it was decided to include the first films in the study. Broad categories of possible etiologies were used. The diagnosis of birth asphyxia required the documentation of moderate to severe encephalopathy and was consistent with the recent British Medical Journal consensus statement on the topic. [10] A neuroradiologist reviewed the radiological finding and child neurologist. Brain atrophy was diagnosed when ventricular dilatation with widening of the sulci is present. A vascular etiology was diagnosed when imaging finding revealed a lesion in the vascular territory (such as porencephaly) consistent with the clinical picture. An infectious etiology required positive cerebrospinal fluid finding or immunological evidence of intra-uterine infection. Congenital brain anomaly, brain atrophy and intracranial hemorrhage (intraventricular, subdural and epidural) required positive finding on MRI or CT scan to support the diagnosis.

When more than one etiology was found in a single case we categorized the main cause as the etiological diagnosis.

For all patients included in the study, complete blood count, liver function tests, kidney function tests, serum uric acid, immunological screen for congenital infection and thyroid function tests were done. Specific laboratory tests like screening for inborn error of metabolism and chromosomal analysis were done when needed. Neuroimaging MRI/CT or both were done for all the children included in the study, if not; they were excluded from the study. Magnetic resonance imaging was the preferred investigation but for those who could not tolerate it because of their medical condition; a computerized tomography scan was done instead. The ethical committee of both institutes approved conducting the study.

Analysis

All information was entered into a recoded form. SPSS software was used to analyze the data. X^2 and p values were calculated to show the association between the type of CP and diagnostic yield; $p<0.05$ was considered to be statistically significant.

Result

Hundred fifty-eight cases with cerebral palsy were included in the study, the sex distribution was 84 males (53%), 74 females (47%), and the gestational age distribution was for preterm 41 (26%) and, 117 (74%) for term. The distribution of the types of CP in relation to the sex and gestational age are shown in Table (1). The distribution of patients according to the type of cerebral palsy is listed in Table (2). The most common type observed in our study was spastic quadriplegia 63 (40%).

Although the number of cases in some groups is small (monoplegic, triplegic and mixed cerebral palsy), our results show increased percentage of abnormal neuroimaging findings with certain types of cerebral palsy as seen in Table (2).

Overall, the frequency of abnormal neuroimaging findings was; for MRI 125(79%), for CT scan 113(71%), while specific neuroimaging yield regarding single etiological diagnosis was determined in 103 (65%) and 97 (61%) by MRI and CT scan, respectively, after excluding patients with nonspecific etiologic finding (category of unknown/ brain atrophy) (Table 2).
For the purpose of the study, the neuroimaging findings were classified into 6 main categories, brain asphyxia 40(25%), Congenital brain anomaly 26 (14%), Intracranial hemorrhage 10(6%), Vascular/infection 25(18.5%), Unknown/ brain atrophy 26(16.5%) and Periventricular leukomalacia 31(20%). (Table 3). Overall, the most common single etiology identified was birth asphyxia 40(25 %), and the second is periventricular leukomalacia 31(20%). Nonspecific brain atrophy was considered as nonspecific finding, that was found most often in patients with dyskinetic CP 5/15(33 %), and in patients with spastic quadriplegia 15/63(24 %) as compared to other groups (Table 4). Most of the patients with congenital brain anomaly have neuronal migration defects such as lissencephaly, schizencephaly and pachygyria. Etiology identified varied according to the type of cerebral palsy. Asphyxia was the most frequently observed etiology 40(25%), seen mainly in the spastic quadriplegia 17 (Table 4).

Nonspecific brain atrophy or no etiology was observed in 26 (16.5%) patients, and was evident more in dyskinetic 5/15(33%) and spastic quadriplegia 15/63 (24%). The most frequently observed etiology among males was equal; for PVL and intrapartum asphyxia 23(27%), while among females the most frequent etiology was vascular/infection 21(28%). Specific etiology was not evident in 12/84 (14%) males and in 14(19%) females. In term babies, the most frequent underlying etiology was intrapartum asphyxia 31(26%) while in preterm babies PVL was 22(54%). Etiology was not identified in 23(20%) in term patients and in 3(7%) in preterm patients (Table 5).

Table (1). The distribution of the clinical types of CP in relation to gender and gestational age.

CP type	Male	Female	Term	Preterm	Total, Percentage
Dyskinetic	6	9	10	5	15 (9.4)
Hypotonic ataxic	11	11	20	2	22 (14)
Mixed	4	5	6	3	9 (5.6)
Spastic diplegia	16	11	15	12	27 (17)
Spastic hemiplegia	6	10	13	3	16 (10)
Spastic monoplegia	2	1	0	3	3 (2)
Spastic quadriplegia	37	26	50	13	63 (40)
Spastic triplegia	2	1	3	0	3 (2)
Total	**84**	**74**	**117**	**41**	**158**

Table (2): CT scan and MRI findings according to the type of CP.

CP type	Normal CT	Abnormal CT	Normal MRI	Abnormal MRI	Total
Dyskinetic	6	8	4	9	15
Hypotonic ataxic	1	20	0	22	22
Mixed	4	5	1	8	9
Spastic diplegia	11	15	5	19	27
Spastic hemiplegia	1	14	1	15	16
Spastic monoplegia	2	1	1	2	3
Spastic quadriplegia	14	48	6	48	63
Spastic triplegia	1	2	0	2	3
Total	**40**	**113**	**18**	**125**	**158**

Table (3): MRI and CT scan results according to category.

Category	Normal MRI	Abnormal MRI	Normal CT	Abnormal CT	Total	Percent
Intrapartum asphyxia.	4	25	7	33	40	25.3
Congenital brain anomaly.	1	24	4	22	26	13.9
Intracranial hemorrhage.	0	10	0	10	10	6.3
Vascular/infection.	2	22	9	14	25	18.4
Unknown/isolated brain atrophy.	4	22	8	16	26	16.5
PVL	7	22	12	18	31	19.6
Total	**18**	**125**	**40**	**113**	**158**	**100.0**

Table (4): Diagnostic findings of neuroimaging according to CP type.

	Intrapartum asphyxia	Congenital brain anomaly	ICH	Vascular/ infection	PVL	Unknown/ brain atrophy	Total
Dyskinetic	5	1	0	2	2	5	15
Hypotonic ataxic	6	7	3	1	3	2	22
Mixed	1	1	0	3	3	1	9
Spastic diplegia	7	3	0	2	13	2	27
Spastic hemiplegia	2	2	4	6	1	1	16
Spastic monoplegia	1	0	0	1	1	0	3
Spastic quadriplegia	17	11	2	10	8	15	63
Spastic triplegia	1	1	1	0	0	0	3
Total	**40**	**26**	**10**	**25**	**31**	**26**	**158**

Table (5): Distribution of etiology according to sex and gestational age.

	Male	*Female*	*Term*	*Preterm*	*Total*
Intrapartum asphyxia.	*23*	*17*	*31*	*9*	*40*
Congenital brain anomaly.	*12*	*14*	*24*	*2*	*26*
Intracranial hemorrhage.	*6*	*4*	*8*	*2*	*10*
Vascular/infection.	*8*	*17*	*22*	*3*	*25*
Unknown/isolated brain atrophy.	*12*	*14*	*23*	*3*	*26*
PVL	*23*	*8*	*9*	*22*	*31*
Total	***84***	***74***	***117***	***41***	***158***

Discussion

Neuroimaging (MRI/CT scan) is part of routine investigation that we perform in our pediatric neurology clinic for a precise evaluation of the pathogenesis of cerebral palsy. All cases included in the study were evaluated clinically, neurologicaly in conjunction with neuroimaging findings by the same neurology team in search for possible etiology. Our study is similar to other studies regarding predominance of term born (74%), males (53%), spastic cerebral palsy (71%) and spastic diplegia (17%). [10, 11]

The etiology of CP is diverse and multifactorial and the injury to the developing brain may be prenatal, natal, or postnatal.

Ashwal et al., reviewed data of 1,426 children who underwent either CT scan or MRI and reported that an abnormality of neuroimaging was seen in 62% to 100% of CP children, (mean for CT 77% and for MRI 89%). For the combined CT scan and MRI, 88% of children had an abnormal neuroimaging. [5] Our study showed that 79% of MRI were having abnormalities, compared to 71% of CT scan, in keeping with other published reports. [5]

Hagberg et al. [12] in a study of 241 neuroimaging performed to Swedish children, reported that 90% showed a finding of periventricular atrophy or cortical/subcortical atrophy with negative perinatal history. Normal MRI/CT scan was documented in only 6% of our patients with spastic hemiplegia. This finding is nearly comparable to the figure of 8% and 9% reported by Okumura et al., [13] and Steinlin et al., [14] respectively.

Normal neuroimaging in our study was frequent in the spastic diplegia CP, 18.5% MRI and 41% in CT scan, while for those with quadriplegic type, 9.5% in MRI and 22% in CT scan. The data obtained in this study are in keeping with Michael I et al., who reported etiologic yield to be unknown in 18% of the cases of cerebral palsy. [11]

The role played by birth asphyxia in causing cerebral palsy is still a major issue. A study done by Mc Lennan[10] reported that 75%-80% of the cases are due to prenatal injury with less than 10% being due to birth asphyxia. In our study, the percentage of cerebral palsy caused by birth asphyxia is 40 (25%) and most frequently observed in dyskinetic 5/15(33%) and in spastic quadriplegia 17(24%). Our study reveals that birth asphyxia is present in a small number of cases and varied among subtypes of cerebral palsy. This is compatible with findings reported by Naeye et al. [15]

Stanley et al., [16] using the Western Australian cerebral palsy registry, identified intrapartum asphyxia as a cause in 24% of quadriplegic cerebral palsy; this figure is similar to our finding (24%) and more dominant than other observations.[10] There was little difference regarding the frequency of intrapartum asphyxia whether the child was born premature 9/41(22%), or at term 31/117(26%).

Cerebral atrophy was observed more often in patients with dyskinetic 5/15(33%) and quadriplegic 15/63(24%) than with other types of cerebral palsy. Our findings are in accordance with earlier previous studies. [17, 18]

Based on history alone, diagnosis of perinatal insult as a cause of brain damage is difficult but, positive neuroimaging findings suggestive of ischemia, infection or hypoxia is very important diagnostic evidence. Wiklund et al., [19] reported that the frequency of etilogical diagnosis found PVL to be the cause of hemiplegic cerebral palsy in 37%, congenital brain anomaly in 17% and vascular causes in 16%. A more recent study of hemilpegic cerebral palsy by Humphrey et al., [20] reported that 46.2% were vascular, PVL 34.2%, and brain malformation 12.9%. In our study, we found that the hemiplegic cerebral palsy have similar etiological profile to previous studies, except relatively high percentage of intracranial hemorrhage (25%), and low percentage of PVL (6%) as a cause of hemiplegic cerebral palsy.

Congenital brain anomalies represent disorders in development occurring early in gestation. Since the advent of neuroimaging, it has become more apparent that children with CP may have congenital malformations.

Data from studies done by Yin et al., [21] Jaw et al., [22] and Krageloh-mann et al. [23] found that 7% of patients who had a CT scan and 12% of those underwent MRI had major brain malformation. In our study, 15% of patients who had MRI and 14% who had CT scan had brain malformations most of which had major brain malformations (lissencephaly, schizencephaly and pachygyria). The etiologic categories used in this study are broadly defined and cannot be considered end points in themselves. Our observation that six categories of etiology are responsible for the majority of observed etiologies of cerebral palsy identified, suggests strategies for prevention and treatment. Our study is in keeping with the concept of heterogeneous etiology of this syndrome called cerebral palsy. Research efforts to minimize cerebral palsy needs to address the identification of factors that underlie and modify eventual outcome.[24]

Conclusion

Data from our study reveal that the finding of an abnormal MRI/CT scan in a child with cerebral palsy is high. In order to establish an etiology and prognosis in children with cerebral palsy, neuroimaging is highly recommended.

Abbreviations

CP=Cerebral Palsy.
CNS=Central Nervous System.
PVL=Peri-ventricular Leukomalacia.
MRI=Magnetic Resonance Imaging.
CT= Computed Tomography scan.

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الشلل الدماغي في الأردن: الخصائص الاكلينيكية والشعاعية

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الملخص:

الهدف: تقييم الموجودات الشعاعية لدى المرضى المصابين بالشلل الدماغي وتقييم وجود علامات شعاعية نوعية مميزة لكل نمط من أنماط مرض الشلل الدماغي.

طريقة الدراسة: أجريت الدراسة على عينه مكونة من 158 مريض يعانون من الشلل الدماغي، ويراجعون عيادات امراض الأعصاب والتطور العصبي في مستشفى الملك المؤسس عبدالله الجامعي على مدى عامين (2007–2008) حيث أجريت دراسة راجعة بالاعتماد على سجلات العيادات، والربط مع الموجودات الشعاعية (في التصوير الطبقي المحوري والرنين المغناطيسي).

النتائج: تم شمول عينة ممثلة بـ 158 حالى شلل دماغي في الدراسة كالاتي:

- 84 مريض من العينة كانوا من الذكور(53% من العينة)
- 74 مريض من العنية كانوا من الأناث (47% من العينة)
- 41 حالة من العينة المشمولة بالدراسة كانوا ولدوا قبل تمام الحمل (Preterm) (26%)
- 117 من العينة المشمولة بالدراسة ولدوا بتمام الحمل (full term) (74%)

وشملت الدراسة

1- 112 حالة من الشلل الدماغي التشنجي (spastic CP) وبنسبة (70.8%) مع ملاحظة كون النمط التشنجي الرباعي صور الأشعة (Spastic quadriplegia) 63 حالة من (40%) من العينة.
2- 22 حالة من الشلل الرخو الرنحي (hypotonic ataxic) 14% من العينة.
3- 15 حالة من نمط عسرة الحركة (Dyskinetic CP).
4- 9 حالات من النمط المختلط (Mixed CP).

وتمت ملاحظة تغيرات شعاعية مرضية باستخدام الرنين المغناطيسي في 15 حالة وبنسبة 79% وباستخدام التصوير الطبقي في 113 حالة وبنسة (71 %)، وتم ملاحظة وجود تغيرات شعاعية نوعية تشير الى:

1- نقص الأكسجة الدماغية(Brain Asphyxia) في40 حالة وبنسبة25 %.
2- الآمات الدماغ الولادية في 22 حالة 14%.
3- النزف داخل الجمجة في 10حالات 6%.
4- اسباب دماغية وجمجمية في 29 حالة 88.5 %.
5- تلين الدماغ حول البطينات 30حالة 20%.
6- أسباب غير معروفة ضمور دماغي معزول 26 حالة 16.5%.

وبناءاً عليه فإن العامل المسبب الأكثر شيوعاً في الشلل الدماغي هو نقص الأكسجة الدماغية (Brain Asphyxia) يليه تلين الدماغ حول البطينات (periventricular encephalomalacia)).

وفي هذه الدراسة تم اعتبار الضمور الدماغي موجود غير نوعية، مع العلم بأنها تشاهد بكثرة لدى المرضى المصابين بنمط عسرة الحركة (Dyskinetic CP) وخمسة مرضى من أصل خمسة عشر شملتها 33%، وكذلك في النمط التشنجي الرباعي 15 مريض من أصل 63 مريض شملتها الدراسة 24% مقارنة بالأنماط الأخرى.

النتيجة:

- التأكيد على أهمية دور التشخيص الشعاعي في فهم العوامل المسببة والآلية الأمراضية في والتثبت من أو العوامل المؤثرة والتي يكون لها دور في الاستشارة الوراثية (مثل التشوهات).
- الرنين المغناطيسي هو وسيلة أكثر حساسية من التصوير الطبقي في تشخيص الآفات الدماغية.

الكلمات الدالة: الشلل الدماغي، الموجودات الشعاعية.