

Cerebral Palsy Among Kurdish Children in the City of Dohuk: A Case- series Study

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Abstract

Background: Cerebral palsy (CP) is defined as non-progressive disorder of movement and posture due to brain insult or injury occurring in the period of early brain growth (generally under three years of age). The clinical characteristic of this common problem had not been studied before in any of the Kurdish cities of the north of Iraq.

Objectives: This is a descriptive study of the clinical characteristics of cerebral palsy in the city of Dohuk at the north of Iraq.

Patients and Methods: This is a case series descriptive study conducted in the children rehabilitation unit of Azadi general hospital and Dohuk children's rehabilitation center in the city Dohuk on children with Cerebral palsy. The following information were studied, the first presenting complaint, age distribution, the antenatal illnesses, antenatal drug use, mode of delivery, gestational age, the state at birth, types of CP, associated congenital abnormalities, any other associated problems and the treatment used.

Results: The total number of patients were 100, males were outnumbering females (1.3:1), spastic CP was the most common type (57%). Most of the patient presented between the age of 7-12 months (66%), the most common first presenting complaints were the delayed milestones (50%), most of the patients came from a vaginal delivery (68%), the associated problems were: swallowing problems (68%), walking problems (65%), epilepsy and seizure disorders (60%), speech problems (46%), neonatal jaundice which was developed in 95% of cases, and finally, most of the patient came to parents who got married as a result of consanguineous marriage(68%).

Conclusions: This is the first descriptive study among children with CP from the Kurdish population highlighting certain demographic and clinical characteristics of them.

Keywords: Cerebral palsy, Kurdish children, Dohuk

(J Med J 2009; Vol. 43 (3):205-211)

Received

March 5, 2008

Accepted

June 15, 2008

Introduction

Cerebral palsy (CP) is a diagnostic term used to describe a group of motor syndromes resulting

from disorders of early brain growth (generally under 3 years of age). The manifestation of a given lesion may change as the nervous system

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mature, but the insult that caused the lesion is no longer present and there is no active disease at the time of diagnosis, although it has historically been considered as a static encephalopathy. This term is not entirely accurate because of the recognition that the neurological features of CP often change or progress over time.^{1,2,3,4} This disease was first described by Dr. Little in 1826.⁵

CP is the most common cause of severe childhood disability affecting 1 in 400 children,⁶ the incidence varies from one country to another but it is around 1-2/1000 live birth, and; prevalence is about 500/100000 population.^{7, 8,9,10}

Traditionally, spastic CP has been the most frequent type, accounting for approximately 50% of cases followed by Athetoid CP seen in approximately 20%.^{11,12}

The clinical findings in some affected children appear to evolve and affected children may exhibit a somewhat different distribution of involvement at different ages.¹³

A team of physicians from various specialties, as well as occupational and physical therapists, speech pathologists, social workers, educators, and developmental psychologists provided important contribution to the treatment of these children.¹

This study was conducted to assess the clinical and demographic characteristics of CP children among the Kurdish population at the North of Iraq.

Patients and Methods

The period of the study extended for one year from May 2002 till April 2003 on patients who were visiting the Dohuk children's Rehabilitation Center (DCRC) and Children Rehabilitation Unit of Azadi General Hospital (CRU) for regular physiotherapy and for treatment of any other associated problem. A special data collection sheet was developed for the purpose of this study. One hundred children with a diagnosis of CP were retrospectively studied.

Information was obtained by the means of direct questionnaire of parents and/or from the registration record of DCRC and the birth records of Azadi general hospital. The author had examined all the patients, regarding the other problems associated with CP. The APGAR scoring system (14) was used to assess the newborn delivery score, being good if more than 8, average 3-7, and low if below 3. The ethical committee of azadi general hospital had approved conducting the study. Consent was taken from all children guardians. Data were analyzed using simple statistical analysis packages (Epi info 6).

Results

One hundred children with CP were studied, male accounting for 57% and females for 43%. The most common first presenting complaint was the delayed milestones (50%) as shown in table (1).

Table (1): The first presenting complaint.

Complaint	No.
Delayed milestones	50
Convulsion	14
Difficulty in swallowing	14
Recurrent LRTI	9
Hand preference	8
FTT	5
Total	100

Majority of the patients presented between the age of 7-12 months (66%), other age distributions are shown in figure (1).

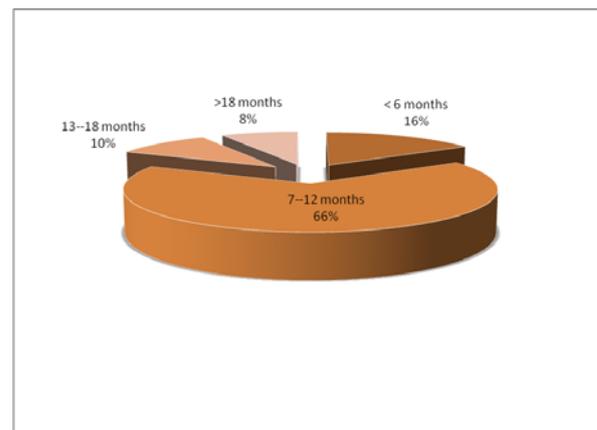


Figure (1): Age of presentation.

Seventy six percent of mothers were attending the antenatal clinics, about 21% of them had pre-eclamptic toxemia.

Sixty eight percent of the patients were delivered by normal vaginal delivery and the other modes of deliveries are shown in figure (2).

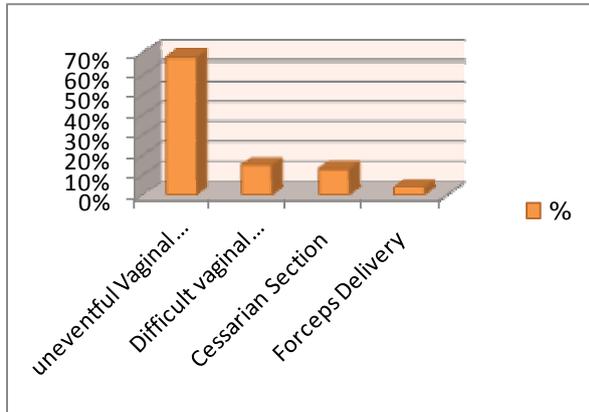


Figure (2): The mode of delivery.

The Apgar score ranged between: good in 48%, average in 39% and low in 13%. History of asphyxia was reported in 48 cases, 45 cases of them had been admitted previously to the NICU; while the remaining 3 cases were home delivery and they had no access to the medical care. Neonatal jaundice developed in 95% of cases, 60% received no treatment, 34% were treated with physiotherapy and 6% required exchanging transfusion.

Spastic CP was the most common type among the studied cases (57%), among the spastic CP; the spastic quadriplegia was the most common (72%); the other types of spastic CP are shown in table (2).

Table (2): Types of CP.

Type	No.
Spastic	57
Quadriplegia	41
Hemiplegia	8
Diplegia	7
Monoplegia	1
Hypotonic	19
Chorio-arthetoid	6
Ataxic	5
Mixed	13
Total	100

Table (3) details the associated congenital abnormalities that were found in 16% of cases and table (4) lists the associated problems.

Table (3): Associated congenital anomalies.

Anomaly	No.
Musculoskeletal	8
Eye	4
Ear	2
Heart	1
Kidney	1
Total	16

Table (4): The other associated problems.

Problem	No.
Swallowing	68
Walking	65
Epilepsy	60
Speech	46
Intellect	42
Vision	32
DDH	31
Behavior	29
Hearing	10

Discussion

Similar to many previous reports, there is slight predominance of males compared to females (57% vs. 43%).^{12,15,16} The main reason of first presentation to any of the health centers in this study was the delayed motor developmental skills (50%). Certain developmental observations were often made first by the parents; suggesting a possible diagnosis of CP, the most frequent one being the delayed motor development often with a dissociation between motor and intellectual development.³

Similar to previous reports, the commonest type of CP is the spastic quadriplegia that has been reported to occur in up to 80%^{17, 18}. Both hand preference and difficulty in walking signify the underlying type of spasticity. One hand preference (especially below one year of age) signifies one side of the body involvement and the later signify spastic hemiplegia.¹

The last presenting complain in this study was the head lag which is the most common presenting feature of chorio-athetosis complicating bilirubin

encephalopathy, as hypotonia predominate in the first year of life then abnormal movements, as athetosis, occur later as myelination will have completed in the next 2 years.¹⁹

Majority of the patients were children of parents who got together by consanguineous marriage; which may explain a genetic or familial predisposition.²⁰

Early diagnosis of CP had been done in 18% of cases below the age of 6 months because they were severe enough in addition to the availability of neuroimaging.²¹

The vast majority of patients were diagnosed between the ages of 7-12 months (66%) because most of the CP patients are diagnosed by the end of the first year of life.¹⁶

Improvement of the antenatal care and follow up of mother with high risk pregnancies did not reduce the total number of CP suggesting a non-obstetrical cause for CP.¹⁶ This also had been observed here because 76% of cases attended the antenatal clinic but still they had ended with babies with CP which may be explained by many reasons; the low availability of equipments for the sake of early diagnosis and detection, miss-management during intra-partum period because of the lack of facilities necessary for fetal management, or because most of the CP cases are developmental in nature.¹

Majority of patients were delivered by normal vaginal delivery, while 15% were products of prolonged and difficult labor which is a known factor for the development of CP due to oxygen deprivation in the birth canal, and the possibilities of complications especially intracranial hemorrhage; which is also a risk factor for those 5% products of forceps delivery.¹⁷ 13% of patients were delivered by the cesarean section which did not prevent the future development of CP, this may be explained in part that in about 70% of cases of CP brain damage occurs before birth, although in the majority of cases it occurs around the time of delivery, or in the first month of life. In many cases, the cause of CP is not known yet.²²

Thirty six percent of patients were premature, which is an important risk factor especially when associated with low birth weight, primarily because of intra-cerebral hemorrhage and periventricular leukomalacia.¹

Six patients only were term; of whom 5 were babies of diabetic mother's .CP occurrence in these patients may be related to many factors such as macrosomia and related birth injuries, placental insufficiency, attacks of hypoglycemia and the increased incidence of congenital malformations in these babies.¹

Forty eight patients sustained asphyxia, of which 45 underwent resuscitation and were admitted to NICU. The APGAR score does not predict the neonatal mortality or subsequent CP and indeed the score is normal in most patients who subsequently developed CP although a score of 0-3 at 20 minutes is associated with high mortality and morbidity.¹ Still, 20% of children with CP who had a normal birth weight and no recorded congenital malformation at birth had a 5 minute APGAR score below 7.²³

The high prevalence of birth asphyxia in out locality may have an effect on the growth and the development of the child.^{24, 25}

Majority of patients had history of neonatal jaundice which may develop in 60% of term and 80% of premature infants.¹

The most common type of CP encountered in the clinical practice is the spastic one ranging from 40% (14) up to 60%¹⁷ followed by the mixed type 9-22%.¹³

The high prevalence of quadriplegia may be due to the high prevalence of birth asphyxia.^{19, 12, 26, 25} Associated congenital malformations were comparable to other studies.²⁷

Other problems associated with CP were comparable to the published studies with the hearing problem being less.³ This is the first study highlighting the clinical profiles of CP children in Kurdish population.

References

1. Johnson MV. Cerebral palsy In: Kliegman RM, Behrman RE, Jenson HB, Stanton BF. Editors. Nelson textbook of pediatrics. 18th ed. USA: saunders; 2008. p. 2494-2495.
2. Richard WI cooke. Trends in incidence of cranial U/S lesion in cerebral palsy in very low birth weight infants 1982-1993, arch dis childh. 1999; 80 (2).
3. Eileen PC vinnig. Cerebral palsy In: Kenneth BR, editor. Manual of clinical problems in pediatrics. 3rd ed. Boston: Little and Brown; 1988; 294-297.
4. Pharoah POD, Price TS, Plomin R. Cerebral palsy in twins: a national study, arch dis child. 2002; 87(2).
5. Haslam RHA. Cerebral palsy In: Behrman RE, Kliegman RE, Jenson HB. Editors. Nelson textbook of pediatrics. 16th ed. Philadelphia; saunders; 2000; 1843-1845.
6. Ubhi T, Bhakta BB, Ives HL, Allgar V, Roussounis SH. Randomized double blind placebo controlled trial of the effect of botulinum toxin on walking in cerebral palsy. arch dis child. December 2002; 83(6).
7. Sulkes SB. cerebral palsy In: Behrman RE, Kliegman RM. Editors. Nelson essentials of pediatrics. 3rd ed. Philadelphia: saunders; 1998; 50-52.
8. Redihough DS, Collins KJ. The epidemiology and causes of cerebral palsy. Aust J of physiother 2003; 49 (1).
9. William HB. Prevention of disability In: Maxcy, Rosenau, Last. Editors. Public health and preventive medicine. 14th ed. Connecticut: Appleton and Lange Stamford; 1998; 1059-1064.
10. Bhatia m, Joseph B. Rehabilitation of cerebral palsy in developing countries: the need for comprehensive assessment (abstract). pediatr rehabil 2000; 4 (2).
11. Shapiro BK, Capute AJ. Cerebral palsy In: McMillan JA, DeAngelis CD, Feigin RD, Warshaw JB, editors. Oski; s pediatrics principles and practice. 3rd ed. Philadelphia: LIPPINCOTT WILLIAMS and WILKINS; 1999; 1910-1917.
12. Baker KA. Cerebral palsy among Iraqi children: a case series-study. Dohuk med j 2008; 1 (1).
13. Karin BN, Kenneth FS, Barry FS. Cerebral palsy In: Kenneth FS. Editor. Pediatric neurology principles and practice. 2nd ed. Philadelphia: Mosby; 1994; 471-487.
14. Steven AR. Resuscitation in the delivery room In: Joh PC, Eric EE, Ann RS. Manual of neonatal care. 5th ed. Philadelphia: LIPPINCOTT WILLIAMS and WILKINS 2003; 57-69.
15. Hannah Kinney. hypotonia, children hospital of Boston. [http://web1tch.harvard.edu/cfapps/AZZ/copyright december 2001](http://web1tch.harvard.edu/cfapps/AZZ/copyright%20december%202001).
16. Colin F, Tim M, Richard N. Cerebral palsy In: McIntosh N, Helms PJ, Smyth RL, editors. For far textbook of pediatrics. 6th ed. London: Churchill livingstone 2003; 968-979.
17. Bethesda M. Cerebral palsy: hope through research. National institute of neurological disorders and stroke. 1st July 2001.
18. Ashutosh G, Patwari AK, Promila Bajaj, Ravi Kashyapand. Gastro-esophageal reflux disease in children with cerebral palsy. Indian J Pediatr. July 2001; 38. <http://www.indianpediatrics.net/July2001/current.htm>.
19. Boosara Ratanawangsa. cerebral palsy in children. 12th feb. 2001. www.emedicine.com/emerg/byname/pediatrics/limp.htm.
20. Terzidou V, Bennett P. Maternal risk factors for fetal and neonatal brain damage. Biol Neonate 2001; 79(3-4).
21. Kuban KCK, Alan Leviton. Cerebral palsy. N Eng J Med. 1994; 330 (3).
22. Alfred IW. Delaware M. gait and clinical movement analysis. Dupont institution. Dupont Hospital for children. <http://gait.aidi.udel.edu>.
23. Dag master, Rolve T, Laventz MI, Tor Blerkedal, Trond Markestad. The association of APGAR scores with subsequent death and cerebral palsy: a problem based study in term infants. J Pediatr. 2001; 138(6).
24. Helen Harrison. Risk factors for cerebral palsy and developmental problems related to prematurity. Copyright June 2002. <http://prematurity.org>.
25. Al-Omeri W. Cerebral palsy an almost cleared old obstetrician's stigma. The abstract book of the scientific committee of Iraqi medical association, 2002.
26. Rose J, Wolf DR, Jones VK, Black DA, Ochler JW, Gamble JG. Postural balance in children with cerebral palsy. Dev Med Child Neurol 2002; 44.
27. Lisa AC, Judith KG, Cynthia JC, Karen BN. Congenital anomalies among children with cerebral palsy; more evidence of prenatal accidents. J Pediatr. 2001; 138 (6).

الشلل الدماغي لدى الأطفال الأكراد في مدينة داهوك: سلسلة من الدراسات

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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 عينة مكونة من 158 مريضاً يعانون شللاً دماغياً، ويراجعون عيادات أمراض 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 حالة شلل دماغي في الدراسة كالتالي:

- 84 مريضاً من العينة كانوا من الذكور (53% من العينة).
- 74 مريضاً من العينة كانوا من الإناث (47% من العينة).
- 41 حالة من العينة المشمولة بالدراسة ولدوا قبل تمام الحمل (Preterm) (26%).
- 117 حالة 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 الرنجي (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، وبنسبة (71%). كما تمت ملاحظة وجود تغيرات شعاعية نوعية تشير إلى:

1- نقص الأكسجة الدماغية (Brain Asphyxia) في 40 حالة، وبنسبة 25%.

2- الآمات الدماغ الولادية في 22 حالة، وبنسبة 14%.

3- النزف داخل الجمجمة في 10 حالات، وبنسبة 6%.

4- اسباب دماغية وجمجمية في 29 حالة، وبنسبة 88.5%.

5- تلين الدماغ حول البطينات 30 حالة، وبنسبة 20%.

6- أسباب غير معروفة، مثل: الضمور الدماغي المعزول، في 26 حالة، وبنسبة 16.5%.

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

أضف إلى ما تقدم أنه تمّ في هذه الدراسة اعتبار الضمور الدماغى موجوداً غير نوعى، علماً أنه يُشاهد بكثرة لدى المرضى المصابين بنمط عسرة الحركة (Dyskinetic CP)، وشملت الدراسة خمسة مرضى من أصل خمسة عشر، بنسبة 33%، كما شملت في النمط الشنتنجى الرباعى 15 مريضاً من أصل 63، وبنسبة 24% مقارنة بالأنماط الأخرى.

النتيجة: تأكيد أهمية دور التشخيص الشعاعى في فهم العوامل المسببة والآلية الأمراضى، والتثبت من العوامل المؤثرة التي يكون لها دور في الاستشارة الوراثية (مثل التشوهات).

الرنين المغناطيسى هو وسيلة أكثر حساسية من التصوير الطبقي في تشخيص الآفات الدماغية.

الكلمات الدالة: الشلل الدماغي، الموجودات الشعاعية، الأكراد، داهوك.