ORIGINAL

PROF-1023

RELATIONSHIP OF TYPE OF CEREBRAL PALSY WITH THE ETIOLOGY



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ABSTRACT... <u>malikshaheer@hotmail.com</u> **Objectives:** To determine the relationship of etiology with the type of cerebral palsy in children. **Study Design:** Descriptive case series. **Setting:** Department of Paediatrics, Allied Hospital, Faisalabad. **Period:** From January 2002 to December 2004. **Patients and Methods:** 120 children from 1-12 years of age of either sex who presented in Paediatric Department with abnormalities of tone, posture and movement and subsequently diagnosed as cerebral palsy on the basis of history, physical examination and investigations, were included. **Results:** Out of 120 cases the majority had spastic CP, 72% (n = 86) such as quadriplegic, diplegic and hemiplegic types. The spastic quadriplegia was mainly associated with birth asphyxia and meningoencephalitis. Prematurity and low birth weight were the major contributors towards diplegic CP, while spastic hemiplegia although less common was caused by meningoencephalitis in 5 cases and intra cranial bleed and asphyxia in 3 cases each. Atonic or hypotonic CP found in 23 cases, were caused by meningoencephalitis, kernicterus, birth asphyxia and prematurity. 10 cases of atonic CP did not have any reason (hence idiopathic). Athetoid CP was mainly due to kernicterus, meningoencephalitis and asphyxia. Ataxic and mixed types of CP were present in 3 cases each and were due to meningoencephalitis and birth asphyxia.

Key words: Cerebral palsy, Children, Hypoxic ischemic encephalopathy, bilirubin encephalopathy, Post meningitic sequelae.

Abbreviations used: CP Cerebral palsy, QP: quadriplegia, DP: diplegia, I/C: intracranial, I/V: intra-ventricular and PV: peri-ventricular.

RELATIONSHIP OF TYPE OF CEREBRAL PALSY

INTRODUCTION

Cerebral palsy is a non-progressive disorder of movements and posture resulting from an insult to the growing brain usually in first 2 years of life¹. The overall prevalence is although 2.5 per 1000 but may vary from 1 to 6 per 1000^{2,3}. Traditionally the most frequent CP is spastic followed by extra pyramidal manifesting in the form of athetosis, ataxia and tremors^{4,5,6}.

The major etiological factors of cerebral palsy are asphyxia, intracranial infection, birth trauma, prematurity, kernicterus and intracranial bleed⁴. Improved obstetric techniques have markedly diminished major birth trauma and the successful prevention of hemolytic disease of newborn has decreased extra pyramidal cerebral palsy of choreoathetoid type^{7,8,9}. Prematurity, low birth weight and placental dysfunctions are becoming major factors in recent years due to the increased survival of newborns¹⁰.

The data about cerebral palsy, its prevalence and its relationship to etiological factor is very scanty in our country. This study focuses and highlights the relationship between the different types of CP to various etiological factors mentioned above.

PATIENTS AND METHODS

A total of 120 children from 1 to 12 years of age of either sex with CP seen in in-patient and out-patient sections of department of Paediatrics at Allied Hospital Faisalabad during the 2002-2004 were included in the study. Children under 1 year of age were excluded as developmental milestones are not fully established under one year of age. Children with storage disorders, degenerative brain diseases, myopathies and neuropathies were also excluded.

A detailed history was taken from the mother or care giver using a pre-structured proforma. Obstetric history including place and mode of delivery, and whether conducted by traditional birth attendants, LHVs or doctor were recorded. Birth weight and gestational age record if available was noted. For a child to be labelled as birth asphyxia, APGAR scores when available were considered. In the absence of these the following criteria were used (i) history of delayed cry >5 min after birth (ii) baby turning blue and requiring oxygen therapy and having difficulty in respiration, lethargy and / or seizures within 72 hours of birth.

Meningoencephalitis was considered in patients with history of fever, fits and unconsciousness and proven diagnosis on CSF examination. Kernicterus was suggested by the history of deep jaundice that needed photo therapy and / or exchange transfusion along with abnormal neurological findings in the neonatal period. Intracranial bleed was considered in patients with history of birth trauma, head injury or bleeding disorder alongwith CT scan finding of old hemorrhage.

Complete physical, developmental and neurological examination including fundoscopy were done in all patients. Hearing was assessed on clinical grounds by asking from mother and by distraction hearing test.

The investigations done in selected cases were X-Ray skull to rule out craniosynostosis and TORCH infections, C.T scan of head for cortical atrophy, peri-ventricular leukomalacia and old hematoma. Data was entered in FoxPro and descriptive statistics were calculated using SPSS soft ware version 10.

Table I Type of cerebral palsy							
Туре	No of cases	%age					
PYRAMIDAL	86	-					
Spastic quadriplegia	56	65%					
Spastic diplegia	15	17%					
Spastic hemiplegia	11	13%					
Mixed	4	5%					
EXTRA PYRAMIDAL	34	-					
Atonic	23	67%					
Athetoid	8	24%					
Ataxic	3	9%					

RESULTS

Out of 120 cases of CP, spastic CP was found in majority

i.e 72%(n=86) while remaining 28% (n=34) cases of CP were of extra-pyramidal type. The break up is shown in Table I.

Table II Etiology of cerebral palsy							
Diagnosis	No of cases	%age					
Birth Asphyxia	43	36%					
Meningoencephalitis	41	34%					
Prematurity and low birth weight	10	8%					
Kernicterus	6	5.5%					
Intra cranial bleed	3	2.5%					
Idiopathic	17	14%					
Total	120	100%					

Birth asphyxia, meningoencephalitis, prematurity and low birth weight, kernicterus and intracranial bleed were important etiologies accounting for 36%, 34%, 8%, 5.5% and 2.5% respectively. No cause was identified in 14% cases of CP. (Table II). This study was conducted to determine the relationship of etiology with the type of CP. Spastic quadriplegic CP was caused mainly by birth asphyxia (48%) and meningoencephalitis (43%). No cause was found in remaining 9% cases of spastic quadriplegic CP.

Prematurity and low birth weight were the major contributor towards diplegic CP present in 53% cases while remaining 34% cases of diplegic CP was caused by birth asphyxia and meningoencephalitis (27% and 7% respectively). No cause was identified in 13% cases of diplegic CP. Hemiplegic CP a less common cause of spastic CP, caused by meningoencephalitis, birth asphyxia and intracranial bleed in 44%, 28% and 28% respectively. Atonic CP was related to meningoencephalitis, kernicterus, prematurity and low birth weight and birth asphyxia in 26%, 13%, 9% and 9% respectively while etiology remained unknown in 43% cases of atonic CP.

Athetoid CP was due to kernicterus and meningoencephalitis in 38% cases each and due to birth asphyxia in remaining 24% cases. Ataxic and mixed CP was caused by meningoencephalitis and birth asphyxia (Table III).

Table III Relationship of type and etiological factors of cerebral palsy															
Etiology	Spastic QP					Spastic Mixed Hemiplegia		Atonic		2	Athete	Athetoid		Ataxic	
	N	%	N	%	Ν	%	Ν	%	Ν	%	Ν	%	Ν	%	
Birth Asphyxia	27	48	4	27	3	28	3	75	2	9	2	24	2	67	
Meningoencephalitis	24	43	1	7	5	44	1	25	6	26	3	38	1	33	
Prematurity and Low Birth Weight	-	-	8	53	-	-	-	-	2	9	-	-	-	-	
Kernicterus	-	-	-	-	-	-	-	-	3	13	3	38	-	-	
Intracranial Bleed	-	-	-	-	3	28	-	-	-	-	-	-	-	-	
Idiopathic	5	9	2	13	-	-	-	-	10	43	-	-	-	-	
Total	56	100	15	100	11	100	4	100	23	100	8	100	3	100	

DISCUSSION

The preponderance of spastic CP in this study is similar

to that reported by others¹¹ but the distribution of subtypes was different. Although spastic diplegia is

generally the commonest form reported from developed countries¹² but in this series spastic QP was most commonly observed similar to other developing countries⁴. and was related mainly to birth asphyxia and intracranial infections, probably due to more extensive damage to cerebral cortex as a result of delayed and poor access to medical facilities, improper treatment and lack of routine Hib immunization.

The role of birth asphyxia in the causation of CP has been challenged¹³. However it was the major causative factor in our study similar to studies from other developing countries^{4,14}. Occurrence of severe birth asphyxia continues to be a major problem in many developing countries where obstetric facilities are virtually non existent for a vast majority of women in rural areas.

The incidence of spastic quadriplegic CP and hence total number of CP cases can be lowered by conducting high risk deliveries at properly equipped hospitals or clinics, good antenatal control of diabetes and hypertension alongwith timely anticipation and management of cephalopelvic disproportion. As more than 80% deliveries are conducted at home by untrained TBAs, training and monitoring of TBAs for neonatal resuscitation and better neonatal care will also reduce the incidence of post asphyxial CP.

Spastic CP due to meningoencephalitis can be reduced by early diagnosis and prompt treatment of CNS infections and use of cost effective routine immunization against H. influenza type b.

Several studies have reported a significant association between low birth weight and diplegic CP, ^{(15), (16), (17)} similar to our study and is related to peri-ventricular leukomalacia because of I/V and P/V hemorrhage damaging costicospinal tract supplying lower limbs.

In developed countries, a progressive decrease in spastic QP and a relative increase in diplegic CP has been attributed to the decrease in perinatal mortality rate, with increasing survival rates of extremely premature infants. This situation has not yet been achieved in our country except in a few tertiary care centers.

While athetiod CP, particularly secondary to neonatal hyperbilirubinemia, has virtually disappeared from many parts of the world^{18,19}. It still constitutes a significant proportion of CP cases in our set up in 5.5% of total cases of CP and 38% cases of athetiod CP, similar to that reported by Singhi PD et al⁴ and those reported from other developing countries^{14,20}.

The reason could be delayed referral from general practitioners and lack of awareness regarding blood groups incompatibility of mothers and babies. Other factors like asphyxia, acidosis, prematurity and septicemia also contribute to kernicterus. With the early diagnosis and aggressive management of hyperbilirubinemia, the incidence of cerebral palsy due to kernicterus has decreased over the recent years. The etiology remained unknown in 14% cases, probably due to limited laboratory facilities and uncertain history.

CONCLUSION

As etiology is evident in most cases of cerebral palsy and determines the specific type of CP. Knowing the association of etiology with the type of CP, special efforts can be done for important risk factors, thereby reducing the frequency of sub types and hence total number of cerebral palsy. This can be achieved by improving obstetric techniques thereby reducing the incidence of birth trauma and birth asphyxia.

Awareness of blood group incompatibilities and early recognition and timely management of jaundiced newborn can decrease the incidence of post kernicterus athetoid CP. Routine immunization against H-influenza B and pneumococcal infections is strongly recommended to prevent bacterial meningitis and subsequently QP CP.

REFERENCES

 Neimann G, Michaelis R. Cerebral palsy II. Clinical symptoms and etiopathogenesis. Klin Pediatr. 1996 Sept. Oct; 208(5); 280-4.

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- 2 Bottos-M; Granato-T; Allibrio-G. **Prevalence of cerebral palsy.** Ger-Med-Child-Neurol. 1999. Jan; 41(1): 26-39.
- 3 Koterazawa-K; Nabetani-M; Miyata-H; Kodama-S; Takada-S. Incidence of cerebral palsy. No-To-Hattatsu.1998 Nov; 30(6): 489-93.
- 4 Singhi PD, Ray M, Suri G, Clinical Spectum of cerebral palsy in North India. An analysis of 1000 cases. J Trop Pediatr 2002 Jun; 48(3) 162-6.
- 5 Piecuch-Rt; Leonard-CH; Cooper-BA; Kilpatrick-SJ; Schlueter – MA; Sola-A. Cerebral palsy epidemiology. J-Paediatr- Neurol. 1997 Nov; 90(5): 809-14.
- 6 Spinillo-A; Fazzi-E; Capuzzo-E;Stronati-M; Piazzi-G; Ferrari-A. Review of cerebral palsy: Description, incidence and etiology. J-Pediatr-Neurol. 1997 Oct; 90 (4): 519-23.
- 7 Lindstrom-K and Bremberg-S. Cerebral palsy epidemiology. ActaPaediatr. 1997 July; 86(7): 736-9.
- 8 Yoon-BH; Jun-JK; Romero-R; Park-KH; Gomez-R; Choi-JH; Kim-IO. Risk factors for cerebral palsy. Am-J-Obstet-Gynecol. 1997 July; 177(1): 19-26.
- 9 Greth-JK and Nelson-KB. Cerebral palsy epidemiology. JAMA. 1997 July 16; 278(3): 207-11.
- 10 Hagberg B, Hagberg G, Zetterstrom R. Decreasing perinatal mortality – increasing in cerebral palsy morbidity. Acta pediatr Scand 1989; 78:664-70.
- 11 Mutch-L, Alberman E et al. Cerebral palsies epidemiology; where are we now and where are we going? Dev.Med Child Neurol 1992; 34: 547-551.
- 12 Sarah Winter, M.D and Andrew Autry et al. Trends on the

prevalence of CP in a population based study. Journal American Academy of Pediatrics. 2000 Dec; 110: 1220-1225.

- 13 Nelson KB, Ellenberg JH. Antecedants of cerebral palsy. Multivariate analysis of risks. New Engl J Med 1986; 315:81-6
- 14 Haque KN. Cerebral palsy in Riyad, Saudi Arabia. Pak Pediatr J 1986; 10: 1-12
- 15 Suzuki-H. Gestational age; birth weight, degree of motorimpairment and causes of brain damage in sixty eight patients with cerebral palsy identified in a community (birth year 1985-1991). No-To-Hattatsu 1997 Jan; 29(1): 27-32.
- 16 Bhushan V, Paneth N et al. Impact of improved survival of very low birth weight infants on recent secular trends in the prevalence of cerebral palsy. Paediatrics 1993; 91: 1094-1100.
- 17 Topp-M; Langhoff-Roos-J; Uldall-P; Pre-term birth and cerebral palsy. Predictive value of pregnancy complications, mode of delivery and Apgar scores. Acta-Obstet-Gynecol-Scand. 1997 Oct; 76(9): 843-8.
- 18 Davis-DW. **Review of cerebral palsy.** Neonatal-Netw.1997 Jun; 16(4): 19-25; quiz 26-9.
- 19 Gaffney G-et at. **Risk factors for cerebral palsy.** Archives of disease in childhood 1996; 72: 361048.
- 20 Khan-NZ; Ferdous-S; Munir-S; Huq-S; McConachie-H; Mortality of urban and rural young children with cerebral palsy in Bangladesh. Dev-Med-Child-Neurol. 1998 Nov; 40(11): 749-53.