

Cerebral palsy: a study in IPGM & R, Dhaka

Dr. F.C. Gami¹

Dr. S.M. Shahnawz Bin Tabib²

Prof. Md. Nurul Islam³

Abstract

This prospective study was carried out on 60 children attending follow-up clinic of Paediatric Neurology and Child Development in the out-patient department of Institute of Post Graduate Medicine and Research (IPGM & R), Dhaka, from May 1995 to April 1996. The present study was conducted to find out various etiological factors responsible for cerebral palsy. Evaluation of 60 cases of cerebral palsy was made by taking detailed history and physical examination. In history, special attention was given to find out antenatal, natal & postnatal factors which predispose to or is directly responsible for the occurrence of cerebral palsy. Of these 60 cases, 38 (63.33%) patients were male and 22 (36.36%) female children. 75% of the cases were of spastic type of cerebral palsy. Hypotonic cerebral palsy was observed in 11.66%; ataxic type in 5%; athetoid in 3.33%; mixed spastic and ataxic in 3.33%, and dyskinetic type of cerebral palsy in one case. History of consanguinity of marriage was found in 23.33% cases. Natal and prenatal factors were most common etiological factors. Among the natal factors birth, asphyxia was found in 55%; prematurity in 6.66%, and sepsis in 5%. Antenatal maternal illness was found in 50% cases including toxemia in 10%; intrauterine infection in 10%, prolonged and obstructed labour in 28.32%. Postnatal factors were found in 30% of cases including hyperbilirubinemia.

The results of this study emphasize the need to reinforce the existing antenatal, natal, obstetric and neonatal services for early and proper management of these conditions and to reduce the incidence and severity of cerebral palsy.

Keywords: IPGM & R; cerebral palsy.

Introduction

Cerebral Palsy is usually the first identifiable developmental disability. It is often an important marker for other developmental problems that may coexist but are not yet manifest.¹ In 1862 William John Little, an orthopaedic surgeon in London, presented his observations of a group of children with tonal and developmental abnormalities which he described as "Spastic rigidity".² Little postulated that the motor defects resulted directly from difficulties in the birth process. This opinion was widely held for over a century. Yet there were early critics, chief among them was Sigmund Freud, who speculated that perinatal difficulties were the result of preexisting abnormalities in the foetus rather than the cause of cerebral palsy.³ The child suspected of having cerebral palsy deserves not only an investigation for etiology but it is necessary to include statements about motor, cognitive, sensory and behavioural aspect of the patients and the presence or absence of epilepsy for comprehensive evaluation to detect impairment in other CNS functions. The aims of this study were to identify types of cerebral palsy, to find out various etiological factors and to identify developmental defects and problems associated with cerebral palsy.

Materials and method

Sixty children in the age group of 3 months - 12 years attending follow-up clinic of paediatric neurology and child development in the out-patient department of IPGMR, Dhaka from May 1995 to April 1996, were the study population. This study was done prospectively. Every child was seen by the authors. History was taken carefully and thoroughly with special attention to pregnancy history, birth history, elaborate pre, peri and post natal history, any major illness and immunization. Family history included history of consanguinity, sibling's death, affected siblings and other family members.

Neurodevelopment history included developmental milestones and any congenital abnormalities. Physical examination was done meticulously on all children including neuro developmental examination and examination of special sensory functions like vision, hearing and speech. Psychological assessment was done by developmental psychologist. Audiometry and retinal examinations were done when necessary. Their clinical data were collected in a prospective way on a specially designed proforma. Appropriate investigations like EEG, ultrasonography of Brain, CT scan, biochemical investigations were done where required and subject to availability of resources. Each case was managed, as far as possible, with special attention to physical training therapy, drug therapy and other multidisciplinary approaches and followed-up prospectively.

Results

Table I and II show that male children were predominant among cerebral palsy patients. Most (50%) were under two years of age. 75% of the patients presented with various forms of spastic cerebral palsy followed by hypotonic type (11.66%) (Table III).

Table I: Sex distribution.

<i>Sex</i>	<i>Number</i>	<i>Percentage</i>
Male	38	66.33
Female	22	36.66
Ratio		1.85:1
Total	60	100

Table II: Age distribution.

<i>Age</i>	<i>Number</i>	<i>Percentage</i>
<6 month	6	10
6 month-<1 yrs	15	25
1 yr-1 1/2 yrs	5	8.34
1 1/2 yrs-2 yrs	6	10
2 yrs-2 1/2 yrs	5	8.34
2 1/2 yrs-3 yrs	4	6.66
3 yrs-3 1/2 yrs	6	10
3 1/2 yrs-<4 yrs	2	3.33
4 yrs->4 yrs	11	18.33
Total	60	100

Table III: Types of Cerebral palsy.

<i>Types</i>	<i>Number</i>	<i>Percentage</i>
A. Spastic	45	75
I. Diplegia	30	50
II. Tetraplegia	11	18.33
III. Hemiplegia	4	6.66
B. Hypotonic	7	11.66
C. Ataxic	3	5
D. Athetoid	2	3.33
E. Mixed spastic and ataxic	2	3.33
F. Dystonic (Rigidity)	1	1.66

Consanguinity of marriage was present in 23.22% cases (Table IV). Table V depicts various causes of cerebral palsy in this study.

Intrauterine infection, toxemia of pregnancy, prolonged labour were predominant prenatal causes. Birth asphyxia topped the list (55%) among prenatal causes and septicaemia in post natal causes.

Table IV: History of consanguinity of marriage.

<i>Consanguinity</i>	<i>Number</i>	<i>Percentage</i>
Present	14	23.23%

Table V: Causes of cerebral palsy.

Prenatal:	<i>Number</i>	<i>Percentage</i>
Family history	5	8.34
Intrauterine infection	6	10
Toxemia of pregnancy	6	10

Anaemia of mother	1	1.86
Labour & delivery:		
Complications of labour and delivery		
Prolonged labour:	13	21.66
Obstructed labour:	4	6.66

Table VI: Causes of cerebral palsy (Continuation).

Prenatal:	<i>Number</i>	<i>Percentage</i>
Birth asphyxia	33	55
Prematurity	4	6.66
Sepsis/CNS infection	3	5
Birth trauma	3	5
Postnatal:		
Hyperbilirubinemia	4	6.66
Septicaemia	11	18.33
Childhood:		
Meningitis	3	5

It is obvious from Table VI that defects in speech, hearing, vision, cognition and epilepsy were the common associated developmental defects and problems.

Table VII: Associated developmental defects and problems.

<i>Defects</i>	<i>Number</i>	<i>Percentage</i>
Speech	30	50
Hearing	14	23.33
Vision	10	16.66
Cognition	13	21.88
Epilepsy/Convulsion	18	30

Discussion

Cerebral palsy is the term used to describe a collection of non progressive disorders that manifest as abnormalities of motion and posture and result from an injury of central nervous system sustained in the early period of Brain development.¹ Although by definition, cerebral palsy is non progressive, its manifestations can change over time reflecting the effects of growth and development on the child. This study analysed etiology and other profiles of neurologically impaired cerebral palsy children. Epidemiological study of childhood disability shows equal involvement in both sexes.⁴ This study shows male preponderance. KB Nelson and JH Ellenberg showed that in 43% of cases, the diagnosis of cerebral palsy is made by 6 months and in 70% by 1 year.^{6,7} In our study, more than 50% children were below 2 years. In this study, spastic cerebral palsy was the commonest form and spastic diplegia being a common pattern followed by Tetraplegia. Nelson K and Ellenberg JH in their study found spastic cerebral palsy the most common form, but the most common patterns are quadriplegia (27%).⁷ Overall, the empirical risk figure of recurrence of cerebral palsy in an affected family is about 10%.⁸ In this study, family history was found in 8.34% cases and history of consanguinity in 23.23% parents. Several investigators have suggested that prenatal events not evident at birth maybe responsible for cerebral palsy.⁹ Paneth notes that in many series of infants with cerebral palsy, there is a more frequent than expected prenatal history of maternal disorders likely to interfere with normal foetal nutrition/oxygenation such as abnormal uterine bleeding/toxemia, anaemia and placental infarction.¹⁰ In our study, there is a

history of intrauterine infection/toxemia and anaemia of mother and complications of labour and delivery. In this study, 55% children have a history of birth asphyxia. In several studies, it has been seen that birth asphyxia as a cause of cerebral palsy varies from 10% to 57%.^{11,12,13} In a study by U. Sharma, birth asphyxia (43%), prematurity (25%) and low birthweight were the predominant causes of cerebral palsy.¹⁴ In addition to problems with movement and posture, almost all children with cerebral palsy have at least one disability associated with damage to the CNS.¹⁵ Sensory defects include vision and hearing problems.¹⁶ In a study by Cohen ME *et al*, hearing loss occurred in 10% of children with cerebral palsy.¹⁷ In our study 23.23% of children has hearing loss.

Expressive and receptive language disorders often parallel the cognitive impairments in affected children.¹⁸ In this study, 50% of children has speech defect. In a study by Aksu F, approximately one third of children with cerebral palsy develop a seizure disorder.¹⁹ This is consistent with our study where convulsion was present in 30% cases.

Conclusion

It can be concluded that cerebral palsy, the first identifier of developmental disability, is common in our country. This study emphasizes the need to reinforce the existing antenatal, natal, obstetric and neonatal services for early and proper management of these conditions and to prevent development of cerebral palsy. Better directed multidisciplinary therapy with the goal of maintaining the child potential is the mainstay of therapy.

References

1. Peggys Eicher, Batshaw Mark L. Cerebral palsy. *Paediatric clinics of North America*. June 1993; **40** (3): 537-549.
2. Little WJ: On the influence of abnormal parturition, difficult labours, premature birth and asphyxia neonatorum, on the mental and physical condition of the child especially in relation to deformities. *Transactions of the obstetric society of London*. **3**: 293-344, 1861-1862.
3. Freud S: *Infantile cerebral paralysis* (1897) (L. Russin Trans). Coral Gables, FL University of Miami Press.
4. Zaman S *et al*. Validity of the Ten Questions for screening serious childhood disability: results from urban Bangladesh. *International J Epidemiology* 1990. **3** (ds): 613-620.
5. Nelson KB, Ellenberg JH. Children who "out grow" cerebral palsy. *Paediatrics* 1982; **69**: 529-536.
6. Piper Mc, Mazer B, Silver KM *et al*. Resolution of neurological symptoms in high risk infants during the first two years of life. *Dev Med child Neurol* 1988; **30**: 26-25.
7. Nelson KG, Ellengerg JH. Epidemiology of cerebral palsy. *Adv. Neurol* 1978; **19**: 421-435.
8. Hughes I, Newton. Genetic aspects of cerebral palsy. *Dev Med Child Neurol* 1992; **34**: 80-86.
9. Coorseen EA, M Sall ME, Duffy LC. Multiple minor malformations as a marker for prenatal etiology of cerebral palsy. *Dev Med Child Neurol* 1991; **33**: 730-736.
10. Paneth N, Kiely J. The frequency of cerebral palsy: A review of population study in industrialized nations since 1950. *Clinics in Developmental medicine* 1984; **87**: 46-56.
11. Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Multivariate analysis of risk. *N Engl J Med* 1986; **315**: 81-86.
12. Nelson KG. What proportion of cerebral palsy is related to birth asphyxia *J paediatr* 1988; **112**: 572-573.
13. Khatoon SA. Out come and long term sequel of birth asphyxiated newborn. *Bangladesh J Child Health* 1994; **15** (314): 66-70.
14. Sharma U. Etiology of cerebral palsy. Scientific abstract, 8th Asian congress of Paediatrics 1994 February, 6-11, P-54.
15. Jones M. Differential diagnosis and natural history of cerebral palsied child. In samilson R (ed). *Orthopaedic aspects of cerebral palsy* Philadelphia J B Lippincot 1975.
16. Sckenk Root Lieb *et al*. The prevalence of cerebral visual disturbance in children with cerebral palsy . *Dev Med Child Neurol* 1992; **34**: 473-480.
17. Cohen BA, Sehenk VA, Sweeney DB. Meningitis related hearing loss evaluated with evoked potential *pediatr Neurol* 1988; **4**: 18-22.
18. Platt LJ, Andrews G, Howie PM. Dysarthria of adult cerebral palsy 11 phonemic analysis of articulation errors. *J speech Hear Res* 1980; **23**: 41-55.
19. Aksu F. Nature and prognosis of seizures in patients with cerebral palsy. *Dev Med child Neurol* 1990; **32**: 661-668.