International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2020): 7.803

A Short Retrospectine Review of CP in Children

Jai Prakash[1], Reghupathy Paneerselvam[2], Mithilesh Kumar[3]

[1, 2]Department of Pediatric Medicine, Government Theni Medical College, Tamil Nadu, India[1]

[3]Department of Pediatric Medicine, Max Super Speciality Hospital, Saket, New Delhi, India[2]

Abstract: ***Cerebral palsy is a static encephalopathy and may be defined as a non-progressive disorder of posture and movement, often associated with epilepsy and abnormalities of speech, vision and intellect resulting from a defect or lesion of the developing brain (1). The exact incidence and prevalence from our country is not known. This study was conducted to evaluate the clinical profile of cerebral palsy and to find out the various predisposing factors. Methods: The present series was a retrospective study involving 210 cases of cerebral palsy. All the cases were thoroughly evaluated and the necessary investigations were done. Minear's classification of cerebral palsy (2), was used to classify and evaluate the patients. Associated handicaps such as subnormal intelligence, ocular defects, speech defects, deafness and convulsions were also noted. Results: The distributions of the cases of Cerebral Palsy are shown in Table I. amongst the 210 cases studied, the sex ratio was observed to be 2.8: 1. The athetoid, rigid, ataxic and mixed types of cerebral palsy were found mostly in males. Ninety two cases (44%) were first born. The head circumference was within 3 SD in 117 (56%) cases and was below 3 SD in 92 (44%) cases. Seventeen cases (8%) had high arched palate and other associated findings such as club foot, hydrocephalus, epicanthic folds etc. were found in 30 cases (14%).***

Keywords: Cerebral palsy, clinical profile, gestational age, post-term, term birth

1. Introduction

Table I: Distribution of Cases of Cerebral Palsy (n=210).

Type of cerebral palsy	Number	Percentage
1. Spastic	173	80.4
a) Diplegia	110	52
b) Quadriplegia	27	13
c) Double Hemiplegia	29	14
d) Hemiplegia	22	10.5
e) Paraplegia	18	8.4
f) Monoplegia	4	2.1
2. Hypotonic	15	7.5
3. Athetoid	10	4.2
4. Rigidity	2	1
5. Tremor	2	1
6.Mixed	6	3
7.Unclassified	2	1

Subnormal intelligence was observed to be the mostcommon handicap in our series (80.2%) of all the cases. Among the spastic cases, severe and profound mental retardation (IQ <35 and 20) was observed to be more common in quadriplegia (74.4% cases) and double hemiplegia (55.8% cases) as compared to diplegia (26.8% cases). All the patients of hypotonic, athetoid, rigid and mixed type of cerebral palsy were mentally retarded.

Convulsions were observed in 119 subjects (56.5%). The frequency of convulsions was 24.5% in the cases of spastic cerebral palsy, 54% in cases of mixed cerebral palsy, 68% in cases with hypotonic cereral palsy and 96.4% in cases of ataxic cerebral palsy. Other studies have reported highest incidence of convulsions in spastic type of cerebral palsy (3, 4). Overall, the incidence of myoclonic seizures was observed to be the highest (8.6% of all the cases) followed by generalized tonic clonic seizures and focal seizures (7% of all the cases each).

A high prevalence of ocular defects has been reported in children with cerebral palsy. Ninety three cases (44.5%) had ocular defects with squint being the commonest (14% of the cases), followed by nystagmus and optic atrophy in 4.6% cases each and cataract in 8% of the cases. Five per cent of the cases had other ocular defects such as refractive errors, chorio-retinitis, *etc*. All cases with cataract had history suggestive of intrauterine infection. Nine per cent of the children were blind without any obvious cause.

A high frequency of the speech defects was observed in our series (56.4% of all the cases). The highest frequency of speech disorders was observed in rigid type of cerebral palsy (98% of the cases) followed by hypotonic type (56%), spastic type (55.4%) and athetoid type (50%).

History of perinatal asphyxia was found in 50.2% of all the cases making it the single most common predisposing factor (*Table II*). Of these, 90% cases had spastic type, 7% cases had hypotonic type and 2.3% cases had athetoid type of cerebral palsy. Six per cent of all the cases were delivered by Breech delivery and history of Forceps applications, during delivery, was present in 7.5% of the cases, all of which were

of spastic type. Infections emerged as the most common postnatal association of cerebral palsy (*Table II*).

Table II: Factors Predisposing to Cerebral Palsy

Prenatal Factors	Number	Natal Factors	Number	Postnatal Factors	Number
Maternal age	81 (38.5)	Birth asphyxia	105 (50.2)	Neonatal jaundice	17 (8)
< 20 or > 30 yrs					
Toxemia	23 (10.9)	Prematurity	51 (24.2)	Pyogenic meningiits	29 (14)
Bleeding	17 (8)	Postmaturity	11 (5)	Tubercular meningitis	8 (4)
Infections	21 (10.2)	Low Birth Weight	38 (18)	others**	47 (22.6)
Trauma	12 (6)	Breech delivery	3 (1.5)		
Drugs	9 (4.2)	Prolonged labour	2 (1)		
Others*	47 (22.7)				

Figures in parantheses indicate percentages
* Others include pregnancy induced hypertension.
** Others include trauma, encephalopathy, prolonged seizures, poisoning, drowning.

2. Discussion

In the present study, among the spastic cases, mental retardation was found to be more severe in quadriplegia and double hemiplegia as compared to diplegia thus confirming the general rule that more the upper limbs are affected, lower is the intelligence (5).

A high frequency of speech defects was observed in our series. In cases of cerebral palsy, speech defects are multifactorial in origin, for example, impaired hearing, cortical damage, and incoordination or paresis of the muscles of tongue, lips, larynx and respiratory tract.

Various predisposing factors causing damage to the developing brain may lead to clinical picture of cerebral palsy and may act prenatally, natally or postnatally. The history of the other siblings suffering from cerebral palsy, present in some cases, may point towards the role of genetic factors, which are thought to contribute in about 2% of the cases (6). The well recognized familial forms of cerebral palsy are familial spastic paraplegia, familial athetosis and familial paroxysmal choreothetosis. In the present series, a family history of cerebral palsy was observed in 12 cases (5.7%) and all of them were of spastic type.

The majority of cases were observed to be due to factors acting during the birth process (*Table II*). Similar observations were made in other studies (7, 8). The causal role of birth asphyxia in cerebral palsy has been questioned and asphyxia has been suggested to be a consequence rather than a cause of the process that leads to cerebral palsy (9). However, in the NCCP project, 20 minute Apgar of less than 3 was found to be associated with 250 fold increase in the risk of cerebral palsy (10). Case control studies comparing children with cerebral palsy with controls, for the markers of birth asphyxia, have suggested an asphyxial cause in 10% of the cases (11).

Low birth weight or gestation age have been associated with an increased risk of cerebral palsy (12). Several mechanisms could be responsible for a high incidence of cerebral palsy in children who were born premature. Premature neonates are more likely to suffer from birth asphyxia and injuries. A higher incidence of breech delivery, cord prolapse and precipitate labour in preterm delivery could expose the neonates to an additional risk of mechanical and asphyxial injuries. The underlying pathological lesions in these children are periventricular leukomalacia (PVL) and periventricular hemorrhagic venous infarcts (PHVI) (13).

In breech delivery, blood is squeezed from body towards the head so that the head enters the birth canal in extremely congested form. These congested vessels readily rupture. Also the umbilical cord is pressed when the head passes through the birth canal thus reducing the blood flow to the brain. Forceps application is known to cause cerebral contusion which may lead to cerebral anoxia.

In this series, infections emerged as the most common postnatal cause of Cerebral Palsy (*Table II*). Intracranial infections can either involve the blood vessels supplying different parts of brain thus leading to anoxic damage or can directly involve particular areas of brain. The importance of infections emerging as the leading postnatal cause of cerebral palsy lies in the fact that many of them are treatable.

In conclusion, this study revealed that the natal causes are the leading predisposing factors in cases of cerebral palsy. There is thus an urgent need to further strengthen the existing maternal and child health services in the country.

References

[1] Haslam RHA. Encephalopathies. *In*: Nelson Textbook of Pediatrics, 15th edn. Eds. Berhman RE, Kliegman RM, Arvin AM. Bangalore, Prism Books Pvt Ltd, 1996; pp 1713-1714.
[2] Minear WL. A classification of cerebral palsy. Pediatrics 1956; 18: 841-852.
[3] Singhi SD, Goraya JS. Cerebral palsy. Indian Pediatr 1998; 35: 37-48.
[4] Eicher PS, Batshaw M. Cerebral palsy. Pediatr Clin North Am 1993; 40: 537-551.
[5] Brown JK. Disorders of the central nervous system. *In*: Forfar and Arneil's Textbook of Pediatrics, 4th edn. Eds. Campbell AGM, Mcintosh N. United Kingdom, Churchill Livingstone, 1992; p 819.
[6] Huges I, Newton R. Genetic aspects of cerebral palsy. Dev Med Child Neurol 1992; 34: 80-
[7] 86.
[8] Srivastava VK, Laisram N, Srivastava RK. Cerebral palsy. Indian Pediatr 1992; 29: 993-996.

[9] O' Reilly DE, Walentynawicz JE. Etiological factors in cerebral palsy. A historical review. Dev Med Child Neurol 1981; 23: 633-642.
[10] Kuban CKC, Leviton A. Cerebral palsy. New Engl J Med 1994; 330: 188-195.
[11] Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Multivariate analysis of risks. New Engl J Med 1996; 315: 81-86.
[12] Gaffney G, Sellers S, Flavell V. Case control study of intrapartum care, cerebral palsy and perinatal death. BMJ 1994; 308: 743-750.
[13] Pharoah POD, Platt MJ, Cooke T. The changing epidemiology of cerebral palsy. Arch Dis Child 1996; 75: F169-F173.
[14] Volpe JJ. Brain injury in premature infants: Is it preventable? Pediatric Res 1990; 27: 528533.