Etiological Factors in Cerebral Palsy- A Hospital Based Study from Delhi

DR. NONICA LAISRAM*, DR. R.K. SRIVASTAVA**, DR. V.K. SRIVASTAVA***

Five hundred and fortyfour cases of cerebral palsy were studied to find the etiology. Male to female ratio was 1.9:1. Prenatal, natal and postnatal factors were found in 42 (7.72%), 238(43.75%) and 142(26.1%) cases respectively. 79(14.52%) cases were found to have more than one factor which could have contributed to brain damage. In 43(7.9%) cases the prenatal, natal and post natal history were normal and the cause was not known. Among the natal causes, birth anoxia was the most common etiological factor and was observed in 24.45% cases. Infections of the central nervous system comprised the major etiopathogenic factors of the postnatal causes. In cases where more than one etiology was present, the most frequent causes were a combination of prematurity or birth anoxia in association with the factors. The present study reveals that majority of the cases were found to have natal or post natal etiology.

The incidence of cerebral palsy in India is quite alarming with various reports having indicated that 1.5 to 3.5 per thousand of all live births may be followed by the child developing cerebral palsy. (NIPCCD Seminar 1989).

The etiopathogenic factors causing the static brain damage in cerebral palsy are of prenatal, natal or postnatal origin. Studies have shown that perinatal, vascular and anoxemic brain injuries are the most frequent etiological factors (Vining etal 1976, Illingworth 1058, Skavedt 1958, Woods 1957) while others (Hagberg etal 1975, Vanja 1982) have reported a high prevalence of cerebral palsy of prenatal origin. This study was carried out to find the etiological factors of cerebral palsy cases attending our hospital.

MATERIAL AND METHOD

A retrospective study of 544 cases of cerebral palsy attending the Department of Pediatrics and Physical Medicine & Rehabilitation of Safdarjang Hospital, New Delhi between 1981 to 1989, was done to find out the various etiological factors. The cases diagnosed as cerebral palsy included those cases who had suffered brain damage in the 'developing brain' as per definition of American Academy for Cerebral Palsy (Davis and Hill 1980). The classification of cerebral palsy was based on the major groupings as described by Mitchell (Mitchell 1961). Etiological factors were grouped as prenatal, natal, postnatal, mixed or unknown. Those cases where the neurological deficit followed a convulsion, with no history of any other etiological factor, were also included in the postnatal group. In cases where there were

* MBBS, DCH, DPMR, DNB (PMR), Assistant Director, Department Of Physical Medicine And Rehabilitation
** MBBS, M.S. (Ortho.), DNB (PMR), Consultant & Head, Department of Physical Medicine & Rehabilitation
*** MBBS, DCH, M.D. (Ped.), Professor & Head of Unit, Department Of Paediatrics
SAFDARJANG HOSPITAL, NEW DELHI : 110 029, INDIA.

multiple etiological factors, they were labelled as 'mixed'. In the absence of history of any etiological factor, cases were labelled as of unknown etiology.

OBSERVATION

Out of 544 cases of cerebral palsy, there were 354 males and 190 females. Maximum cases were due to natal causes (43.75%) followed by postnatal causes (26.1% cases). 14.52% cases had more than one etiological factor, while in 7.9% cases there was no known cause. The various etiological factors operating in the prenatal, natal and postnatal period are shown in Table-2 Incases where more than one etiology was present, the most frequent causes were a combination of prematurity or birth anoxia in association with the factors (Table 1).

Of the neuromotor types of cerebral palsy, spasticity comprised the maximum number of cases (91.36%). The distribution according to topographic involvement of these 497 cases are shown in Table- 3. One case had a mixed clinical picture. No case of rigidity was found. Of the spastic group, it was observed that majority had causes of natal origin (44.98% cases), followed next by postnatal causes (26.51% cases). The distribution of the major etiological groups in relation to each clinical type is shown in Table-3. The different types of cerebral palsy in relation to each etiological factor are shown in Table-4 a,b,c.

DISCUSSION

A male prependerance was observed in our study, 65.07% cases were males while 34.93% cases were females. Izuora and Okoro (1981) have also reported the higher incidence in males. Our finding is in agreement with Basu (1969) who observed 60.8% males as against 39.2% females. Pederson etal (1982) and Makwabe and Mgone (1984) have observed very little difference between the two sexes. The male preponderance found in our study may not be a true high incidence of cerebral palsy, but a reflection of the traditional Indian family taking more interest in the male sibling in all spheres including medical attention.

Of the 544 cases, natal factors were responsible for majority of the cases (43.75%), followed by postnatal factors in 26.1% cases. Prenatal causes were least common (7.72% cases) (Table-I). Our observations are in accordance with Perlstein (1952), and O'Reilly and James (1981) who have found natal etiology to be the most common cause of cerebral palsy i.e. in 60% cases and 46.3% cases to be most common (48% cases). Makwabe and Mgone (1984) found birth anoxia and convulsions to be the predominant etiopathogenic factors. However, Vanja (1982) observed perinatal factors in 33% cases. In the present study, only 7.7% cases had prenatal etiology, while O'Reilly and James (1981), Perlstein (1952) and Hagberg etal (1975) found 38.5%, 30% and 21% cases respectively to be of prenatal origin. Vanja (1982) had reported the highest incidence of prenatal causes i.e. in 50% cases.

The incidence of postnatal cause was found to be high (45% cases) in the study of Manreen and Ogale (1982). A relatively high incidence was also found in our study (26.5% cases), while O'Reilly and James (1981), Perlstein (1952), Vanja (1982) and Hagberg etal (1975) have reported 15.2%, 10% and 6% respectively. In 14.5% cases of our series we have found multiple etiological factors while Vanja (1982) had reported 7% cases to be mixed etiology. Hagberg etal (1975) and Makwabe and Mgone (1984) observed that in 21% cases and 4% cases respectively, the cause was untraceable. In our study, 7.9% cases were of unknown etiology. (Table-5).

Of the etiological factors (Table-1) responsible for cerebral palsy, we found that of prenatal

factors, microcephaly (1.84% cases) and toxemia (1.29% cases) were the most common. Among natal factors, anoxia was the most common factor (24.45% cases). Infections of the central nervous system comprised the major etiopathogenic factor of the postnatal causes. 11.95% cases had encephalitis, while 5.15% cases had meningitis. O'Reilly and James (1981) in their study found multiple pregnancy and idiopathic factors to be the most frequent causes in the prenatal group i.e. 5.6% and 1.9% cases respectively. The most common natal etiology in their study was prematurity (22.7% cases) followed by anoxia (7.7% cases). In the postnatal group, encephalitis has been reported to be the most frequent factor (6.9% cases), which is similar to our observation.

Analysis of the etiological groups in relation to each clinical type revealed natal causes to be most common. Incidence of this etiological group in cases of monoplegia, paraplegia, quadriplegia, diplegia, ataxia and hypotonia were found to be 80.0%, 41.67%, 54.73%, 49.58%, 37.5% and 36.66% respectively. Birth anoxia has been repeatedly emphasised to be an important contributor to subsequent neurological handicap in the child, though the extent of anoxia which may be responsible for subsequent neurological handicap still remains a matter of controversy. We have observed that anoxia was consistently the most common etiologic factor among those cases with monoplegia, paraplegia, quadriplegia, diplegia and ataxia i.e. in 0.55%, 1.29%, 11.76%, 6.07% and 0.55% cases respectively. (Table-6).

In view of the large number of natal and postnatal causative factors responsible for cerebral palsy, which are by and large preventable, it is suggested that the maternal and child health services existing in the country be reinforced. The priorities should include provision of skilled obstetric and pediatric care at all levels of health delivery. In addition, measures should be taken to increase the awareness of the preventive aspects of cerebral palsy, in the community as well as medical professionals alike. This would go a long way in preventing and reducing the incidence of this major handicapping disorder.

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TABLE - 1 : MIXED ETIOLOGICAL FACTORS (n = 79)

					No. of cases
Prematurity	+	Anoxia			14
Forceps	+	Anoxia			7
Prematurity	+	Toxemia			6
Prematurity	+	Toxemia	+	Anoxia	6
Prematurity	+	Twin	+	Anoxia	5
Caesarean	+	Anoxia			5
Prolonged labour	+	Anoxia			5
APH	+	Anoxia			4
Toxemia	+	Anoxia			4
Prematurity	+	Twin			4
Prematurity	+	Breech			3
Prematurity	+	PH	+	Anoxia	3
Prematurity	+	APH			3
Breech	+	Anoxia			2
Toxemia	+	Low birth weight			2
Toxemia	+	PH			2
Toxemia	+	APH	+	Anoxia	2
Precipitale delivery	+	Anoxia			1
Microcephaly	+	Meningitis			1

*APH = Antepartum haemorrhage

TABLE - 2: DISTRIBUTION OF ETIOLOGICAL FACTORS (n = 544)

Prenatal	No.	(%)	Natal	No	.(%)	Postnatal	No.	(%)
Microcephaly	10	(1.84)	Anoxia	133	(24.45)	Encephalitis	65	(11.95)
Toxemia	7	(1.29)	Prematurity	22	(4.04)	Meningitis	28	(5.15)
Rubella	1	(0.18)	Prolonged labour	18	(3.31)	Convulsions	20	(3.68)
Toxoplasmosis	1	(0.18)	Forceps	15	(2.76)	Head Injury	13	(2.39)
Other maternal infections	6	(1.10)	Breech	12	(2.21)	Ac. Inf.Hemiplegia	8	(1.47)
			LBW < 2500g)	11	(2.02)	Neonatal Jaundice	8	(1.47)
APH	5	(0.92)	Post maturity	9	(1.65)			
Hydrocephalus	3	0.55)	Caesarean	8	(1.47)			
Drugs	3	(0.55)	Precipitale labour	5	(0.92)			
Twins	3	(0.55)	Cord around neck	3	(0.55)			
Consanguinity	1	(0.18)	Face presentation	2	(0.37)			
Diabetes	2	(0.37)						

TABLE - 3: ETIOLOGICAL GROUPS IN DIFFERENT TYPES OF CEREBRAL PALSY

Type	Total	Prenatal	Natal	Postnatal	Mixed	Unknown
		No. (%)	No. (%)	No (%)	No. (%)	No. (%)
Spasticity	n = 497	36 (7.24)	224 (45.07)	132 (26.56)	67 (13.48)	38 (7.65)
Quadriplegia	n = 190	8 (4.21)	104 (54.73)	44 (23.15)	22 (11.57)	12 (5.78)
Hemiplegia	n = 156	9 (5.77)	46 (29.49)	75 (48.08)	14 (8.97)	12 (7.69)
Diplegia	n = 119	13 (10.92)	59 (49.58)	8 (6.72)	28 (23.53)	11 (9.24)
Paraplegia	n = 24	4 (16.67)	10 (41.67)	5 (20.83)	2 (8.0)	3 (12.50)
Monoplegia	n = 5	-	4 (80.00)	- -	1 (20.00)	- -
Triplegia	n = 3	2 (66.67)	1 (33.33)	- -	-	- -
Hypotonia	n = 30	4 (13.33)	11 (36.66)	1 (3.33)	10 (33.33)	4 (13.33)
Ataxia	n = 8	1 (12.5)	3 (37.5)	2 (25.0)	1 (12.5)	1 (12.15)
Athetosis	n = 7	- -		6 (85.71)	1 (14.3)	- -
Tremor	n = 1	- - - -		- -	- -	1 (100.00)
Mixed	n = 1	-	-	1 (100.0)	- -	- -

TABLE - 4 a : TYPE OF CEREBRAL PALSY IN RELATION TO ETIOLOGY (PRENATAL GROUP)

Etiology	Mono	Hemi	Para	Tri	Quad	Dip	Total	Athet	Ataxia	Hypo	Tremor
	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)
Microcephaly	-	1(0.18)	-	1(0.18)	2(0.37)	6(1.1)	10(1.84)	-	-	-	-
Toxemia	_	2(0.37)	2(0.37)	-	-	2(0.37)	6(1.10)	-	-	1(0.18)	-
Other Maternal infections	-	3(0.55)	2(0.37)	-	1(0.18)	-	6(1.10)	-	-	-	-
A.P.H.	-	-	-	-	1(0.18)	4(0.74)	5(0.92)	-	-	-	-
Hydrocephalus	-	-	-	-	-	-	-	-	1(0.18)	2(0.37)	-
Drugs	-	3(0.55)	-	-	-	-	3(0.55)	-	-	-	-
Twins	-	-	-	1(0.18)	1(0.18)	1(0.18)	3(0.55)	-	-	-	-
Diabetes	-	-	-	-	1(0.18)	-	1(0.18)	-	-	1(0.18)	-
Consanguinity	-	-	-	-	1(0.18)	-	1(0.18)	-	-	-	-
Rubella	-	-	-	-	1(0.18)	-	1(0.18)	-	-	-	-
Toxoplasmosis	-	-	-	-	1(0.18)	-	1(0.18)	-	-	-	-

TABLE - 4b: TYPE OF CEREBRAL PALSY IN RELATION TO ETIOLOGY (NATAL GROUP)

Etiology	Mono	Hemi	Para	Tri	Quad	Dip	Total	Athet	Ataxia	Hypo	Tremor
	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)	No.(%)
Anoxia	3(0.55)	16(2.94)	7(1.29)	1(0.18)	64(11.76)	33(6.07)	124(22.79)	-	3(0.55)	6(1.10)	-
Prematurity	-	7(1.29)	-	-	2(0.37)	12(2.21)	21(3.86)	-	-	1(0.18)	-
Prol.Labour	-	4(0.74)	1(0.18)	-	11(2.02)	2(0.37)	18(3.30)	-	-	-	-
Forceps	-	5(0.92)	1(0.18)	-	6(1.10)	-	12(2.21)	-	-	3(0.55)	-
Breech	-	4(0.74)	1(0.18)	-	5(0.92)	2(0.37)	12(2.21)	-	-	-	-
LBW	-	2(0.37)	-	-	4(0.74)	5(0.92)	11(1.29)	-	-	-	-
Postmaturity	-	1(0.18)	-	-	3(0.55)	4(0.74)	8(1.47)	-	-	1(0.18)	-
Caesarean	1(0.18)	4(0.74)	-	-	3(0.55)	-	8(1.47)	-	-	-	-
PPT.Labour	-	2(0.37)	-	-	3(0.55)	-	5(0.92)	-	-	-	-
Cord around neck	-	-	-	-	2(0.37)	1(0.18)	3(0.55)	-	-	-	-
Face Presentation	-	1(0.18)	-	-	1(0.18)	-	2(0.37)	-	-	-	-

TABLE - 4 c : TYPE OF CEREBRAL PALSY IN RELATION TO ETIOLOGY (POSTNATAL GROUP)

Etiology	Mono No.(%)	Hemi No.(%)	Para No.(%)	Tri No.(%)	Quad No.(%)	Dip No.(%)	Total No.(%)	Athet No.(%)	Ataxia No.(%)	Hypo No.(%)	Tremor No.(%)
Encephalitis	-	24(4.41)	-	-	29(5.33)	8(1.47)	61(11.21)	1(0.18)	2(0.37)	-	-
Meningitis	-	22(4.04)	1(0.18)	-	5(0.90)	-	28(5.15)	-	-	-	-
Convulsions	-	10(1.84)	4(0.74)	-	5(0.90)	-	19(3.49)	-	-	1(0.18)	-
Head injury	-	11(1.29)	-	-	2(0.37)	-	13(2.39)	-	-	-	-
Ac infantile hemiplegia		8(1.47)	-	-	-	-	8(1.47)	-	-	-	-
Neonatal Jaundice	-	-	1(0.18)	-	2(0.37)	-	3(0.55)	5(0.92)	-	-	-
Mixed	1(0.18)	14(2.51)	2(0.37)	-	22(4.04)	28(5.15)	67(12.37)	1(0.18)	1(0.18)	10(1.84)	-
Unknown	-	12(2.21)	2(0.37)	-	12(2.21)	11(2.02)	37(6.80)	-	1(0.18)	4(0.74)	1(0.18)

TABLE -5: COMPARISON OF ETIOLOGICAL FACTORS WITH OTHER STUDIES

Author/Place of Study	Prenatal	Natal/Perinatal*	Postnatal	Mixed	Unknown
1. Peristein, U.S.(1952)	30%	60%	10%	-	-
2. Hagberg etal, Sweden (1975)	21%	48%*	6%	-	21%
3. O'Reilly and James, Missouri (1981)	38.5%	46.3%	15.2%	-	-
4. Vanja, North West, U.S.(1982)	50%	33%*	10%	7%	-
5. Maureen & Ogale, Nigeria(1982)	-	48%	45%	-	-
6. Makwabe & Mgone, Tanzania(1984)	-	24%	72%	-	4%
7. Present study, Delhi (1990)	7.7%	43.8%	26.1%	14.5%	7.9%