International Journal of Contemporary Pediatrics
Makwana M et al. Int J Contemp Pediatr. 2017 Jul;4(4):1146-1152
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: http://dx.doi.org/10.18203/2349-3291.ijcp20172065

Original Research Article

A clinico-epidemiological study of cerebral palsy in western Rajasthan

Mohan Makwana, Harish Kumar Mourya*, Saroj Mourya, B. D. Gupta, Ratan Lal Bhati, Yogesh Garg

Department of Pediatrics, Dr. Sampurnanand Medical College, Jodhpur, Rajasthan, India

Received: 03 April 2017
Revised: 21 April 2017
Accepted: 06 May 2017

***Correspondence:**
Dr. Harish Kumar Mourya,
E-mail: harishmourya1@gmail.com

ABSTRACT

Background: Cerebral palsy (CP) is one of the leading causes of disability in children. Incidence of cerebral palsy varies from one to six per thousand live births. Besides handicapping a child, it causes considerable psychological and social trauma to the parents and financial burden to family and community. A cure for cerebral palsy (CP) has not yet been discovered, hence, a need for primary prevention of disease. But unfortunately, the etiology of cerebral palsy is poorly understood thereby eluding a definitive prevention strategy.
Methods: All cases of non-progressive neurological disorder in the age group of 0-9 years were enrolled in the present study. Thorough and complete Obstetric history including antenatal, natal and post-natal were recorded. Woodside scale is used for assessment of neurodevelopment and a complete anthropometric measurement, physical examination including detailed Central Nervous System and all neuroimaging and EEG were recorded. Severity of cerebral palsy was assessed according to Minear's classification. Statistical analysis was done by standard statistical methods.
Results: Majority of patients were males (78.26%) as compared to females. The mean age of male patients was 2.04±1.53 years and females was 3.67±3.14 years. Spastic type of CP was most common (93.47%), followed by mixed (4.34%) and atonic (2.17%) type. Quadriplegic CP was the most common (76.75%), followed by diplegia (11.62%) amongst the spastic variety. Risk factors found are maternal pyrexia (17.39%), home vaginal delivery (58.70%), birth asphyxia (67.39%), Neonatal seizures (39.13%) other factors being icterus neonatorum (13.04%) and meconium aspiration syndrome (13.04%), ICH (10.86%), septicemia (8.69%) and renal failure (4.34%). Majority of the patients (78.26%) had class III and IV functional impairment. Cortical atrophy was the most commonly (71.73%) seen abnormality on neuroimaging with CT-scan. Epilepsy was present in 47.82% patients.
Conclusions: Cerebral palsy along with its associated dysfunctions definitely disturbs the routine of children's life as well as family life. We have to go a long way before something definitive can be done for these children and their families.

Keywords: Cerebral palsy, CT scan, Etiology, EEG

INTRODUCTION

Cerebral palsy (CP) is one of the leading causes of disability in children. Incidence of cerebral palsy varies from one to six per thousand live births. Besides handicapping a child, it causes considerable psychological and social trauma to the parents and financial burden to family and community. A cure for cerebral palsy (CP) has not yet been discovered, hence, a

need for primary prevention of disease. But unfortunately, the etiology of cerebral palsy is poorly understood thereby eluding a definitive prevention strategy. Cerebral palsy can be defined as "non-progressive disorder of central origin involving defects of movement and posture dating back to brain damage sustained during perinatal period of brain development, associated with abnormalities of speech, vision, intellect, hearing and behavior".[1] There are increasing evidences suggesting rise in prevalence of CP.[2-5] Modern improved obstetric and advanced prenatal care has resulted in increased survival of low birth weight babies and is associated with an increased proportion of cerebral palsy in these babies.[2-8] Etiology of cerebral palsy has been reported to be prenatal in 7-50%, natal in 33-60% and postnatal in 26-45% cases.[4,5]

METHODS

The present study was conducted at Department of Pediatrics, Regional Institute of Maternal and Child Health, Umaid Hospital for Women and Children, Dr. S.N. Medical College, Jodhpur.

All cases of non-progressive neurological disorder in the age group of 0-9 years were enrolled in the present study. Complete information about these patients including age, sex, religion, paternal and maternal age, place of residence was recorded in a specially designed Performa.

Thorough and complete Obstetric histories including antenatal, natal, postnatal and developmental abnormalities were recorded. Related neuroimaging and some diagnostic tests like Cranial USG, CT scan of head, EEG, CSF and IgM or IgG for TORCHS in baby and mother performed.

Woodside scale was used for neurodevelopment assessment. Anthropometric measurements, CNS examination and related neuroimaging performed. Classification of cerebral palsy was done according to Minear's classification as shown in Table 1.

Table 1: Classification of cerebral palsy.

Physiologic	Topographic	Etiologic	Functional
Spastic	Monoplegia	Prenatal (e.g. Infections, metabolic, anoxia, toxic, genetic, infarction)	Class I: No limitation of activity
Athetoid	Paraplegia	Perinatal (e.g. Anoxia)	Class II: Slight to moderate limitation
Rigid	Hemiplegia	Postnatal (e.g. Toxins, trauma, infection)	Class III: Moderate to great limitation
Ataxic	Triplegia		Class IV: No useful physical activity
Tremor	Quadriplegia		
Atonic	Diplegia		
Mixed	Double hemiplegia		
Unclassified			

The statistical analysis was performed by using student's "t" tests to find out the significance of difference in mean between two variables. P value <0.05 was considered as significant with either negative or positive correlation on account of biological variability.

RESULTS

In our study male to female ratio was 3.6:1 with mean age of male patients was 2.04±1.53 years and females was 3.67±3.14 years. The mean paternal age at birth was 27.25±4.78 years and mean maternal age was 23.13±4.45 years. The difference in the mean age in both sexes was not statistically significant (p >0.9)

Majority of patients were from rural background (58.69%) as compared to urban areas (41.31%).

Table 2 shows that distribution of various demographic factors in different types of cerebral palsy again showing a male preponderance, more so in spastic quadriplegia and diplegia. The children from rural background outnumber in severest forms of cerebral palsy. The above table also reveals that there is no clear relationship with extremes of paternal or maternal age when any of the types of cerebral palsy.

Spastic type of CP was most common (93.47%), followed by mixed (4.34%) and atonic (2.17%) type. Quadriplegia was the most common (76.75%), followed by diplegia (11.62%) amongst the spastic variety.

The mean birth order of all the patients was 2.32±1.92. A comparison of mean birth order amongst various types of CP was statistically insignificant (p >0.3).

Maternal pyrexia was the commonest antenatal factor seen in 17.39% patients with cerebral palsy while preeclampsia was present in only 2.17% patients.

Approximately, ninety one percent patients in the present study were born at term, and only 6.52% patients were delivered prematurely

Table 2: Demographic trends in patients of cerebral palsy.

	Spastic (n=43)			Atonic (n=1)	Mixed (n=2)	Total (n=46)
Demographic characters	Quadriplegia (n=33)	Diplegia (n=5)	Others (n=5)			
Male (%)	77.14%	80%	80%	100%	100%	78.26%
Female (%)	22.86%	20%	20%	0.00%	0.00%	21.74%
Mean paternal age (yrs)	27.07±4.63	28.8±8.19	25.80±3.03	32.0±0.0	27.5±3.50	27.25±4.78
Mean maternal age (yrs)	22.78±3.85	24.6±7.09	22.80±3.63	27±0.00	24.0±5.65	23.13±4.15
Rural (%)	63.63%	66.66%	66.66%	100%	50%	58.69%
Urban (%)	36.37%	33.34%	33.34%	0.0%	50%	41.31%

Majority of these patients were delivered at home and unfortunately, only 41.30% were institutional deliveries. 89.13% patients were delivered by normal vaginal route, rest 10.86% by a caesarean section. 19.56% patients also had prolonged IInd stage of labour.

A history suggestive of birth asphyxia was present in 67.39% patients but the documentary evidence of hypoxic ischemic encephalopathy (HIE) was available only in 10.86% patients.

Neonatal seizures were the single most common factor in 39.13% patients, other factors being icterus neonatorum and meconium aspiration syndrome (13.04% each), ICH (10.86%), septicemia (8.69%) and renal failure (4.34%).

Malnutrition was common among these patients (59.56%) and 17.38% patients had severe degree of malnutrition. Majority of our patients had microcephaly (80.44%) and 30.44% patients were short stature as compared to their peers.

Table 3: Associated clinical features in patients of cerebral palsy.

Clinical features	Spastic			Atonic (n=1)	Mixed (n=2)	Total (n=46)
	Quadriplegia (n=33)	**Diplegia (n=5)**	**Others (n=5)**			
Delayed milestones	33 (100%)	5 (100%)	5 (100%)	1 (100%)	2(100%)	46 (100%)
Optic atrophy	4 (12.12%)	-	-	-	-	4 (8.69%)
Loss of vision	1 (3.03%)	-	-	-	-	1 (2.17%)
Loss of hearing	1 (3.03%)	-	-	-	-	1 (2.17%)
7th nerve palsy	1 (3.03%)	1 (20%)	-	-	-	2 (4.34%)
Squint paralytic	-	-	-	-	-	-
Non-paralytic	6 (18.18%)	1 (20%)	-	-	-	7(15.22%)
Epilepsy	16 (48.48%)	2 (40%)	4 (80%)	-	-	22(47.82%)
Abnormality of speech	9 (27.27%)	1 (20%)	1 (20%)			11(23.91%)

Developmental retardation was observed in all the patients. Epilepsy was present in 47.82%, 23.91% patients had abnormality of speech, 15.22% had squint, 8.69% had optic atrophy, 4.34% had 7th nerve palsy and 2.17% had loss of hearing and vision. These problems had highest association with patients having spastic quadriplegia. Other significant features in patients of cerebral palsy were abnormality of speech, optic atrophy (8.6%), loss of hearing and vision (2.17% each). 7th nerve palsy (4.34%) and non-paralytic squint 7 (15.22%), in addition to delayed milestones and epilepsy. Majority of these findings were observed in patients of spastic quadriplegia. The mean developmental quotient (DQ) was 34.41±19.16 in all the patients. It was lowest in atonic (25.0±0.0) and spastic quadriplegia (31.2±17.98). There was no statistically significant difference in DQ among various varieties of cerebral palsy ($p > 0.2$). Majority of the patients (78.26%) had class III and IV

functional impairment and were unfortunately left with minimal to no useful physical activity.

Variable degree of cortical atrophy was the most commonly (71.73%) seen abnormality on neuroimaging with CT-scan. Among these, 50% patients had generalized cortical atrophy and 21.73% patients had focal atrophy. 10.86% each had porencphaly, leucomalacia, infarction and calcifications. Focal pachygyria was observed in 6.52% patients.

Table 4: CT-scan in patients of cerebral palsy.

	Spastic			Atonic (n=1)	Mixed (n=2)	Total (n=46)
CT scan features (45)	**Quadriplegia (n=33)**	**Diplegia (n=5)**	**Others (n=5)**			
Gen. Cortical atrophy	17 (51.51%)	3(60%)	3 (60%)	-	-	23 (50%)
Focal cortical atrophy	8 (24.24%)	-	1 (20%)	1(100%)	-	10 (21.73%)
Leucomalacia and porencephal	3 (9.09%)	1 (20%)	1 (20%)	-	-	5 (10.86%)
Infarction	3 (9.09%)	1(20%)	1 (20%)	-	-	5 (10.86%)
Calcification	3 (9.09%)	1 (20%)	1 (20%)	-	-	5 (10.86%)
Hydrocephalus	1 (3.03%)	1 (20%)	-	-	-	2 (4.34%)
Others pachygyria	2 (6.06%)	1(20%)	-	-	-	3 (6.52%)

Table 4 depicts various CT-scan findings in cerebral palsy. It was observed that 33 (71.75%) patients had significant cerebral atrophy. Generalized cortical atrophy was revealed in 23 (50%) patients of cerebral palsy and associated with leucomalacia and porencephaly in 5 (10.86%) patients. 10 (21.73%) patients had focal cortical atrophy, all in frontal region and 3 (6.52%) of these were also having atrophy in parietal regions. This was observed more commonly in patients of spastic quadriplegic type of cerebral palsy. Infarction was observed in 5 (10.86%) patients, out of which 3 (6.52%) were in the region of basal ganglia and the remaining in parietal and frontal regions. Intracranial calcification was detected in 5 (10.86%) patients.

This was seen most common in basal ganglia 3 (6.52%), followed by frontal cortical region. Diffuse calcification was seen in one patient. These calcifications were thought of TORCH infection, but we could investigate only one patient, which proved to be CMV infection.

Table 5: EEG findings in patients of cerebral palsy.

	Spastic			Atonic (n=1)	Mixed (n=2)	Total
EEG changes (n=40)	**Quadriplegia (n=33)**	**Diplegia (n=5)**	**Others (n=5)**			
Abnormal activity						
Spikes and sharp waves	14(42.42%)	01(20%)	02	-	1 (50%)	18(39.13%)
Spikes	05(15.15%)	02(40%)	(40%)	-	02 (100%)	9(19.56%)
Sharp waves	06(18.18%)		-01(20%)			7(15.21%)
High volt. Slow waves	08(24.24%)	01(20%)	01(20%)	01(100%)		11(23.91%)

Other findings detected were hydrocephalus 2 (4.34%) and focal pachygyria 3 (6.52%) in bilateral frontal regions. Abnormal electroencephalographic (EEG) activity was picked up in quite significant number of patients. Generalized spikes and sharp waves pattern was most common observed in 36.95% patients followed by high voltage slow waves in 23.91% patients and isolated spikes and sharp waves in 19.56% and 15.21% patients respectively. Epilepsy was present in 47.82% patients. Abnormal electro-encephalographic activity was detected in quite significant number of patients. Generalized spikes and sharp waves pattern was most commonly (36.95%) detected abnormality, followed by high voltage

slow waves (23.91%) spikes (19.56%) and sharp waves (15.21%).

DISCUSSION

The interest of clinicians in cerebral palsy has fluctuated markedly over the years and has been influenced by diagnostic considerations, classifications, epidemiological studies, etiological and pathogenic controversies, neonatal practice and follow-up, and by the variety of pattern of care, treatment and support available for children with neurodevelopment disabilities, and their families.

In our study according to sex, 36 (78.26%) patients were males and 10 (21.73%) patients were females. Similar male preponderance has been reported earlier in so many other studies also.[2,6,7,9-12]

Maximum numbers of patients at presentation were in the age group of 1-3 years in both sexes. Mean age of 2.04±1.53 years was observed in males and 3.67±3.14 years in females with an overall mean age of 2.39±2.04 years among all patients.

On an assessment of parental age mean age of the fathers were 28.07±1.50 years and mothers were 23.52±1.40 years. 16 (34.78%) mothers were of age ≤ 20 years (mean age 19.00±1.26 years) and 3 (6.52%) fathers were in the age group of > 35 years (mean age 38.66±2.88 years). As Kapoor et al and others also found no association with the parental age.[6,7,10,12]

As per their living area no specific pattern of CP with rural 27(58.69%) or urban 19 (41.31%) background was found in review of literature.

On clinical classification of these 46 (100%) patients, 43 (93.47%) patients were spastic, 2 (4.34%) were of mixed and 1(2.17%) was of atonic variety. 33 (76.75%) patients among the spastic variety were quadriplegic, 5 (11.62%) diplegic and remaining were hemiplegic, paraplegic and double hemiplegic. These findings were similar to the majority of studies conducted in India (15-86.4%) and developed countries (27-80%).[1,2,4,5,10,13,14] Eicher et al in their study reported that spastic quadriplegia was the commonest variety (27%).[12] Surprisingly, Sharma et al reported spastic diplegia (54%) to be the commonest type of cerebral palsy.[9] This high incidence may be due to a chance clustering of patients with spastic diplegia during a given time. Among prenatal factors associated with cerebral palsy in Indian perspective the mean birth order was 2.15±1.58 in spastic quadriplegia, 4.0±3.93 in spastic diplegia and 2.00±1.22 in other form of spastic CP. The mean birth order for all patients of CP was 2.32±1.92. our results were matching with the other study that majority of cases of cerebral palsy were having at least one normal elder sib.[15,16] Others antenatal factors were maternal pyrexia in 8 (17.39%) and eclampsia 1 (2.17%). Powell et al, Grethers et al and others found no significant association between maternal hypertension and CP.[15,17]

In our study, relevant perinatal factors in patients of cerebral palsy 42 (91.30%) patients were born at term, only 3 (6.52%) were preterm deliveries and one (2.17%) post-term delivery. That were very much similar to Srivastava et al who observed only 4% premature deliveries in their study.[4] contrary to this studies from across the world have reported that majority of patients with CP were born premature.[2,5,6,18]

Mode of delivery was home vaginal delivery 41 (89.14%) by normal vaginal route and 5 (10.86%) by caesarean section. No clear association was observed between LSCS deliveries and cerebral palsy. Our findings were similar to Powell et al who reported that caesarean section was not significantly associated with CP.[17] This is in contrast to a couple of other studies which have shown significant association between LSCS and cerebral palsy.[7,19]

There was significant history of prolonged labour in 9 (19.56%) and birth asphyxia was 31 (67.39%) patients. Torfs et al, Blair et al, Sharma et al, Suanand et al and others found similar higher risk up to 53 % of CP in Birth asphyxia. Majority of them were patients of spastic quadriplegia (66.66%). 5 (10.86%) patients were having documentary evidence of hypoxic ischemic encephalopathy.[13,16,20]

On analysis of various postnatal factors, neonatal seizures were single most common factor in 18 (39.13%) patients. Our findings were supported by a pioneer study conducted by Kapoor et al, Grether et al and Nelson.[10,12,15]

The next most common post-natal factor observed was meconium aspiration syndrome (MAS) in 6 (13.04%) patients and icterus neonatorum in 6 (13.04%) patients, maximum being observed in patients with spastic quadriplegia, on the contrary, higher proportion of icterus among patients of CP was reported by Kapoor et al, Dale and Stanley and Pharoah et al reported higher proportion of icterus among patients of CP.[6,7,12,19]

Intracranial hemorrhage and neonatal septicemia were observed as risk factors in 5 (10.86%) patients each. Grether et al, and others observed both were significantly associated with increased risk of developing CP. [10,15,19,20,21] Naeye et al and Nelson also reported that CNS infections had a high relative risk of 15.4 (95%) cl: (4.6-51.8) and 6.6-30.5 respectively.[2,3,19]

In study renal failure, hypoglycemia and hypocalcemia found one (2.17%) in each, also reported increased association of these factors with cerebral palsy.[13,16] In the present study, we observed that majority 32 (69.56%) of our patients had very poor nutrition and 8 (17.39%) patients had severe degree of malnutrition that was

similar to similar to Gangil et al.[22] 37 (80.44%) patients in our study were having microcephaly i.e. less by more than 3 SD, for the age, maximum being patients of spastic quadriplegia (87.87%). Similar observations were also, made by Aneja et al who reported microcephaly in 64% cases of spastic quadriplegia and 22.3% cases of diplegia.

All patients of cerebral palsy had delayed developmental milestones. The overall mean DQ of all the varieties of CP was observed to be 34.41±19.16. The mean DQ in spastic quadriplegia was 31.21±17.98; spastic diplegia had a mean DQ of 47.16±24.6, while the mean DQ in other types was 40.0±22.78. The DQ of different varieties of spastic CP was compared by applying student's "t" tests and was observed to be statistically insignificant ($p > 0.2$). Similar findings were reported by Sharma et al.

Epilepsy was an important associated condition observed in 22 (47.82%) patients. out of that 16 (48.48%) patients with spastic quadriplegia, 2 (40%) with diplegia and 4 (80%) with other types of spastic CP. Our findings were similar to Singhi et al (25-33%) and Eicher et al (33%), Sharma et al 25.6% and Aneja et al 14.5% who observed highest incidence of convulsions in patients with spastic type of cerebral palsy.[9,13,23,24] Other significant findings were linguistic milestones in 40 (86.95%), squint (non-paralytic) in 7 (15.22%), optic atrophy in 4 (8.69%), unilateral 7th nerve palsy in 2 (4.34%) and loss of hearing and vision in one (2.17%) patient each. Sharma et al, reported speech defects in 53.5%, ocular defects in 35.8% (squint 12%, cataract 6%, nystagmus 4.8%, optic atrophy 4.8%).[9] A slightly higher frequency of speech defects (36.95%) as compared to present study has been reported by Srivastava et al, who also reported visual and hearing impairment in 9.01 and 2.94% cases respectively.[4] Speech defects in CP are multi factorial in origin e.g. impaired hearing, cortical damage and in coordination or paresis of muscle of tongue, lips, larynx and respiratory tract, although it is difficult to exactly delineate association of these factors in the present study.[13]

Neuroimaging as cranial ultrasound, CT-scan, MRI, SPECT, PET etc.; have been used to find out structural CNS abnormalities in patients with CP. Most commonly observed abnormality was varying degree of cortical atrophy in 33 (71.73%) patients. 23 (50%) patients were showing generalized cortical atrophy; among these 5 (10.86%) patients had associated leucomalacia and porencephalic cysts. Focal cortical atrophy was seen in 10 (21.73%) involving frontal region while 3 (6.52%) of these were also having atrophy in parietal region. 5 (10.86%) patients revealed infarction, most commonly in basal ganglia followed by parietal and frontal regions. The presence of microcephaly in patients of CP in our study was not always associated with cortical atrophy.

Intracranial calcification thought to be due to TORCHS infection was shown in 5 (10.86%) patients and focal pachygyria in bilateral frontal region was observed in 3 (6.52%) patients. Our CT scan findings were similar to the observations made by Aneja et al.[24] They observed variable degree of cerebral atrophy in 43.1% cases. Periventricular leucomalcia and porencephaly have also been reported in quite significant proportion of patients of CP by various other workers.[25-27]

In the present study, most commonly observed abnormality in EEG was generalized spikes and sharp waves pattern in 18 (39.13%) patients, followed by high voltage slow waves in 11 (23.91%) patients; 9 (19.56%) patients were showing spikes and 7 (15.21%) patients were depicting only sharp waves. These features were further classified on the presence or absence of epilepsy in the patients of CP. Among all, 22 patients (47.82%) had epilepsy. In these patients, EEG findings were generalized spikes and sharp waves pattern in 11 (50%), high voltage slow waves in 5 (22.73%), only spikes in 4 (18.18%) and sharp waves in 2 (9.09%) patients. Those patients who were not epileptics also showed significant

EEG abnormalities, 7 (29.16%) had generalized sharp waves and spikes, 5 (20.83%) each had isolated spikes and sharp waves while 6 (25%) had high voltage slow waves. Aneja et al analyzed EEG in all patients of CP with epilepsy and observed that interictal EEG was abnormal in 59.4% of patients. The majority of patients had diffused or generalized paroxysmal abnormality.[24]

In the present study, it is very clear that majority of the patients with CP have abnormal EEG changes which apparently do not correlate well with presence or absence of epilepsy. It was observed that no consistent pattern of EEG abnormality was seen in all the patients of CP having epilepsy despite the fact that these patients had a common seizure type. Recent neuroimaging techniques (PET etc) may be more helpful in delineating metabolic activity of brain tissue.

CONCLUSION

Cerebral palsy, a non-progressive CNS disorder, is affecting not only an individual, but also the family and society as a whole. The present study aims to have a better understanding of the disease in Indian scenario. It has been our endeavor to make all efforts to correlate the observations of this study.

These patients require a multidisciplinary team approach for their management and to improve their social and vocational capabilities. We hope that this study will go a long way in contributing to the knowledge about cerebral palsy. However, further studies with more specific aims are required to improve the quality of life of these children.

ACKNOWLEDGEMENTS

Authors would like to thank colleague Dr. Harish Kumar Mourya for helping author a lot to do framing and writing the manuscript. Author would also like to give thanks to

department of pediatrics for their supports. Furthermore, Author wish to give special thanks to Professor and Unit Head Dr. BD Gupta sir for his outstanding support to conduct this study.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Haslam RHA. Encephalopathies. In: Nelson Textbook of Pediatrics, 15th edn. Eds. Berhman RE, Kliegman RM, Arvin AM. Banglore, Prism Books Pvt Ltd. 1996;1713-4.
2. Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Multivaried analysis of risk. N Engl J Med. 1986;315(2):81-6.
3. Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Univariate analysis of risks. Am J Dis Child. 1985;139(10):1031-8.
4. Laisram N, Srivastava VK, Srivastava RK. Cerebral palsy an etiological study. Indian J Pediatr. 1992; 59:723-8.
5. Hagberg B, Hagberg G. The origins of cerebral palsy. Recent advances in Pediatr. 1993;11:67-81.
6. Pharoah POD, Platt MJ, Cooke T. The changing epidemiology of cerebral palsy. Arch Dis Child 1996;75:169-73.
7. Pharoah POD, Cooke T, Cooke RWI, Rosenbloom L. Birth weight specific trends in cerebral palsy. Arch Dis Child. 1990;65:602-6.
8. Cummins SK, Nelson KB, Grether JL and Velie E M. cerebral palsy in four Northern California Countries Birth 1983 through 1985. The Journal Pediatr. 1993;(8):230-7.
9. Sharma U, Sharma P, and Kabra A. Cerebral palsy: A clinical profile and predisposing factors Indian Pediatr. 1999;36:1038-42.
10. Izuora GI, Okara ABO. Some clinical aspects of cerebral palsy among Nigerian Ibo children. Centr Afr J Med. 1981;27:155-9.
11. Basu B. Cerebral palsy in childhood. J Indian Med Association. 1980;22:13-25.
12. Suvanand S, Kapoor SK, Reddaiah VP, Singh U, Sundaram KR. Risk factors for cerebral palsy. Indian J Pediatr. 1997;64:677-85.
13. Eicher PS, Batshaw ML. Cerebral palsy. Pediatr Clin North Am. 1993;40:537-51.
14. Bhushan V. Cerebral palsy and birth asphyxia. Myth and Reality. Indian J Pediatr. 1994;61:49-56.
15. Grether JK, Nelson KB, Emery S, Cummins SK. Prenatal and Perinatal factors and cerebral palsy in very low birth weight infants. J Pediatr. 1996;128: 407-14.
16. Torfs CP, Vanden Berg BJ, Oechsli FW, Cummins S. Prenatal and perinatal factors in etiology of cerebral palsy. J Pediatr. 1990;116:615-9.
17. Powell TG, Pharoah POD, Cooke RWI, Rosenbloom L. Cerebral palsy in low birth weight infants, (I). Spastic hemiplegia; association with intrapartum stress. Dev Med Child Neurol. 1988; 30:11-8.
18. Rosenbloom L. Diagnosis and management of cerebral palsy. Arch Dis Child. 1995;72:350-4.
19. Dale A, Stanley FJ. An epidemiological study of cerebral palsy in Western Australia, 1956-1975 II. Spastic cerebral palsy and perinatal factors. Dev Med Child Neurol. 1980;22:13-25.
20. Naeye RL, Peters EC, Bartholomew M, Landis JR: Origins of cerebral palsy. Am J Dis Child. 1989; 143:1154-61.
21. Ramar S. Cerebral palsy in children can be prevented. Indian J of physical medicine and rehabilitation. 1993;6(2):7-9.
22. Gangil A, Patwari AK, Aneja S, Ahuja B, Anand V K. Feeding problems in children with cerebral palsy. Indian Pediatr. 2001;38:839-46.
23. Singhi PD. The child with cerebral palsy-clinical considerations and management. Indian J Pediatr 2001;68(6):531-7.
24. Aneja S, Ahuja B, Taluja V, Bhatia VK. Epilepsy in children with cerebral palsy. Indian J Pediatr. 2001; 68(2):111-5.
25. Wiklund M, Uvebrant P. Hemiplegic cerebral palsy. Correlation between CT morphology and clinical findings. Dev Med Child Neurol. 1991;33:512-23.
26. Volpe JJ. Brain injury in premature infants: is it preventable? Pediatr Res. 1990;27:28-33.
27. Uvebrant P. Hemiplegic cerebral palsy aetiology and outcome. Acta Paediatrica. 1988;77(s345):1-00.

Cite this article as: Makwana M, Mourya HK, Mourya S, Gupta BD, Bhati RL, Garg Y. A clinico-epidemiological study of cerebral palsy in western Rajasthan. Int J Contemp Pediatr 2017;4:1146-52.