Clinical, Neurodevelopmental and Etiological profile of children with Cerebral Palsy

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Abstract

Background: Clinical spectrum of cerebral palsy (CP) is different in developing and developed countries. We evaluated the clinical profile, etiological factors and co-morbidities of children with CP in central India. **Methods:** 50 children with CP came to the hospital in our rehabilitation center were compared with our previous study done in India and western countries. **Results:** Spastic quadriplegia is the commonest type of CP (90%). Birth asphyxia remains the main (48%) etiological factor. Prematurity was second most common (22%), Pre-eclamsia (10%), convulsions (10%), infection (8%) and pathological jaundice (8%) other important etiological factors. due to bilirubin-encephalopathy remained same (~30%). Microcephaly (56%), Speech problems (34%), seizures (36%), auditory defect (8%) and visual defect (4%) are common co-morbidities. Common neuroimaging findings include cortical atrophy (50%), periventricular leukomalacia (12.5%) and developmental defect (12.5%). **Conclusion**: The spectrum of CP is evolving in the developing countries. Lack of proper antenatal and perinatal care, malnutrition and infections appeared to be the major factors for development of CP.

Keywords: Asphyxia, Cerebral palsy, Spastic, Quadriplegia, Seizures

Introduction

Cerebral palsy (CP) is a common developmental disability first described by William Little in the 1840s. It is one of the three most common life long developmental disabilities (other two being autism and mental retardation) causing considerable burden to affected individuals and their families due to lifelong disability[1]. The condition poses considerable diagnostic and therapeutic challenges to the pediatrician and pediatric neurologist with variable degree of involvement ranging from minimal disability to total dependent of bed ridden severe form.

Cerebral palsy is primarily a disorder of movement and posture. It is defined as an "umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development".[2] Cerebral palsy happens when the areas of the brain that control movement and posture do not develop correctly or get damaged. It is often associated with epilepsy and abnormality of speech, vision intellect. It is a static encephalopathy in which, even though the primary lesion, anomaly or injury is static, the clinical

Manuscript received: 15th Apr 2014 Reviewed: 16th Apr 2014 Author Corrected; 29th Apr 2014 Accepted for Publication: 10th May 2014 pattern of presentation may change with time due to growth and developmental plasticity and maturation of the central nervous system.

Cerebral Palsy is a common problem; the worldwide incidence is estimated between 2 to 2.5 per 1000 live births [3]. Despite recent advances in neonatal management and obstetric care incidence and severity of CP is increasing [4]. This is because of more and more survival of premature, very low birth weight and severe birth asphyxiated babies. The incidence in premature babies is much higher than in term babies. For the vast majority of term infants who develop CP, birth asphyxia or obstetric complications cannot be ascribed as the cause [5, 6].

Recent clinical research in cerebral palsy has focused on the identification of prenatal or perinatal risk factors [7]. Despite these efforts, uncertainty continues to exist regarding the spectrum of etiologies and their relative contribution. Advance in technology and their appropriate use in clinical practice have certainly increased the identification of possible cause and reduced the percentage of cerebral palsy of unknown cause. Knowledge of the causes of cerebral palsy, including their distribution and relative frequency, have significant

implications for ongoing efforts directed at intervention and prevention strategies.

We have conducted this study to find out possible and probable etiological factors in our scenario & to have better understanding of modes of clinical presentation. We have also assessed developmental profile of these children.

Material and Methods

The present study was hospital based study comprising 50 patient of cerebral palsy between 6 months to 8 years of age. Patient were selected randomly who were came to attend high risk clinic in department of pediatrics, Gandhi Medical College and Composite regional centre for persons with disability, Bhopal (MP), without any bias for sex.

Malnutrition grade IV
Clinically significant systemic illness (Any systemic illness more than 1 month duration)

1. Progressive neurological disorder

Each child was subjected to complete medical and neurological examination to detect cases with CP. These diseased children were subjected to meticulous neurological and medical assessment, brain MRI, EEG. A detailed history including antenatal, perinatal, postnatal, developmental history and obstetrical history of mothers especially birth asphyxia and prolong labour were taken. Present illness and past significant history related with disabilities were taken.

Clinical examination with particular emphasis on neurological examination was done. Findings were recorded on restructured proforma with special emphasis on developmental profile and developmental quotient. Necessary investigations were done in all patients.

Exclusion Criteria-

Results

50 children of cerebral palsy between 6 months to 8 years of age were included in this study.

Table No 1: Age and Sex distribution of Patients

			Gender				
Age group	No of cases	%	Male		Female		
			No	%	No	%	
6 Months- 1 Year	03	06	02	04	01	02	
1 Year- 5 Years	34	68	24	48	07	14	
5 Years-8 Years	13	26	12	24	04	08	
Total	50	100	38	76	12	24	

This table shows that maximum number of patients (68%) was in the age group of 1-5 years and minimum numbers of patients (6%) were in the age group of 5-8 years. Male to female child ratio was 3:1.

Table No 2: Gestational maturity of patients (n=50)

Maturity	Male	Female	Total	Percentage
Pre-term	10	01	11	22
Term	25	12	37	74
Post-term	02	00	02	04
Total	37	13	02	100

Table No-02 depicts that majority of patients were term born children (74%). 22% children were born premature.

Table No 3: Maternal age and Birth Order in affected children

Maternal Age			B	Birth Order		
Age group	No. of cases	%	Order of Birth	No. of cases	%	
< 20 Years	07	14	1 st	18	36	
20-30 Years	37	74	2^{nd} - 3^{rd}	19	38	
>30 Years	06	12	4 th -5 th	07	14	
Total	50	100	$\geq 6^{th}$	06	12	
			Total	50	100	

This table shows that in 74% cases maternal age at the time of birth of child was between 20-30 years. In 14% cases maternal age was less than 20 years and in 12% cases it was more than 30 years. In our study 36% of affected children were found in first birth order and 38% in 2^{nd} / 3^{rd} birth order.

S.	Symptoms			Associate defects		
No.	Name	No. of cases	%	Name	No. of cases	%
01	Delayed milestone	49	98	Microcephaly	28	56
02	Feeding difficulties & drooling of saliva	19	38	Convulsion	18	36
03	Convulsion	18	36	Speech defect	17	34
04	Speech defects	17	34	Auditory defect	04	08
05	Distribution of tone & posture-			Visual defect	02	04
	1. Stiffness	04	08			
	2. Limpness	01	02			

Table No 4: Presenting symptoms and associated defects

Above table shows that delayed milestone is most common presenting symptoms in 98% cases, followed by feeding difficulty, convulsion and speech defect in 38%, 36% and 34% cases. Similar microcephaly is commonest associate defect in 56% cases.

Table No 5: Probable Etiological Factors

Etiological factors	No of cases	Percentage		No	%
Antenatal	08	16	1.Pre-eclamsia	05	10
			2.Antipartum Hemorrhage	03	06
Perinatal	24	48	1.Birth Asphyxia	24	48
			2.Abnormal Labour-		
			a. Prolong Labour	01	02
			b. Forceps delivery	01	02
			c. Cesarean Section	02	04
			3.Prematurity	11	22
Postnatal	09	18	1.Infection	04	08
			2.Convulsion	05	10
			3.Pathological Jaundice	04	08
Mixed	02	04			
Unknown	07	14			
Total	50	100			

Table No 05 shows that perinatal insult were the most common cause of cerebral palsy in 48% cases. In 14% cases despite using best possible diagnostic modalities, cause remains unknown. Birth asphyxia was the commonest cause (48%) of cerebral palsy. In 22% cases prematurity was culprit.

Table No 6: Clinical and Topographic presentation of Cerebral Palsy

Clinical Type	No of cases	%	Topographic Type	No. of cases	%
Spastic	45	90	Quadriplegia	23	46
Atonic	04	08	Diplegia	19	38
Mixed	01	02	Hemiplegia	08	16
Total	50	100		50	100

Table No 06 shows that spastic type was the most common (90%) type of cerebral palsy , followed by atonic or hypotonic with 08% prevalence. There was no case of athetosis in our study. In topographic distribution quadriplegia was the

commonest type (46%) followed by diplegic type (19%). There was no case of monoplegia, paraplegia, triplegia or double hemiplegia in the study.

Neuroimaging findings			EEG Pattern		
Findings	No. of cases	%	Total EEG done	Abnormal EEG	Normal EEG
Cortical atrophy	12	50	18	15	03
Maldevelopment	03	12.5			
Periventricular Leucomalacia	03	12.5			
Basal ganglia lesion	00	00			
Others findings	01	04.17			
Normal findings	08	33.33			
Total	24	100			

Table No 7: Neuroimaging and EEG finding in Cerebral Palsy

Table No 7 shows that total 24 patients were undergone radiological assessment. It was normal in 8 (33%) of patients and abnormal in 16 (66%). Most common radiological finding was cortical atrophy in 50% cases, maldevelopment and periventricular leukomalacia were other common findings (12.5% each). Nearly one third patients (33.34%) there was normal neuroimaging. EEG was done in 18 cases, those having convulsions , out of them 15 patients having abnormal EEG findings.

Discussion

Cerebral palsy is the commonest cause of physical and mental disability in children. Despite huge research work that done in this field, yet in many case cause remains unknown. In birth cohorts from developed countries, the prevalence is 1-2/1000 live births. The prevalence rises dramatically with decreasing gestational age at birth such that among extremely low gestational age newborns. In gestational age < 28 weeks, the prevalence is about 100 per 1000 of surviving infant, a 100-fold higher risk than infants born at term. As a function of all live births, the prevalence has been remarkably stable for decades, but this has not been the case among very low birth weight and very preterm infants, among whom prevalence

increased after the introduction of neonatal intensive care and has begun to decrease in the past decade [1]. This study aimed at highlighting some of the major clinical presentation and etiological risk factors, which is an essential first step in reducing the burden of this disease in our country.

In our study 68% patients belong to 1-5 years of age group, because in this age group developmental retardation and physical disability become more and more obvious and child is brought to the hospital by worried parents. In less than 1 year of age, features of cerebral palsy are not very obvious and only can be identify by qualified and experienced pediatrician. By age of 8 years parents understand the nature of disease very well and become reluctant to come to the hospital. This is the reason why less than 1 year and more than 8 years children were less in numbers in hospital. Regular checkup of child can bring down the age of presentation, as age of presentation is very important for a spastic child, because early intervention can lead to better outcome in terms of quality of life. So health care workers should trained for early reorganization of sign of developmental delay.

There was a male preponderance in our study with male: female ratio 3.16:1. Cerebral palsy (CP) and related developmental disorders are more common in males than in females, but the reasons for this disparity are uncertain. This can be simple byproduct of our social practice where any health related problem of male child is given more concern by parents, while females are ignored till last. Males born very preterm also appear to be more vulnerable to white matter injury and intraventricular hemorrhage than females [8]. Experimental studies in adult animals and data from adult patients with stroke indicate that sex hormones such as estrogens provide protection against hypoxic-ischemic injury, and the neonatal brain is also influenced by these hormones [9]. However, hormonal influences on the fetus and neonates are substantially different from those on adults. Recent data from neonatal rodents subjected to hypoxia-ischemia also demonstrate differences between males and females [10].

Our study shows that gestation age has profound effect on risk of cerebral palsy. Though cerebral palsy developed in all gestational patients but majority of patients (74%) belong to term category. These results are just opposite to mentioned in literatures. Neonate survivors born before week 33 are up to 30 times more likely to risk of cerebral palsy than a baby born at term.[11] Most of diaplegic cerebral palsy developed in preterm babies. Recent research indicate that there is decrease incidence of cerebral palsy after 2000, which is due to improve perinatal care [12]. But prematurity represents one of the risk factors of newborns' mortality, morbidity and derangements of long-term neurological development especially cerebral palsy and mental retardation. With the increase of number of preterm newborns, problems with subsequent neurological development have also increased [13].

In present study 74% mothers delivered the affected child at the age between 20-30 years. It is insignificant because this is the age of highest fertility where most of parents complete their family. NA Fletcher et al also observed in his study that there is no effect of parental age or birth in spastic quadriplegia or diplegia, but a paternal age effect was detected in those with athetoid / dystonic cerebral palsy and congenital hemiplegia [14]. Maternal age >35 years is a important risk factor for cerebral palsy [15]. Multiple pregnancy, medicine used in early pregnancy, harmful environment and recurrent vaginal bleeding during pregnancy are other important risk factors of cerebral palsy [15]. Father under 20 years, first child or fifth (or more) child and baby of low birth weight (less than 2.5 pounds) can increase chance of cerebral palsy.

In present study delay milestone was the commonest presenting symptoms in 98% cases. Feeding difficulty, drooling of saliva, convulsion and speech defect were the other common symptoms, for which child brought to the hospital. Not all signs are visible at the time of birth or after few months of birth. In infants low muscle tone (baby feels 'floppy' when picked up), unable to hold up its own head while lying on their stomach or in a supported sitting position, muscle spasms or feeling stiff, poor muscle control, reflexes and posture, delayed development (can't sit up or independently roll over by 6 months), and preference to use one side of their body are very important early sing of cerebral palsy. Cerebral palsy is a complex disability and diagnosis is not always an easy process. Doctors may suspect cerebral palsy if a baby has slow motor development, has tight or floppy muscle tone, or displays unusual postures. The 'General Movements Assessment' can be conducted from birth until 3 months of age. It has been shown to be a strong predictor of cerebral palsy, particularly when certain changes to the brain are seen on an MRI. However, it cannot predict the severity of cerebral palsy.

Research Article

The distribution of etiological factors according to period of their operation shows that antenatal factors were involved in 16% cases, perinatal in 48% cases and postnatal in 18% cases. Efforts linking CP to specific etiological factors have been attempted worldwide [16-18]. Risk factors identified in this study are more or less similar to the ones reported by others. In about a half of the children (48%) birth asphyxia was most common causative factor, followed by prematurity in 22% cases. Pre-eclampsia (10%), infection (8%), convulsions(10%) and pathological jaundice(8%) are other important factors. Many etiological factors were overleaping and many patients having more than one etiological factors. Among all these factors, the presence of infection or fever during pregnancy was more prominent in the poor-resource community. National Institutes of Health Collaborative Perinatal Project (NCPP) also revealed that moderate to severe inflammatory infiltrates present within the placenta increase the risk of developing CP in both preterm and term infants [19, 20]. Significant association between preterm and CP has been seen in various Western studies [21]. This is mainly because of the increased survival of preterm and low birth weight babies due to the availability of advanced obstetric and neonatal care, which indirectly increases the risk of CP in these infants. However, unlike Western figures, most CP children examined in this particular study were born at term. Lack of antenatal care specially in underdeveloped and developing counties, put mother and unborn child to many antenatal, natal and postnatal complication and thereby increasing risk of cerebral palsy. There is a contradictory result of birth asphyxia as a causative agent of CP throughout literature. Western studies show that there is no significant association, while the developing countries especially North India, Nigeria and Malta found a strongly suggestive history of birth asphyxia in the affected children [22]. Observations supporting asphyxia as a contributory factor were also seen in this poor resource community. Acquired causes of CP, like kernicterus, meningoencephalitis and neonatal seizures, greatly contributed to a significant portion of cerebral palsy. Post natal infections and neonatal seizures however, continue to be a strong cause of CP throughout the world [23, 24]. In our study spastic type of cerebral palsy was most common accounting for 90% of total cases. Ouadriplegic (46%) and diplegic (38%) are important topographic type of CP. This finding was consistent with other study result

[25]. However, there are discrepancies between the developed and developing countries when it comes to identifying the commonest subtype of spastic CP. The spectrum of CP is evolving in the developing countries with an increase in diplegic and a decrease in quadriplegic CP. The quadriplegic CP rates have been reported to be much higher in poor-resource countries as compared to

• Genetic and metabolic investigations not done due to lack of fund and facilities.

Despite the limitations, it paves way for further studies that need to be conducted in this population on a larger scale to better assess and understand the various contributory components leading to the development of CP. That will facilitate the process of developing effective preventive and rehabilitation strategies.

Conclusion

Spastic cerebral palsy is most common form of disease. Lack of proper antenatal care, delivery in a non-hospital setting and immediate neonatal care, malnutrition and infections appeared to be the major factors contributing towards the development of CP. Enhancing parents' knowledge and awareness about their initially un-born and later born child's condition as well as targeting the preventable causes could be crucial in modifying the trend cerebral palsy. Majority of parents are not able to continue therapy due to lack of treatment facilities , suggesting the dire need for setting up specific centers dedicated solely towards the management of this debilitating disease.

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the developed countries [24]. The proposed reason for this decrease in quadriplaegic type of CP in developed countries is due to the increased survival of extremely premature infants [24]. Consanguineous marriage and lack of antenatal care were important community-related risk factors identified for spastic CP in some studies

Only few reports exist on neuroimaging abnormalities in children with cerebral palsy (CP) from India. In this study out of 50 patients 24 patients had undergone neuroimaging study. Neuroimaging abnormalities were seen in 66% of children, which is not comparable to other western studies that have reported incidence of 86 to 91% [27-29]. The neurological abnormalities in the present study were different compared to those of a systematic review by Krageloh-Mann et al. [27], which included 14 studies and 388 children. The incidence of various abnormalities in the present study vs. that of Krageloh-Mann et al. are malformations (12.5 % vs. 9%), periventricular changes (12.5% vs. 56%). Due to lesser number of preterm (n=11 & 22%) in our study incidence of periventricular changes was less. Malformations presenting as CP may have different management treatment, bearing on subsequent pregnancies and thus may require genetic counseling, and have medicolegal importance to obstetricians and neonatologists. Hence, the emphasis is on neuroimaging in children with clinically diagnosed CP. As pathogenic events affecting the developing brain can cause abnormalities depending on the stage of brain development, MRI has high potential to elucidate type, extent, and possible time of brain damage in children with CP. Further studies are needed to confirm the same in Indian children [26].

In our study EEG was done in 18 cases. EEG was positive in 83.3% cases. Senbin N et al (2002) also found abnormal EEG in 90.3% cases. Tetraplegic CP has higher incidence of epilepsy. Focal epileptiform activity, generalized slowing, and multifocal epileptiform activity were significantly frequent in epileptic CP [30].

Limitation of our study

There were several limitations to our study.

- The study only included a single block of the community who were came hospital for evaluation and not the whole community.
- Due to the nature of the research, lack of funds and manpower, a door-to-door survey was not conducted, which could have helped us identify further cases.
- Small sample size makes it difficult for us to accurately derive any true associations between the observed risk factors and CP.

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