DOI: 10.21276/sjams.2016.4.12.30

Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2016; 4(12B):4328-4334 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublisher.com

Original Research Article

Analytical Study of Clinical Profile of Cerebral Palsy

Dr. Vamshi Krishna Kondle[1], Dr .P. Sharath Chander Rao[2], Dr. Kranti Kondle [3]
[1]Associate Professor, Department of paediatrics, Kamineni institute of medical sciences, Narketpally, Nalgonda, Telangana
[2]Assistant Professor, Department of paediatrics, Malla reddy Women's Medical College, Hyderabad, Telangana
[3]Junior resident, Department of Obg, RVM institute of Medical Sciences and Research Center, Mulugu, Telangana

***Corresponding author**
Dr. Vamshi Krishna Kondle
Email: vamshikondle@gmail.com

Abstract: Cerebral palsy is a disorder of movement and posture that result from a non-progressive lesion or injury of the immature brain. The prevalence of cerebral palsy among children is about 2 per 1000 live births. it is much higher in developing countries. To analyse the clinical picture of cerebral palsy in Gulbarga area the present study conducted in government general hospital , Basaveshwar teaching &general hospital, Gulbarga attached to M.R medical college, Gulbarga were included in this study over a period of two years from march 2001 to march 2003 over 50 patients. Majority of cases were belongs to1-5years (52%).male female ratio was 2.5:1.the commonest physiological presentation was spastic type (82%).and commonest topographical presentation was quadriplegia.
Keywords: cerebral palsy, perinatal factors, postnatal factors, topographic distribution.

INTRODUCTION

Cerebral palsy is a static encephalopathy that 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 (Robert ha Haslam) [1].

Many definitions have been given for the condition. All the definitions emphasize several points. Firstly all children of cerebral palsy have suffered some form of brain damage and this has involved the motor system. Secondarily they indicate that the condition is non-progressive and hence exclude conditions such as degenerative brain diseases, cerebral tumour etc and those disorders of posture and movement which are
1) Of shorter duration
2) Due to progressive disease or
3) Due to solely to mental deficiency

Cerebral palsy is a symptom complex, rather than a specific disease. It is " umbrella term covering a group of non-progressive but often changing, motor impairment, syndrome secondary to lesions or anomalies of the brain arising in the early stages of its development" [2]. Although the essential diagnostic sign is a motor defect the possibility that there may be other associated symptom. Complexes of cerebral dysfunction are implicit in the stipulation of central nervous system pathology [3]. The upper age limit of insult to the brain is not strictly defined but in 1985 mackeith and polani described as A persisting qualitative motor disorder appearing before the age of 3 years(little club memorandum,1959) [4].

Although the pathology is static the manifestations may be dynamic, changing with brain maturation [5]. It is a group of disorders, and the difference between separate groups remains in their aetiology, pathology and clinical features. The aetiology is diverse changing and still too often unknown or imprecisely defined. The pathology is variable depending on the brain. The clinical features very according to the pathology. The lesion may be a single or multiple locations of the brain, resulting in definite motor and possibly sensor abnormality. It occurs as a result of in utero factors, events at the time of labour and delivery(congenital cerebral palsy),or a variety of factors in the early developing years (acquired cerebral palsy).the former accounts to 85% and the latter 15% of cases [6]. The prevalence of cerebral palsy among children is about 2 per 1000 live births. It is much higher in developing countries. In India the prevalence is 1.5-2.5 per 1000 live births. The exact incidence and

prevalence figures from our country are not available [7]. So the study of cerebral palsy demands a lot of attention and needs utilization of modern diagnostic tools like ultra sonography, CT scan, MRI etc. to illuminate the dark areas of knowledge regarding cerebral palsy.

AIMS AND OBJECTIVES

To analyse various clinical profile in cerebral palsy cases, who are admitted in government general hospital, Basaveshwar teaching &general hospital, Gulbarga attached to M.R medical college, Gulbarga.

MATERIALS AND METHODS

In this study of cerebral palsy in children, patients of the age group of 0 to 12 were taken, 50 consecutive cerebral palsy patients attending to government general hospital , Basaveshwar teaching &general hospital, Gulbarga attached to M.R medical college, Gulbarga were included in this study over a period of two years from march 2001 to march 2003.

History- children more than 12 years age with cerebral palsy and in those with no clear history of perinatal events are excluded. In the perinatal factors, the criteria applied for retrospective diagnosis were as follows

Maturity- as exact birth weight or maturity were not available in most of the cases, gestational age and baby size at birth were used as crude indicators of maturity.

Prolonged labour- total labour duration exceeding 16 hours in prime and 12 hours in multipara was considered as a prolonged one.

Asphyxia- apnoea or delayed cry, cyanosis or pallor, poor or absent cry, poor sucking, correlation in the early neonatal period and at birth were considered as indicator of asphyxia. Site of delivery and type of delivery was noted in an attempt to assess the impact of obstetric on cerebral palsy.

A detailed developmental history covering motor, adaptive, language and social milestones was obtained in each case. As many of the patients were grossly retorted in development and intelligence in whom no developmental or intellectual assessment scale was even satisfactorily applicable, we had to assess these factors grossly depending upon milestones achieved (motor and adaptive for developmental quotient. Language and social for intelligence quotient).In general examination, apart from the routine protocol, presence and severity of associated handicaps were noted. In the neurological examination, special attention was paid to motor system. Tone of muscle, power and deep tendon jerks were noted carefully to determine the clinical type and topographic distribution of the neurodeficit. Involuntary movement were carefully observed for their presence and type, frequency, relation to motion and distribution. All the neonatal reflexes were examined and their pathological persistence and asymmetry were noted. After thorough examination, the clinical diagnosis of cerebral palsy was established and its clinical type of topographical distribution was recorded.

Relevant investigations to exclude the simulating conditions and to detect the possible cause were carried out as follows:

1) fundoscopy- fundi were examined in both eyes of patients to detect optic atrophy or abnormal findings due to other causes.

2) x- ray skull- both antero-posterior and lateral views were done in patients to detect any calcification in the brain or any other relevant abnormality and to exclude craniosynostosis.

3) CT scan- was done in few patients without any selection basis.

RESULTS

A total of 50 patients were examined and diagnosis of cerebral palsy was established on clinical grounds. The various findings of the present study are as follows.

Table 1: Age distribution of patients

Age group	No.of cases	percentage
Less than one year	4	8
One year one month to 5 years	26	52
5 years I months to 10 years	18	36
More than 10 years (10-12 years)	2	4
Total	50	100

Maximum or about half of the total patients i.e 26(52%) studied were in the age of one to 5 years, more than one fourth of the total patients in this study are of age 5 to 10 years i.e 18(36%). 4 cases (8%) were in the age of less than one year and in the age group of 10 to 12 years, there were only 2 (4%) cases.

Table 2: sex distribution

Sex	No. of cases	Percentage
Male	36	72
Female	14	28
Total	50	100

Out of 50 patients in the study 36 (76%) were male and 14(28%) were females, showing a male predominance. In this study, the male: female ratio is 2.5:1.

Table 3: presenting symptoms

Symptoms	No.of cases	percentage
Delayed milestones	47	94
Feeding difficulties and excessive drooling	28	56
Speech defects	12	24
Convulsions	33	66
Disturbance of tone and posture	6	12
Hearing	3	6
Vision	4	8
Involuntary movement	6	12
Contractures	8	16
Respiratory tract infections	3	6

In the present study, delayed milestone was the commonest symptoms constituting 47(94%) of the cases followed by convulsions i.e. 33 (66%) cases feeding difficulties and excessive drooling constituted 28 (56%) of cases, speech defects 12 (24%) of the cases.

Table 4: Perinatal factors

Perinatal factors		No. of cases	percentage
Birth asphyxia		33	66
Abnormal labor	prolonged	20	40
	assisted forceps	3	6
	LSCS	1	2
Prematurity		9	18

Asphyxia, presumptive diagnosis of birth asphyxia based on early neonatal events like delayed cry, cyanosis, limpness was obtained in 33 (66%) of cases.

Abnormal labour: in 40 % of case s, labour is prolonged and in one case (2%) it is LSCS.

Prematurity: in 9(18%) of total cases it was retrospectively diagnosed as premature taking into account gestational period and size of the baby at birth, as birth weight could not be accurately reported by the parents.82% were born at term.

Birth trauma: in our study, no cases of birth trauma was noted

Table 5: Post natal factors

Postnatal factors	No.of cases	percentage
Neonatal jaundice	4	8
Tb meningitis	1	2
Pyogenic meningitis	1	2
Others (encephalopathy or convulsions)	1	2

Jaundice: in the present study 4(8%) cases of cerebral palsy had severe neonatal jaundice. Both had a history of exchange transfusion. In one case Rh incompatibility as the cause was confirmed by blood grouping.

Convulsions: convulsion in the neonatal and post neo natal period without any apparent cause was the only possible aetiology in 1(2%) of cases.

Infections: In the present study there was 1(2%) case of pyogenic meningitis and 1(2%) of TB meningitis.

Trauma: No case of cerebral palsy following trauma was seen in the present study.

Table 6: Clinical types of cerebral palsy

Physiological distribution	No. of cases	Percentage
Spastic	41	82
Atonic	5	10
Mixed	4	8
Total	50	100

In the present study spastic type was the most common type, accounting 41(82%) of the total cases. The incidence of hypotonic type was 5(10%). There were 4(8%) mixed type, one showing combination of hypotonia with athetosis and other three are spasticity with athetosis. there was no case of cerebral palsy with only athetosis in our study.

Table 7: Topographic distribution

Topographic distribution	No. of cases	Percentage
Quadriplegia	28	56
Diplegia	15	30
Hemiplegia- right side	5	10
Left side	2	4

In the present study, quadriplegia was the commonest 28(56%) neurodeficit followed by diplegia 15(30%). There are 7 (14%) cases of hemiplegia, among these 5 were right sided and 2 were left sided.

Table 8: Functional types

Functional type	No. of cases
No limitation of activity	-
Slight limitation of activity	7
Moderate great limitation of activity	11
Severe/no useful activity	32
Total	50

In our study, 11(22%) cases were of moderate great limitation of activity, 7(14%) were having slight limitation of activity and 32 cases have severe or no useful activity. No case was observed without any limitation of activity.

Table 9: Associated defects

Associated defects	No.of cases	percentage
Subnormal intelligence	47	94
Convulsion	33	66
Ocular defects		
squint	5	10
Optic atrophy	1	2
Speech defects	22	44
Micro cephalous	20	40
Auditory defects	6	12

In this study, the most commonest is subnormal type of I.Q deficit in 47 (94%) of the cases followed by convulsion, speech defects, microcephaly and auditory defects 66%, 44%, 40% and 12% respectively. Visual defects were present in 6(12%), among which 5 cases non- paralytic squint and in one case optic atrophy were noted.

DISCUSSION

The present study of cerebral palsy was conducted to determine the clinical profile and predisposing factors. This study includes 50 consecutive cases of cerebral palsy in the age group of 0-12 years admitted to government general hospital, Basaveshwar teaching & general hospital and sangameshwar hospital, Gulbarga attached to M.R medical college, Gulbarga during the period from March 2001 to march 2003.

Table 10: Age distribution

Age	Present study no. and percentage	Sharma *et al.*[4]; no. and percentage
Less than one year	4(8)	42(19.2)
1-5 years	26(52)	113(51.6)
5-10 years	18(36)	52(23.7)
More than 10 years	2(4)	12(5.5)

In our study the maximum number of patients were in the age group of 1-5 years i.e 26 (52%) and 5-10 years 18 (36%). in the study by Sharma [4] *et al.;* similar findings where in 51.6% were in the age group of 1-5years. This is because, during this period developmental retardation becomes more and more obvious and parent becomes worried and the child is brought to the hospital.

Table 11: Sex

Study	Male		female	
	No.	percentage	No.	percentage
Garge *et al.;* (1965)	74	59.7	50	40.3
Misra *et al.;* (1973)	39	62.9	23	37
Sharma *et al.;* (1981)	150	68.5	69	31.5
Present study	36	72	14	28

A high incidence of the male sex has been reported by garg [8] *et al.;* Misra [4] et *al.;* and Sharma [4] *et al.;* and is consistent with our findings.

CLINICAL FEATURES:

In the present study, delayed milestones was the commonest symptom in 47 (94%) cases, for which the child was brought to the hospital .Prabhakar quotes that almost 80% of cases present with this symptoms. Udani pm (1963) [9] also quotes that it is the commonest symptom. Feeding difficulties and excessive drooling and speech defects were the next common symptoms.

In our study perinatal is 66% and post natal is 14%. In the study by Srivastava *et al.;* in 1992 [10] post natal was 26.1 and garg *et al.;* reported 25% [8].

Prematurity- Hagberg B and Hagberg G [11] reported 68% of their diplegics were born preterm. In our study diplegics were born preterm in 80%. Our findings of 18% premature are comparable to the findings of Manchanda [12] and garg *et al.;* [8]. A high incidence of prematurity is seen in diplegics. This association is due to

a) The fact that certain periventricular vascular structures make this region (periventricular of the preterm infant particularly vulnerable to brain ischemia)
b) The pressure –passive cerebral circulation of preterm of infants, particularly when sick and generally depressed in state.
c) The enhanced vulnerability of the actively differentiating and /or myelinating periventricular glial cells.

Malpresentation- in our study no case is found, which can be compared to Manchanda [12].

Abnormal labour- in our study 48% cases was of abnormal labor, garg *et al.;* reported 33% of abnormal labour cases [8]. In our study, history of forceps application is in 6%, which is higher than the reports of Manchanda [12] and garg *et al.;* [8]. History of LSCS was present in 2% which is comparable to garg *et al.;* (1.6%) [8].

The fact that delivery was abnormal does not prove that it was the cause of cerebral palsy.

Asphyxia- presumptive diagnosis of birth asphyxia was obtained in 33(66%) of our cases. Garg *et al.;* [8] reported 28.2% and Manchanda [12] 66.9% which is comparable with our study.

Thus most of the perinatal factors, except genetic and birth trauma appear to play a significant role in the genesis of cerebral palsy often simultaneously and/or inter dependently operating in the same patient. Our study findings were in consistent with Manchanda, which gives ahigh incidence of birth asphyxia as 66.9%. Lower figures can be ascribed to improved neonatal care.

Jaundice- in the present study severe neonatal jaundice accounted for 4 (8%) of total cases, and is comparable with figures quoted by udani pm [9] 6%, garg *et al.;* [8] 6.5%, Sharma *et al.;* [4] 6.8%.

Convulsions – convulsion in the neonatal and post natal period without any apparent cause was the only possible etiology in 1(2%) cases. Udani pm [9] reported an incidence of 14% and Sharma *et al.;* [8] 0.9%.

Infections- in the present study, there was one case of cerebral palsy following pyogenic meningitis and one case following tuberculous meningitis put together 4%. Sharma *et al.;* [8) reported an incidence of 6.8% for meningitis.

Stanley fj [13] (1982) reported that over 11% of all cases of cerebral palsy in western Australia over a 20 years period apparently were due to postnatally acquired brain damage. This corresponds well with our findings of 7(14%) Of cases.

In the present study spastic type was the most common, according for 82% of total cases. This finding is consistent with that of Sharma et al.; (4, 14), Misra et al.; [4] and garg et al.; [8)]. The incidence of hypotonic type in the present study was 10% cases. The finding is consistent with those of Misra et al.; [4] and garg et al.; [8]. Udani pm [9] reported a higher incidence of hypotonic type (28%). There were no mixed type in our study comparable with Misra et al.; In our study, the dyskinetic type were of mixed type of i.e. 1 case with hypotonia and athetosis and 4 with spastic athetosis, there are no cases of cerebral palsy with only athetosis or tremor in the present study.

In the present study, diplegia was in 30% of cases. Sharma *et al.;* [4, 14] 45.3% quadriplegia is common in our study with 56%, whereas garg [8] 34.8%. In our study 7(14%) are hemiplegic and Sharma [4, 14] and garg *et al.;* [8] 34.8%. udani also reports a higher incidence of right side, as also in our study. There were no cases of monoplegia, paraplegia or triplegia in our study.

Table-12: Perinatal factors

Study	Prematurity (%)	Malpresentation (%)	Abnormal labor			Birth asphyxia (%)
			prolonged	Assisted		
				forceps	LSCS	
Udani pm (1963)	9	4	24	-	-	25
Garg *et al.;* (1965)	35	12.1	15.3	3.2	1.6	28.2
Manchanda(19630	27	-	-	4	-	66.9
Present study	18	-	40	6	2	66

Table 13: post natal factors

Study	Jaundice (%)	Convulsions (%)	meningitis	Infections (%)		Trauma
				encephalitis	Others	
Udani pm (1963)	6	14	8	22	2	1
Garg et al (1965)	6.5	-	8	6.5	-	-
Sharma(1981)	6.8	-	6.8	-	-	-
Present study	8	2	2	-	2	-

Table-14: Clinical types

Study	Distribution of cerebral palsy according to clinical types					
	Spastic (%)	Dyskinetic (%)		Ataxia (%)	Hypotonia (%)	Mixed (%)
		athetosis	tremors			
Udani pm (1963)	59	22	6	12	28	27
Garge *et al.;* (1965)	74.2	14.5	-	-	6.5	-
Mishra(1973)	77.4	9.7	-	4.8	8.1	-
Sharma(1981)	82.7	4.1	-	0.9	5	7.3
Present study	82	8	-	-	10	-

Table 15: Topographic distribution of cerebral palsy

Topographic type		udanipm	Garg *et al.;*	Sharma *et al.;*	Present study
Diplegia		10.2%	-	45.3%	30%
Quadriplegia		25.3%	34.8%	4.4%	56%
Hemiplegia	Right	30.5%	2%	34.8%	10%
	Left	23.56%	16.3%	-	4%
Paraplegia		6.7%	17.4%	-	-
Triplegia		1.7%	-	5.5%	-
Monoplegia		-	6.5%	4.2%	-

Table 16: Associated defects

Study	Speech (%)	Microcephaly (%)	Convulsion (%)	Visual defects (%)	Auditory defects (%)
Udani pm	25	13	58	5	5
Garge	43.4	-	23.4	34.6	16.8
Sharma et al	3.6	14.2	8.2	10.5	10
Kant	45.3	-	23.1	17.5	5.5
Present study	44	40	66	12	12

Speech defect- in our study it is 44% of patients. It is comparable with those of Kant [15], 45.3% and garg [8], Manchanda [12].

Microcephaly- in our study microcephaly was present in 405 of cases, Sharma *et al.;* [4, 14] udani pm [9] and Manchanda [12] found in 14.2%, 13 %and 9% cases respectively.

Convulsions-In our study convulsion were present in 66% of patients, which is comparable to udani [9] (58%)

Visual defects – in our study visual defects were present in 12% of cases. Our figures are comparable to the figures reported by Kant *et al.;* [15] (17.5%)

Auditory defects –in our study, 12% of cases were having auditory defects, which is comparable to the figures reported by Sharma [4] (10%) and garg [8] (16.8%).

CONCLUSIONS

The present study includes 50 consecutive cases of cerebral palsy admitted to government general hospital, Basaveshwar teaching & general hospital and sangameshwar hospital, Gulbarga attached to M.R medical college, Gulbarga during the period from march 2001 to march 2003.From the analysis of the result of the above case studied, following conclusion can be drawn

1. Majority of the cerebral palsy i.e. 52% cases were between the age group of 1-5 years.
2. The incidence of cerebral palsy was more in males than in females with a male to female ratio of 2.5:1
3. The common clinical symptoms for which the child was brought to the hospital were delayed milestones, feeding difficulties, excessive drooling, speech defects and convulsions.
4. The commonest physiological presentation is spastic type(82%0 and the commonest topographical presentation is quadriplegia accounting for 56% followed by diplegia (30%) and hemiplegia (14%) in which right sided hemiplegia(12%) and left side hemiplegia (2%)
5. Mental retardation, convulsion, speech defects, microcephaly, visual, auditory defects were the

common associated findings with cerebral palsy.

REFERENCES

1. Robert HA, Haslam, textbook of Ped, nelson14th edn. Kuban ck, Leviton a, "cerebral palsy", new Engl, j med. 1994; 330:188-195.
2. Pediatric rehabilitation by Gabriela Molnár, 2ndedn, chap, 11; p.193-217.
3. Misra pk, Sharma b," cerebral palsy; a clinical study", archives of child health, July 1973:183-189. Pcna, June 1993; vol.40; no.3.
4. Bax MC. Terminology and classification of cerebral palsy. Developmental Medicine & Child Neurology. 1964 Jun 1; 6(3):295-7.
5. Alfred l Scherzer, Ingrid tscharmnuter, early diagnosis and therapy in cerebral palsy, 2nd edition, p.8, 10.12.
6. Singhi SD, goraya js, "cerebral palsy", Indian pediatr, 1998; 35:37-48.
7. Garg BK, Srivastava JR. Cerebral palsy: a clinical study of 124 cases with a review. Indian pediatrics. 1965 Jun; 2(6):195-208.
8. udani pm," cerebral palsy: a study of 100cases", Ind. J.Child health, 1963; 12:755-770.
9. Srivastava vk, Laishram n, Srivastava rk, "cerebral palsy", Indian pediatr 1992; 29:993.
10. Hagberg b, Hagberg g, in: "recent advances in Ped", 11:p.67-84.
11. Manchanda S. Cerebral palsy. A review of 275 cases. The Indian journal of child health. 1963 Jan; 12:1.
12. Stanley fj, eve Blair, dev. Med, child. Neurol, 24: 575-585.
13. Sharma M, Saxena S, Prakash P. A study of cerebral palsy with special reference to psycho-social and economic problems. Indian pediatrics. 1981 Dec; 18(12):895.
14. Kant L, Dewan S. Associated handicaps in cerebral palsy. Indian pediatrics. 1986; 23(11):954-6.