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A Comparison of Spastic Diplegic and Tetraplegic Cerebral Palsy

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The aim of this study was to compare spastic diplegic and tetraplegic cerebral palsy. Thirty-eight children had spastic diplegic cerebral palsy and 48 spastic tetraplegic cerebral palsy. Risk factors of cerebral palsy, seizures, severity of cerebral palsy, electroencephalogram, and magnetic resonance imaging findings were analyzed. Gestational history, low birth weight, and perinatal pathologies were present in similar percentages in both groups. Lower values of the Apgar score were recorded more often in the tetraplegic cerebral palsy group than the diplegic group. The children with spastic diplegia were classified more frequently into levels I and II of the Gross Motor Function Classification System, but patients with spastic tetraplegia were classified more frequently into levels IV and V. Similarly, mental retardation was observed more frequently in the patients with spastic tetraplegia. In magnetic resonance imaging, periventricular leukomalacia was detected in a higher proportion of children with spastic diplegia than in patients with tetraplegia. Cerebral atrophy occurred more frequently in the tetraplegic group compared with diplegic patients. Twenty-four (50.0%) children with spastic tetraplegia had epilepsy compared with six children with spastic diplegia. The incidence of intractable epilepsy was higher in the tetraplegic patients than in the children with spastic diplegia. **© 2005 by Elsevier Inc. All rights reserved.**

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Introduction

The motor abnormalities that define cerebral palsy are often accompanied by other neurologic problems, including cognitive impairment, seizure disorders, or impairment in vision, hearing, or speech [1-4]. Spastic diplegia cerebral palsy is the most common cerebral palsy syndrome in children born at preterm [1,2]. Spastic tetraplegia (quadriplegia) is the most severe type of cerebral palsy, with pareses of the upper limbs being of the same degree or more severe than those of the lower limbs [1]. Epilepsy and cognitive deficits are common in spastic tetraplegia and rare in spastic diplegia [2,3]. Magnetic resonance imaging provides detailed information about the brain lesions. It seems to be sensitive in determining the type, site, and severity of lesions of prenatal or perinatal hypoxic-ischemic encephalopathies, which are the most important factors in cerebral palsy [5-7]. Some authors have recommended routine magnetic resonance imaging studies in children with cerebral palsy as an assessment of etiology and a predictor of outcome [5,6]. Recently, we have compared two forms of hemiparetic cerebral palsy [4].

The present study compares risk factors, clinical patterns, and magnetic resonance imaging in predicting motor impairment and cognitive disabilities in children with spastic diplegic cerebral palsy and spastic tetraplegic cerebral palsy.

Patients and Methods

The medical records of children with cerebral palsy referred to our Pediatric Neurology and Rehabilitation Department in Bialystok from October 1994 to September 2004 were reviewed. Eighty-six children (49 males and 37 females, mean age 7.89 ± 4.20 years, range 4-15) with spastic diplegic cerebral palsy and tetraplegia were investigated. Of these children, 48 had spastic tetraplegic cerebral palsy, and 38 had spastic diplegia. The diagnosis of cerebral palsy was confirmed in each case by

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the authors. Children with postnatal meningitis, encephalitis, trauma, and metabolic or degenerative disorders were excluded from the study.

Motor Function

The children were each assigned a Gross Motor Function Classification System according to Palisano et al. [8] by an occupational therapist. Level I - walks without restrictions; II - walks without assistive devices, limitations in walking outdoor; III - walks with assistive devices; IV - self-mobility with limitations, children are transported or use powered mobility; V - self-mobility is severely limited.

Cognitive Function

The patients were assigned to one of three groups, depending upon their level of academic achievement, supplemented by the results of formal psychological testing: (1) Normal: normal school performance to at least first grade level, with no evidence of specific learning difficulties. All the children in this group had one or more formal psychological assessments (the typical Wechsler Intelligence Scale for Children, Polish version or Terman Merril). (2) Mentally handicapped: formal psychological testing results indicated function in the mentally deficient range. (3) Mental retardation was divided into the following: mild: 70-84 intelligence quotient; moderate: 50-69 intelligence quotient; severe: <50 intelligence quotient. Normal children had intelligence quotient >90. Eleven children with spastic tetraplegia were not testable because they either were at behavioral observation, too mentally impaired, or uncooperative to obtain a standardized score.

Electroencephalography

Electroencephalographic signals were recorded during wakefulness or sleep in uncooperative patients from scalp electrodes (according to the International 10/20 System), all correlated with the vertex reference. The signals were recorded, amplified, and filtered by a Medelec DG Compact 32 (Surrey, Great Britain). Ag/AgCl electrodes with an impedance less than 5 kΩ were used. Frequencies below 1 Hz and above 70 Hz were eliminated by digital filtering.

Magnetic Resonance Imaging

All magnetic resonance scans were obtained using a 1.5 T magnetic resonance scanner (Picker Edge Eclipse) with the use of a standard circularly polarized head coil. The images were assessed by the neuroradiologists and separately by the child neurologists (W.K., W.S.), who were unaware of the prenatal and perinatal histories or of the results of clinical evaluations. The magnetic resonance diagnosis in any patient with multiple abnormal findings was based on the dominant finding. Cerebral atrophy was diagnosed when diffuse sulcal widening of the cerebrum with symmetrical ventricular dilatation without periventricular signal abnormalities was observed [9]. Periventricular leukomalacia was diagnosed in patients who had ventriculomegaly with irregular outlines of the body and trigone of the lateral ventricle, a reduced quantity of periventricular white matter, deep prominent cerebral sulci, and periventricular signal abnormalities of low intensity on T_1-weighted images and high intensity on T_2-weighted images. Imaging sequences: T_1-weighted FAST scan (repetition time 300 ms, echo time 4.5 ms, 5-mm-thick sections) pre- and post-intravenous contrast media administration (0.1 mmol/kg Megnevist, Schering) were performed. Fast spin-echo T_2-weighted and fluid-attenuated inversion-recovery series (repetition time 5000 ms, echo time 127.6 ms, 5-mm-thick sections) were used.

Definitions

Cerebral palsy was defined as motor disabilities caused by nonprogressive damage to the developing brain [2]. Cerebral palsy was classified as spastic tetraplegia (spasticity of all four limbs and of similar involvement), and spastic diplegia (spasticity of lower limbs being greater than upper).

Epilepsy was defined as a separate occurrence of two or more apparently unprovoked seizures [10]. The seizure outcome was defined as good if the patient was seizure-free for more than 2 years. Intractable epilepsy was defined as two seizures per month despite appropriate drug therapy [11]. The epileptic seizures were divided into the following three groups: (1) partial (including simple partial, complex partial, and partial with secondary generalization); (2) generalized (generalized seizures other than infantile spasms, including tonic, tonic clonic, myoclonic, and atypical absence seizures); and (3) the Lennox-Gastaut syndrome.

Prematurity was defined by the World Health Organization as an infant with a gestation of less than 37 weeks from the first day of the last menstrual period. Severe prematurity was defined as an infant with a gestation of less than 28 weeks. Asphyxia is defined as an Apgar score ≤4. Prenatal pathology included preeclampsia, premature disruption of the placenta, bleeding, and sepsis. Diagnosis of a mental abnormality was based on clinical assessment supplemented by standard tests if available at the time of diagnosis, and the need for special education.

Statistical Analysis

The differences between the groups were determined by the parametric *t* test and nonparametric statistical tests: Fisher's Exact Test or chi-square test were used where appropriate. All *P* values were two-tailed. Statistical significance was defined as $P < 0.05$. Statistics were calculated using Statistica 6.0. The study was approved by the Ethics Committee of the Medical University of Bialystok.

Results

Clinical Data

The clinical data for all patients are summarized in Table 1. Eighty-six (49 males, 37 females) patients with cerebral palsy were recruited. Forty-eight children had spastic tetraplegic cerebral palsy and 38 had spastic diplegic cerebral palsy. No differences in severe preterm, preterm, and term between spastic diplegia and tetraplegia were observed. A greater number (not significantly) of pregnancies and deliveries were observed in the spastic diplegic cerebral palsy group as compared with the tetraplegic cerebral palsy group. Similarly (but insignificantly), more asphyxia was recorded in the tetraplegic cerebral palsy group than in the spastic diplegic cerebral palsy group. No differences in birth weight between the groups were observed. A significantly greater number of children with spastic diplegia were classified into levels I and II of the Gross Motor Function Classification System compared with the tetraplegic group. On the other hand, the patients with spastic tetraplegia were classified more frequently into levels IV and V of the Gross Motor Function Classification System than were patients with spastic diplegia. The locomotion function was affected in similar proportions in the tetraplegic and diplegic cerebral palsy children with or without epilepsy (data not presented).

Table 1. Characteristics of subjects with spastic tetraplegia and spastic diplegia cerebral palsy

Variable	Tetraplegia (n = 48)	Diplegia (n = 38)	*P* Value
Gestation	26-41	25-41	NS
	35.68 ± 2.74	34.48 ± 4.58	
Female/Male	21/27	16/22	NS
Preterm	26	21	
Term	22	17	NS
Number of pregnancies	1-6	1-7	NS
	2.04 ± 1.45	2.29 ± 1.60	
Number of deliveries	1-4	1-7	
	1.60 ± 1.01	1.96 ± 1.34	NS
Apgar score at 1 min			
<4	23	12	NS
>4	25	26	NS
Weight at birth (gm)	1100-4000	850-4150	
	2664 ± 796	2480 ± 1047	NS
GMFCS levels, n			
I	0	17	<0.001
II	1	16	<0.05
III	10	5	NS
IV	27	0	<0.001
V	10	0	<0.01
Mental development			
Normal	0	8	<0.01
Small delay	4	24	<0.001
Mild	17	6	NS
Severe	27	0	<0.001
Epilepsy	24	6	<0.05

P value from *t* test and Fisher's Exact Test between groups.

Abbreviations:
GMFCS = Gross Motor Function Classification System
NS = Not significant

Similarly, mental development differed significantly between the groups. A higher proportion of epilepsy in tetraplegic cerebral palsy was documented. Significant differences in milestones (sitting, standing, walking, and speaking) of children with spastic diplegic cerebral palsy and in children with tetraplegic cerebral palsy were evident (data not shown).

Forty-seven (54.6%) children with spastic tetraplegia and diplegic cerebral palsy were born preterm, and 39 (45.3%) at term. The prenatal risk factors of asphyxia and low birth weight were observed in 44 (51%) and 40 (46.5%) patients, respectively. Perinatal risk factors (prelabor rupture, abruptio placenta, fetal distress, preeclampsia, respiratory distress syndrome, and sepsis) ranged from 5% to 39% in spastic tetraplegic and diplegic cerebral palsy children.

Risk Factors

Male gender was not associated with an increased risk of tetraplegic cerebral palsy or diplegic cerebral palsy, nor was gestational history related to an increased risk of tetraplegic cerebral palsy or diplegic cerebral palsy (Table 2). The percentages of cesarean sections in both groups were comparable. Very low birth weight (<1500 gm) and low birth weight (<2500 gm) were not significantly associated with an increased risk of tetraplegic cerebral palsy or diplegic cerebral palsy. Perinatal pathologies (prelabor rupture, abruptio placenta, fetal distress, preeclampsia, respiratory distress syndrome, and sepsis) were present in similar proportions in the tetraplegic cerebral palsy and the diplegic cerebral palsy groups.

Magnetic Resonance Imaging

Significant abnormalities relevant to the paresis were evident on imaging in 83 (96.6%) patients (Table 3). Three children had normal magnetic resonance imaging scans. A similar percentage of the magnetic resonance imaging abnormalities were detected in both groups, 48 (100%) in patients with tetraplegic cerebral palsy and 35 (92.1%) in patients with diplegic cerebral palsy. The most common finding on magnetic resonance imaging was periventricular leukomalacia in 50 (58.1%) patients, with a higher proportion in diplegic cerebral palsy children. Periventricular leukomalacia was observed in a similar proportion in preterm or term patients (data not shown). Porencephalic cysts were evident in eight patients. Of the eight cysts, five were present in patients with spastic tetraplegia, and three in spastic diplegic children. Cerebral atrophy was observed more often ($P < 0.05$) in children with spastic tetraplegia than in diplegic patients, 15 (31.2%) vs 2 (5.2%). Multicystic encephalomalacia lesions were present in two patients with spastic tetraplegia. An open-lip schizencephaly was found only in two children with spastic tetraplegia. Similarly, agenesis of the corpus callosum was observed in two patients with tetraplegia. Holoprosencephaly was observed in one child with spastic tetraplegia and in one with spastic diplegia.

Electroencephalography

We found the abnormalities to be predominantly generalized and multifocal in the electroencephalographic recordings (Table 4). The electroencephalographic abnormalities in the nonepileptic tetraplegic cerebral palsy patients included multifocal changes in eight children, paroxysmal generalized in six patients, focal changes (slow or sharp waves) in the left or right hemisphere in three children, and in the centro-parietal area in four children Only one electroencephalographic recording was normal.

All electroencephalograms in children with tetraplegic cerebral palsy and epilepsy were abnormal. Paroxysmal changes were observed in 12 tetraplegic cerebral palsy patients and multifocal in six. Focal changes in the left hemisphere were detected in four patients, in the right hemisphere in two patients, and in the centro-parietal area in two patients.

The electroencephalographic abnormalities in the nonepileptic diplegic cerebral palsy patients included multifocal changes in seven children, paroxysmal generalized in

Table 2. Risk factors of children with spastic tetraplegia and spastic diplegia cerebral palsy

Variable	Tetraplegia n (%) (n = 48)	Diplegia n (%) (n = 38)	Tetraplegia vs Diplegia OR (95% CI)	*P* Value
Male sex	27 (56.2)	22 (57.1)	0.97 (0.47-1.96)	NS
Asphyxia	23 (47.9)	21 (55.2)	0.86 (0.41-1.79)	NS
Gestational history				
Preterm	26 (54.1)	21 (55.2)	1.14 (0.54-2.38)	NS
Severe preterm	10 (20.8)	8 (21.0)	0.98 (0.35-275)	NS
Term	22 (45.8)	17 (44.7)	1.02 (0.47-2.19)	
Postterm	0 (0)	0 (0)	0	
Cesarean section	16 (33.3)	11 (28.9)	1.15 (0.47-2.76)	NS
Low birth weight <2500 gm	22 (45.8)	18 (47.3)	0.96 (0.45-2.05)	NS
Very low birth weight <1500 gm	4 (8.3)	3 (7.8)	1.05 (0.22-5.00)	NS
Prelabor rupture of membranes	12 (25)	15 (39.1)	0.63 (0.26-1.51)	NS
Abruptio placenta	5 (10.4)	3 (7.8)	1.31 (0.29-5.87)	NS
Fetal distress	8 (16.6)	7 (18.4)	0.90 (0.30-2.71)	NS
Preeclampsia	4 (8.2)	3 (7.8)	1.05 (0.22-5.00)	NS
RDS	7 (14.5)	6 (15.7)	0.92 (0.28-2.97)	NS
Sepsis	4 (8.2)	2 (5.2)	1.58 (0.27-9.11)	NS

Abbreviations:
CI = Confidence interval
NS = Not significant
OR = Odds ratio
RDS = Respiratory distress syndrome

five children, focal changes (slow or sharp waves) in the centro-parietal area in 13 children, and in the left or right hemisphere in three children. Four children had normal electroencephalographic recordings.

All electroencephalograms in children with diplegic cerebral palsy and epilepsy were abnormal. Paroxysmal changes were documented in three diplegic cerebral palsy patients, multifocal in two patients, and focal changes in the left hemisphere were detected in only one patient.

Epilepsy

Thirty children with cerebral palsy had epilepsy. Frequency of epilepsy was significantly ($P < 0.05$) higher in the tetraplegic cerebral palsy (24 [80.0%]) than the diplegic cerebral palsy (6 [20.0%]) patients (Table 1). The mean duration of monitoring was 5.45 ± 3.67 years (range 4-13 years). The mean age at onset of epilepsy was 0.95 ± 0.83 years in the tetraplegic cerebral palsy children. Neonatal seizures occurred in seven children with spastic tetraplegic cerebral palsy (Table 5). Status epilepticus affected six children (25%) with tetraplegic cerebral palsy and one child from the diplegic cerebral palsy group. Epilepsy attacks in the tetraplegic cerebral palsy group included generalized seizures in 10 patients, partial secondarily generalized seizures in seven patients, and the Lennox-Gastaut syndrome in seven patients. Partial secondarily generalized seizures were observed in five children with diplegic cerebral palsy, and one had generalized seizures. Fourteen (58.3%) tetraplegic cerebral palsy children had poor seizure control and were on polytherapy. Only two (33.3%) patients of the diplegic cerebral palsy

Table 3. Magnetic resonance imaging abnormalities in children with spastic tetraplegia and spastic diplegia cerebral palsy

MRI Findings	Tetraplegia (%) of Patients n = 48	Diplegia (%) of Patients n = 38	*P* Value
Normal	0	3 (7.8)	NS
PVL	21 (43.7)	29 (76.3)	NS
Cerebral atrophy	15 (31.2)	2 (5.2)	<0.01
Posthemorrhagic porencephaly	5 (10.4)	3 (7.8)	NS
Multicystic encephalomalacia	2 (4.1)	0 (0)	NS
Holoprosencephaly	1 (2)	1 (2.6)	NS
Schizencephaly	2 (4.1)	0 (0)	NS
Agenesis of corpus callosum	2 (4.1)	0 (0)	NS

Abbreviations:
MRI = Magnetic resonance imaging
NS = Not significant
PVL = Periventricular leukomalacia

Table 4. Visual analysis of EEG in children with spastic tetraplegia and spastic diplegia cerebral palsy

EEG Findings	Tetraplegia n = 48	Diplegia n = 38
	Nonepileptic n = 24	Nonepileptic n = 32
Normal	1	4
Multifocal*	8	7
Focal right hemisphere	1	2
Focal left hemisphere	2	1
Focal centroparietal	6	13
Paroxysmal generalized	6	5
	Epileptic n = 24	Epileptic n = 6
Normal	0	0
Multifocal*	6	2
Focal right hemisphere*	2	0
Focal left hemisphere	4	1
Focal centroparietal*	0	0
Paroxysmal generalized	12	3

* Sharp and slow waves.

group had intractable epilepsy. Ten (41.6%) tetraplegic cerebral palsy children had their epilepsy well controlled by monotherapy. Four (66.6%) of the diplegic cerebral palsy patients were on monotherapy.

In the present study, polytherapy with no more than two first-line antiepileptic drugs (carbamazepine or valproate) and second-line antiepileptic drugs (vigabatrin, lamotrigine, clonazepam, nitrazepam) was administered. Our department discontinues antiepileptics after patients with cerebral palsy are free of seizures for at least 3 years. Antiepileptic drugs were not discontinued in any patients with spastic diplegia or tetraplegic cerebral palsy (data not shown).

Discussion

In the present study, some significant differences were found between the clinical patterns of paresis in the tetraplegic cerebral palsy and diplegic cerebral palsy groups. Gestational history was not related to an increased risk of tetraplegic cerebral palsy or diplegic cerebral palsy. Similar proportions of cesarean sections were noted in both groups. Similar percentages of low birth weight, perinatal pathologies (prelabor rupture, abruptio placenta, fetal distress, preeclampsia, respiratory distress syndrome, and sepsis) were present in the tetraplegic cerebral palsy and diplegic cerebral palsy patients. These findings are partially in agreement with the study of Hagberg and Hagberg [12]. In Sweden in the years 1954-1990, they observed the continuously increasing number of surviving very preterm infants. Bilateral spastic cerebral palsy, including spastic and ataxic diplegia, tetraplegia, and spastic-dyskinetic cerebral palsy was the most prevalent clinical group of cerebral palsy syndromes, and presented in approximately 75% of preterm and 45% of term cerebral palsy. In the present study, 54.6% of children were found to be born prematurely.

Tetraplegic cerebral palsy children and the diplegic cerebral palsy children were different with respect to motor and mental development. The severity of spastic cerebral palsy differed between the two types of children. Our findings are in accordance with previous studies [12,13].

Beckung and Hagberg, [13] in a population-based series of children with cerebral palsy, studied birth characteristics, data on gross motor function, and level of handicap at 5 to 6 years of age. Low handicap scores and mild levels of gross motor disability were present in children with hemiplegic cerebral palsy; moderate scores in children with diplegic cerebral palsy, simple ataxia, and athetotic cerebral palsy; and high scores were recorded in children with dystonic cerebral palsy and tetraplegic cerebral palsy.

Periventricular leukomalacia is a form of hypoxic-ischemic damage typical of the immature brain and is most commonly observed as a complication of preterm birth. Because this lesion was observed in children born at term, it was considered to reflect a cerebral injury that had

Table 5. Characteristics of epilepsy in children with spastic tetraplegia and spastic diplegia cerebral palsy

	Tetraplegia n (%) n = 24	Diplegia n (%) n = 6	*P* Value*
Neonatal seizures	7 (29.2)	0 (0)	NS
Seizures during the first year of life	14 (58.3)	1 (16.6)	NS
Status epilepticus	6 (25)	1 (16.6)	NS
Types of epilepsy			
PSG	7 (29.2)	5 (83.3)	NS
Generalized	10 (41.6)	1 (16.6)	NS
Lennox-Gastautsyndrome	7 (29.2)	0 (0)	NS
Intractable epilepsy	14 (58.3)	1 (16.6)	NS
Monotherapy	10 (64.2)	4 (66.6)	NS
Polytherapy	14 (58.3)	2 (33.3)	NS

* Fisher's Exact Test between groups.

Abbreviations:
NS = Not significant
PSG = Partial seizures secondary generalized

occurred in utero. Cerebral maldevelopment caused by an early intrauterine lesion (cortical/subcortical lesions) is also considered to be of major importance in diplegia and tetraplegia [6,9,14].

In the present report, the most common finding on magnetic resonance imaging was periventricular leukomalacia in 58.1% of the patients, the proportions of which were similar in the tetraplegic cerebral palsy and the diplegic cerebral palsy groups. However, periventricular leukomalacia was observed slightly more frequently in children with spastic diplegia. It is important to note that almost half the cerebral palsy children with periventricular leukomalacia were born at term, with no history suggestive of perinatal asphyxia and low birth weight.

In contrast to our findings, Miller et al. [15] reported 12 term children with periventricular leukomalacia to delineate its long-term clinical correlates. The reason for the assessment was developmental delay in 10 (83.3%) patients, one with seizure, and one patient with attention-deficit hyperactivity disorder. Three children had normal motor examinations, three were hypotonic, three had spastic diplegia, two had spastic quadriplegia, and one manifested fine-motor abnormalities. Nine children (75%) manifested developmental delay (severe global delay in six), and two children (16.7%) had epilepsy; electroencephalograms were abnormal in six (50%). They concluded that periventricular leukomalacia in term children presented with a spectrum of neurologic abnormalities, particularly developmental delay and heterogeneous motor findings not limited to classic spastic diplegia.

Cerebral atrophy was observed more often in children with spastic tetraplegia than in patients with spastic diplegia. Our findings are in accordance with earlier studies [9,14].

Kwong et al. [6] analyzed magnetic resonance brain imaging in 122 children with spastic cerebral palsy. Forty-three patients had spastic hemiplegia, 61 had spastic diplegia, and 18 had spastic tetraplegia. Magnetic resonance imaging abnormalities were observed in 75% of patients. Periventricular leukomalacia accounted for 66% of abnormalities observed in patients with spastic diplegia. In the patients with spastic tetraplegia, two types of magnetic resonance imaging abnormalities predominated: congenital brain anomalies and term-type brain injuries, 42% and 33% respectively. In contrast to our findings, they reported a high incidence of congenital brain anomalies in the tetraplegic children; however, their group was smaller than ours. In the present study, only 10% of these brain anomalies were found in children with tetraplegia.

An overall percentage of electroencephalographic abnormalities of 94.1% in the tetraplegic and diplegic cerebral palsy children was documented. We observed similar results in our earlier report on hemiparetic cerebral palsy [4]. Electroencephalographic abnormalities were detected in all of the epileptic tetraplegic and diplegic cerebral palsy children. Only four nonepileptic diplegic and one tetraplegic cerebral palsy patients had normal electroencephalographic recordings. Focal and multifocal changes were observed among both nonepileptic and epileptic spastic cerebral palsy children. Epileptiform activity was observed more frequently in the epileptic than in the nonepileptic group of tetraplegic cerebral palsy. The present study is in agreement with Al-Sulaiman's report [16]. He studied 151 patients with different forms of cerebral palsy. The electroencephalographic abnormalities in the seizure group included slow waves and epileptiform activity (including isolated sharp waves, isolated spikes, and spike-wave and polyspike-wave complexes). Only six electroencephalographic recordings were normal, leading to an overall percentage of abnormality of 92.6%.

In the present study, epilepsy affected 34.8% of the children with tetraplegic and diplegic cerebral palsy. The frequency of epilepsy differed with the two forms of spastic cerebral palsy (50.0% for tetraplegic cerebral palsy and 15.7% for diplegic cerebral palsy). Our results are similar to those of earlier reports [3,17-21]. Zafeiriou et al. [17] studied 178 patients with cerebral palsy and epilepsy, and compared them to a control group of 150 epileptic patients without cerebral palsy. The overall prevalence of epilepsy was 36.1%. Almost 56% of children with tetraplegia had epilepsy. First seizures occurred during the first year of life in 69.7% of patients with epilepsy and cerebral palsy. In the present study, the mean age of the onset of epilepsy was 0.95 ± 0.83 years in spastic tetraplegia. Epilepsy was present for 4 years in diplegic patients.

Carlsson et al. [20] evaluated 146 children with cerebral palsy and epilepsy. The frequency of epilepsy was found to be 38%. All children with tetraplegic cerebral palsy and about one third of the children with other cerebral palsy types developed epilepsy. Children with tetraplegic cerebral palsy tended to have an earlier onset of epilepsy than children with other cerebral palsy types.

Neonatal seizures are more commonly observed in patients with cerebral palsy and epilepsy than in cerebral palsy [19,21]. The presence of neonatal seizure is considered to be a factor for subsequent development of neurologic disabilities. In this study, neonatal seizures were observed in seven patients with spastic tetraplegia. Status epilepticus is more common in patients with neurologic abnormalities [19,21,22]. In the present study, six of the tetraplegic cerebral palsy children with epilepsy had a history of status epilepticus. Partial secondarily generalized seizures were present in most of our patients, which is in accordance with previous studies [19,20]. Aksu [21] and Delgado et al. [22] both reported that focal or secondarily generalized seizures were common in children with cerebral palsy, whereas primary generalized epilepsies were less frequent.

The present study demonstrated that epilepsy in children with tetraplegic cerebral palsy was associated with a relatively poor prognosis. None of the epileptic children with tetraplegic or diplegic cerebral palsy were seizure-free for more than 3 years. This outcome could be related to the higher percentage of intractable epilepsy in our

study. In Kwong's [19] study, 16% of epileptic children with cerebral palsy were seizure-free for more than 2 years, and 12% were seizure-free in Aksu's [21] study.

Several reports mention a significant relationship between mental retardation, motor impairment, and epilepsy [19,23,24]. Carlsson et al. [20] found that children with cognitive impairment had a higher frequency of epilepsy than those without cognitive impairment. These findings are in agreement with our data. In the present study, a higher proportion of magnetic resonance imaging abnormalities was observed in children with tetraplegic cerebral palsy and epilepsy compared with cerebral palsy. Our findings are comparable with previous studies [19,23,24]. This study leads us to conclude that the tetraplegic cerebral palsy and diplegic cerebral palsy forms of cerebral palsy have comparable risk factors but different clinical patterns. Spastic diplegic children were more frequently classified into Gross Motor Function Classification System levels I and II, but spastic tetraplegic patients were classified more often into Gross Motor Function Classification System levels IV and V. Mental retardation and epilepsy were observed more frequently in the patients with spastic tetraplegia cerebral palsy than in patients with spastic diplegia. The incidence of intractable epilepsy was higher in tetraplegic patients.

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