ORIGINAL ARTICLE

Cerebral palsy in children in north-eastern Poland

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Abstract

The aim of this study was to identify antenatal, intrapartum and neonatal risk factors for cerebral palsy (CP) among babies. Antenatal, intrapartum, and neonatal events were compared between 204 children with CP born between 1983 to 2000 and 206 controls, children matched by birth weight, gestational age, and sex via a retrospective case-control method. Antenatal, intrapartum and neonatal factors were expressed as odds ratios and 95% confidence intervals. Multiple logistic regression was conducted, entering only those variables found to be significant at the bivariate level. Factors associated with an increased risk of CP identified as antenatal and intrapartum risk factors were abruptio placenta, pre-labour rupture of membranes, prematurity, preterm labour, cesarean section and low birth weight (<2500 gram). Respiratory distress syndrome, prolonged ventilation, septicemia, meningitis, hyperbilirubinemia, neonatal seizures, and severe cranial ultrasound abnormality were associated with an increased risk of CP in the neonatal period. In the logistic regression models prematurity and Apgar scores ≤ 4 at 1st min were significantly associated with an increased risk of CP. Several antenatal, intrapartum and neonatal risk factors for CP among preterm and term babies are responsible for the etiology of CP. Our findings are in agreement with reports from Western countries. (*J Pediatr Neurol* 2004; 2(2): 79-84).

Key words: neonatal, risk factors, cerebral palsy.

Introduction

During the last 20 years there has been an increase in the survival of preterm babies which was accompanied by a sharp increase in the rate of cerebral palsy (CP) in this group (1,2). Several hypotheses have been proposed to explain the origins of CP in very preterm babies. It may be the result of an ischemic insult in utero leading to both preterm birth and damage to the white matter (3). The immature babies are particularly vulnerable to cerebral hemorrhage and ischemia (4). Neonatal factors such as: seizures, prolonged ventilation, transfusion, ventilation, sepsis, hyponatremia, and total parenteral nutrition are associated with an increased risk of CP (5). Recently, most studies on CP have been carried out in the Western countries with very few having been addressed among the Eastern European countries. In view of this, we tried to identify risk factors associated with CP in north-eastern Poland. A better understanding of the etiology of CP in preterms is necessary for preventive strategies and treatments to be developed. The aim of this study was to investigate antenatal, intrapartum and neonatal risk factors for CP using a hospital-based case control study.

Materials and Methods

Subjects

We reviewed the medical records of children with CP referred to our Department of Pediatric Neurology in Białystok. The hospital serves a population of 1,200,000 in the Podlaskie Province, north-east Poland. The children were referred from various sources including: ambulatory, child health clinics, physiotherapists, pediatricians, and neurologists. All the selected babies (n=204) were born between 1983 and 2000. In each case of CP, diagnosis was evaluated by the authors of the study. Multiple births were excluded from this study as current evidence suggests that the risk factors for CP in this group may differ from those in singleton births (6).

Controls

A total of 206 children without CP were

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Table 1. Characteristics of subjects with CP and controls

Variables	CP subjects (n=204)	Controls (n=206)	P value
Gestational age (week)	24-43 36.60 ± 4.08	26-42 39.01 ± 1.97	<0.001
Girl/boy	85/119	92/114	NS
Number of pregnancies	1-7 2.66 ± 1.55	1-7 2.08 ± 1.30	NS
Number of deliveries	1-7 2.23 ± 0.94	1-7 1.96 ± 1.17	NS
Weight at birth (g)	980-4600 2705 ± 831	1010-4450 3303 ± 527	<0.001

Data are expressed as mean ± standard deviation, P value from *t*-test and Chi-square test between groups. NS: not significant.

included in the study. Controls were selected from the entire geographical population. All the children were healthy and statistically similar age to the CP group as regards age and sex (Table 1).

The following variables were analysed: sex, age, Apgar score, birth weight, prenatal pathology (pre-eclampsia, premature disruption of placenta, bleeding and septicemia) pregnancy, delivery, cesarean section, neonatal seizures and types of CP.

Definitions

CP is defined as motor disabilities caused by non-progressive damage to the developing brain (1). The type of CP are classified into spastic hemiplegia, spastic diplegia, spastic tetraplegia, extrapyramidal, ataxic and mixed types. In each case of CP the diagnosis was confirmed by the authors. The term of prenatal refers to the period before the onset of labour resulting in delivery, perinatal to the period from the onset of labour until the 7th day of life, neonatal to the first 28 day of life, and postnatal to the period from the 28th day of life until 2 years of age. Prematurity was defined by the World Health Organization as a newborn with a gestational age of less than 37 weeks from first day of the last menstrual period. Pregnancy-induced hypertension, or pre-eclampsia, is defined as a blood pressure >140/90 mmHg during the second half of pregnancy in a previously normotensive mother. Maternal disease is defined as the presence of any of the following disease at the onset of the pregnancy: diabetes, asthma, glomerulonephritis and hypertension (5). Hyperbilirubinemia is defined as a maximal bilirubin level >10 mg/dL. Premature rupture of membranes is defined as rupture of membranes that occurred more than 18 hours before delivery. Fetal distress included persistent fetal tachycardia (>200 beats per minute), bradycardia (<100 beats per minute), late fetal heart rate decelerations, poor fetal heart rate variability on fetal monitoring, or a cord pH of less than 7.20.

Severe cranial ultrasound abnormality is defined as a grade 3 or 4 periventricular hemorrhage, parenchymal infarction, periventricular leukomalacia, or ventricular dilatation at discharge (5).

The approval of the Medical University ethics committee was obtained before starting the study.

Statistical analysis

Each of the obstetric and neonatal factors listed in Table 1, in total population of children with CP (n=204) was compared with those matched controls (n=206) using independent Student's 2-tailed *t*-test and Chi-square analyses. The odds ratio associated with a given factor estimates the risk of CP given the factor relative to the risk of CP without the factor. The 95% confidence intervals for crude odds ratios, univariate regression analysis and multiple logistic regression were calculated with the programme (NCSS 2001). Only variables with a significant association with CP remained significant ($P < 0.05$) independent of the other variables. We included these factors in further regression models, looking for relations between antenatal, intrapartum and neonatal factors and CP.

Results

Data were available for all 204 children with CP and 206 controls, a total of 408 babies (Table 1). Mean gestational age at birth for children with CP was 36.1 ± 4.1 weeks versus a mean of 39.0 ± 2.0 , ($P < 0.001$). As this difference in gestational age confounds any comparison between cases and controls, odds ratio estimates were adjusted for gestational age. Further adjustment for birth weight did not affect the results; hence odds ratios are reported without this adjustment.

Distribution of CP

In our study, spastic hemiplegia occurred significantly more frequently occurred in the term group than in the preterm and very preterm

Table 2. Distribution of CP types according to gestational age

CP types	Gestational age			Total n (%)
	< 32 weeks n (%)	32-36 weeks n (%)	≥ 37 weeks n (%)	
Spastic hemiplegia	3 (5.6)	10 (18.8)	40 (75.4) ^{cd}	53 (26)
Spastic diplegia	9 (15.7)	17 (29.8)	31 (54.3) ^{bc}	57 (28)
Spastic tetraplegia	16 (21.3)	19 (24.6)	33 (47.1) ^a	70 (34)
Extrapyramidal	2 (18.1)	1 (9)	8 (72.7) ^e	11 (6)
Mixed	0	2 (22)	7 (77.7)	9 (4)
Ataxic	0	2 (40)	3 (60)	5 (2)
Total	30	51	124	204

P-values from Chi-square test: a: $P < 0.001$, vs <32 weeks, b: $P < 0.01$ vs <32 weeks, c: $P < 0.05$ vs <32 weeks, d: $P < 0.001$, vs 32-36 weeks, e: $P < 0.05$ vs 32-36 weeks.

Table 3. Maternal factors and obstetric histories

Factors	CP subjects n (%)	Controls n (%)	OR (95% CI)	P value
Mean maternal age (year)	28.2 ± 5.9	28.7 ± 5.19	-	0.880
Age < 30 years	159 (88)	148 (72)	1.38 (0.88-2.17)	0.530
Age ≥ 30 years	45 (22)	58 (28)	0.78 (0.50-1.24)	0.322
Bad obstetric history	22 (10.7)	12 (5.82)	1.76 (0.86-3.48)	0.086
Spontaneous abortion	42 (20.5)	22 (10.6)	1.88 (1.11-3.19)	0.012
Maternal disease	19 (9.31)	13 (6.31)	1.47 (0.71-3.06)	0.289
Pre-eclampsia	17 (8.33)	12 (5.80)	1.43 (0.66-3.07)	0.353
Abruptio placenta	28 (13.70)	9 (4.30)	3.14 (1.44-6.82)	<0.01
Placenta previa	21 (10.3)	16 (7.75)	1.32 (0.67-2.58)	0.411
Prelabour rupture of membranes	25 (12.2)	1 (6.31)	2.29 (1.10-4.78)	0.020
Prematurity	85 (41.6)	15 (7.28)	4.18 (2.69-6.50)	<0.001

Data are described as mean ± standard deviation or n (%); OR: odds ratio; CI: 95% confidence interval.

(Table 2). Similarly spastic diplegia and spastic tetraplegia occurred more frequently in the term group as compared with other groups. There were no significant differences between preterm and very preterm groups.

Maternal factors

Maternal age and (age < 30 years and age ≥ 30 years) were comparable in CP and controls (Table 3). A bad obstetric history, maternal disease, pre-eclampsia and placenta previa did not differ significantly between CP and controls. Spontaneous abortion, abruptio placenta, prelabour rupture of membranes and prematurity were associated with a higher risk of CP.

Intrapartum factors

Oxytocin therapy and fetal distress did not differ significantly between the CP and controls. (Table 4). Preterm labour, cesarean section and low birth weight (<2500 g) were associated with an increased risk of CP. Almost 74% of children with CP and 55% of children in controls were delivered by cesarean section due to acute fetal distress (data are not shown).

Neonatal factors

An Apgar score, ≤ 4 at 1st minute, was significantly associated with an increased risk of CP (odds ratio 6.60 (3.56-12.21) (Table 5). The respiratory distress syndrome, prolonged ventilation, septicemia, meningitis, hyperbilirubinemia, neonatal seizures, and severe cranial ultrasound abnormality were associated with CP. Neonatal seizures occurred in 21 (10.3%) babies and were associated with a significantly increased risk of CP. Cerebral ultrasound scans were available for a total of 163 (79.9%) babies, with a similar proportion for cases and controls.

Antenatal, intrapartum, and neonatal factors in the logistic regression

These factors were analysed separately in three models of the logistic regression. The first model included antenatal factors (spontaneous abortion, abruptio placenta, prelabour rupture of membranes, and prematurity) and the second intrapartum factors (preterm labour, cesarean section, birth weight < 2500 g). The last model included neonatal factors (Apgar score at 1st minute ≤ 4, respiratory distress syndrome, prolonged ventilation,

Table 4. Selected intrapartum factors in CP subjects and controls

Factors	CP subjects n (%)	Controls n (%)	OR (95% CI)	P value
Oxytocin therapy	42 (20.5)	36 (14.7)	1.18 (0.71-1.97)	0.590
Fetal distress	69 (33.8)	56 (27.1)	1.24 (0.90-1.33)	0.336
Preterm labour	83 (40.6)	15 (7.28)	5.58 (3.01-10.46)	<0.001
Cesarean section	65 (31.8)	41 (19.9)	1.60 (1.01-2.53)	<0.05
Birth weight, < 2500 g	66 (32.3)	11 (5.39)	6.06 (3.00-12.51)	<0.001

Data are expressed as n (%); OR: odds ratio; CI: 95% confidence interval.

Table 5. Selected neonatal factors in CP subjects and controls

Factors	CP subjects n (%)	Controls n (%)	OR (95% CI)	P value
Apgar score at 1st minutes ≤ 4	85 (41.6)	13 (6.31)	6.60 (3.56-12.21)	<0.001
Respiratory distress syndrome	48 (23.5)	14 (6.80)	3.46 (1.78- 6.80)	<0.001
Prolonged ventilation	52 (25.4)	15 (7.28)	3.50 (1.85-6.27)	<0.001
Septicemia	16 (7.84)	5 (2.42)	3.23 (1.08-10.27)	<0.05
Meningitis	18 (8.82)	4 (1.94)	4.54 (1.41-16.13)	<0.01
Hyperbilirubinemia	46 (21.1)	17 (8.25)	2.73 (1.46-5.14)	0.001
Neonatal seizures	21 (10.3)	5 (2.42)	4.24 (1.47-13.08)	0.005
Severe cranial ultrasound abnormality * (data were received from 163 CP and 165 controls)	68 (41.7)	6 (3.63)	11.44 (4.66-29.93)	<0.001

* Grade 3 and 4 bleeding, periventricular leukomalacia, and ventricular dilatation.

Data are expressed as n (%); OR: odds ratio; CI: 95% confidence interval.

septicemia, meningitis, hyperbilirubinemia, neonatal seizures, severe cranial ultrasound abnormality.

In the logistic regression models only prematurity (B0-regression coefficient=4.04 $\pm$ 0.77, P <0.001) and Apgar scores of ≤ 4 at 1st min (B0-regression coefficient=4.39 $\pm$ 1.65, P=0.008) were significantly related to an increased risk of CP.

Discussion

Several antenatal, intrapartum, and neonatal factors investigated in this study of term and preterm and very preterm babies were associated with an increased risk of CP. The weaknesses of our study include its retrospective nature. The problems encountered were some missing information in the records (ultrasound examinations), limited number of histopathological examination of the placentas (chorioamnionitis) so these variables were not analyzed. The strong points of the present study are that all children with CP were from the same a geographically area. Eight-five percent of children were at least 3 years old at diagnosis.

Almost 40% of CP children had preterm labour. In our study, spastic hemiplegia, spastic diplegia and tetraplegia occurred significantly more frequently in the term group than in the preterm and very preterm groups. Our findings are consistent with previous

studies (5,7).

The survival of preterm children has improved during the last decade but there has been no change in the prevalence if neurologic impairment, including CP. Preterm birth is the most important risk factor for CP. The risk of CP is inversely proportional to gestational age and the relative risk is 60 times higher at <28 week of gestation than at term (8). These findings are consistent with our results. Wilson–Costello et al. (5) compared between 72 singleton inborn very low birth weight (VLBW <1500 g) who had neurologic impairment at 20 months (including 50 with CP and 22 with other neurologic impairments) and 72 neurologically normal VLBW. In the subgroup with CP significant differences included days on the ventilator, septicemia, and severe cranial ultrasound abnormality. Multivariate analysis revealed direct and independent effects of severe cranial ultrasound only. Grether et al. (10) in a recent study of prenatal and perinatal factors and CP in VLBW infants, considered the interaction between prenatal and neonatal events and found chorioamnionitis to be associated with CP only when seizures occurred in the neonatal period.

Maternal age (age < 30 years and age ≥ 30 years) were comparable in CP and controls. In previous studies maternal age, bad obstetric history, maternal disease, pre-eclampsia, and placenta previa have not

been associated with CP (2-5). The present results confirm these findings. In contrast, hypertensive disease and pre-eclampsia have been found to be associated with a reduced risk of CP (5,8).

Our analysis suggests that there is an increased occurrence of CP in infants whose histories reported maternal spontaneous abortion, abruptio placenta, prelabour rupture of membranes and prematurity. This is in agreement with the previous reports (2,8).

In earlier reports oxytocin therapy and fetal distress have not been related with CP (5,8). The data from this study support these observations. In contrast Richmond et al. (9) identified fetal distress more frequently in children with CP who were born at term (24%) than among controls (11%). They suggested that its complete elimination might be expected to reduce the birth prevalence of CP by 15.6%. Often the subtle signs of fetal distress are missed; these are a change in the grade of meconium in the amniotic fluid, a rising base-line fetal heart rate, the absence of accelerations, the presence of 'atypical' variable decelerations or a combination of the above.

In this study, cesarean section and low birth weight (<2500 g) were associated with an increased risk of CP. These results are not consistent with the previous studies (5,8). They found no association between cesarean section and CP (5,8). On the other hand, there is no evidence that cesarean section can prevent CP in term infants (4). We think that the urgent indications (eg. acute fetal distress) for cesarean sections are more related with CP than the sections. An association between low birth weight and CP has also been reported by others (1,8,10).

Apgar scoring is a quick and subjective method of assessing the condition of newborn infants (11). Low Apgar scores do not indicate the cause of the poor condition, which may result from many different factors of which acute intrapartum hypoxia is only one. In preterm infants the Apgar score is highly limited in this respect. Now, the duration of low Apgar scores is more likely to indicate the low effectiveness of resuscitation than to predict the outcome. In our study we evaluated Apgar score at 1st minute. We found a strong association between ≤ 4 Apgar score at 1st minute and CP. Our data are in an agreement with previous reports (1,5,11-13).

In earlier reports seizures, ventricular dilatation have been associated with periventricular leukomalacia and CP (9,14). Prolonged ventilation, and respiratory distress have been associated with both periventricular leukomalacia and CP (9,15). The findings in this study are consistent with these observations, supporting the hypothesis that respiratory disturbances have a role in the etiology of cerebral ischemia in CP.

Several studies have shown associations

between neonatal septicemia, meningitis and both periventricular leukomalacia and CP (14,16,17). Recent studies suggest that fetoplacental uterine infection is an important in the initiation of preterm labour and for the development of central nervous system injury and CP (5,11). Inflammatory cytokines released during the course of intrauterine infections have been implicated in the genesis of brain white matter lesions and subsequent CP (18). Yoon et al. (19) studied amniotic fluid concentrations of the tumor necrosis factor-alpha, interleukin-1 beta, interleukin-6, and the natural interleukin-1 receptor antagonist in women with complicated preterm gestations. Periventricular white matter lesions of the neonate were diagnosed by neurosonography. Acute histologic chorioamnionitis was more common in the placentas of neonate with white matter lesions than in those without these lesions. Neonates with white matter lesions were delivered at a lower mean gestational age and birth weight and had a higher rate of significant complications (including respiratory distress syndrome, intraventricular hemorrhage, and infection-related complications) than did those without white matter lesions. Yoon et al. (19) also found that antenatal exposure to intra-amniotic inflammation and evidence of a systemic fetal inflammatory response (funisitis) were strong and independent risk factors for the subsequent development of CP at the age of three years. Our data support this hypothesis that neonatal sepsis and meningitis were associated with CP.

Earlier studies concerning antenatal and intrapartum risk factors for CP in very preterm babies revealed a strong association between maternal infection and, in particular, chorioamnionitis and an increased risk of CP (1,13,20). Maternal infection followed by neonatal sepsis was strongly associated with CP in preterm babies. However, in our study we found a small proportion of the study population.

The present study suggests a role for several factors in the etiology of CP in term and preterm babies. The difficulty in interpreting these findings, however, lies in determining which neonatal factors are causes of CP and which are consequences of earlier disturbances in the antenatal and intrapartum periods and already part of the outcome (19).

Previous report on periventricular leukomalacia and CP showed an association between ultrasonically diagnosed parenchymal damage and ventricular dilatation and CP (21,22). It is possible, that cerebral ultrasound lesions arise incidentally as a result of severe physiological disturbances which in themselves cause CP. This is unlikely, though, because ultrasound findings are so much more predictive of CP than are cardiorespiratory complications.

Cerebral palsy has been often defined as an

umbrella term covering a group of non-progressive, but in spite of motor impairment, behavioural, sensory, cognitive disabilities and retardation and epilepsy are reported (13,14,23-25). It makes substantial demands on medical, educational, therapeutic, and social services. Knowledge of important risk factors for CP is essential for the better care of mother and child during pregnancy, labour and neonatal period. It may reduce the occurrence of CP in future.

In conclusion we suggest that CP has multiple risk factors, both causes and modifiers, but that a proportion of cases of CP among preterm and term singletons have their origins in the antenatal, perinatal and neonatal period. Our findings are in agreement with the reports from Western countries.

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