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Children with dyskinetic cerebral palsy are severely affected as compared to bilateral spastic cerebral palsy

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Short title: Disease severity in dyskinetic cerebral palsy

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ABSTRACT

Aim

We aimed at describing clinical findings in children with dyskinetic as compared to bilateral spastic cerebral palsy (CP).

Methods

Data was extracted from the Danish nationwide CP register. Participants were born in 1999-2007 and were 5-6 years at ascertainment.

Results

The total number of CP cases was 1,165 of which 92 had dyskinetic and 540 bilateral spastic CP. Prevalence of dyskinetic CP was 0.16 per 1000 live-births. In participants with dyskinetic compared to bilateral spastic CP, there was more frequently an Apgar level less than five at five minutes (22.7% versus 11.2%) and neonatal seizures (43.5% versus 28.5%), but less respiratory deficiency, hyperbilirubinaemia, and sepsis. Impairment based on gross motor function classification was more severe in dyskinetic CP (level III-V 90.0% versus 66.0%). In dyskinetic CP there was a high rate of reduced developmental quotient (68.1%), visual impairment (39.3%), and epilepsy (51.6%). Basal ganglia lesions were more prevalent in dyskinetic compared to bilateral spastic CP (27.7% vs. 12.8%).

Conclusion

Cases of dyskinetic CP had overlapping clinical features with cases of bilateral spastic CP, but differed significantly in several perinatal risk factors. The children with dyskinetic CP had experienced more peri- or neonatal adverse events, and neurodevelopmental impairment was severe.

Key words: cerebral palsy, dyskinetic, paediatric, prevalence, spasticity

Key notes:

- We aimed at describing clinical findings in children with dyskinetic cerebral palsy as perinatal care may influence prevalence and severity

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- The prevalence of dyskinetic cerebral palsy was 0.16 per 1000 live-births and was most prevalent in term-born, appropriate for gestational aged children with a low Apgar score

- Compared to bilateral spastic cerebral palsy motor impairment was more pronounced in dyskinetic cerebral palsy and neurodevelopmental outcome was severe

INTRODUCTION

Cerebral palsy (CP) is one of the most common disabilities in childhood, posing significant demands on health, educational, and social services (1). The overall prevalence of CP is estimated to be 1.8-3 per 1000 live births (2,3,4). CP is defined as a group of permanent but not unchanging disorders of movement and/or posture and motor function, which are due to a non-progressive interference, lesion, or abnormality of the developing/immature brain (1). The Surveillance of Cerebral Palsy in Europe has classified CP in three main groups according to their neurological signs: spastic, dyskinetic and ataxic. Dyskinetic CP has previously been subdivided into dystonic and choreoathetotic subtypes (5), but in the recent two decades many have adapted the Surveillance of Cerebral Palsy in Europe definition of dyskinetic CP as “involuntary, uncontrolled, recurring, occasionally stereotyped movements. Primitive reflexes pattern predominate, muscle tone varies” (1).

Dyskinetic CP is the second most common subtype, affecting 10-20% of children with CP (2,4,6). Spasticity and dyskinesia may coexist, and in these cases the type is classified according to which is the most prominent. Some studies indicate that severe motor impairment (7) and intellectual disability is more frequent in dyskinetic CP than in other subtypes (8,9). Dyskinetic CP may have a wide array of underlying aetiologies, but is classically seen in term born children with severe peri-or neonatal adverse events, leading to a distinct pattern of brain injury (10).

Dyskinetic CP is of interest as there are more modifiable factors involved related to neonatal care and other treatment options as compared to the more prevalent spastic CP. Treatment options differ, and neuromodulatory interventions, such as intrathecal baclofen and deep brain stimulation are promising treatment options (5). A frequent neuroimaging finding in dyskinetic CP is basal ganglia and thalamus lesions combined with cortical injury but recent studies have also shown other imaging findings, such as white matter damage (11,12).

In this population-based study we have analysed the prevalence of dyskinetic CP and the perinatal and neonatal factors, accompanying impairments and neuroimaging findings. Comparison with
children with bilateral spastic CP was performed aiming at identifying early distinctive markers and differences in outcome.

**MATERIALS AND METHODS**

**Participants**

Participants were children born in 1999-2007 with dyskinetic or bilateral spastic CP identified from the Danish national CP register, which is one of the largest single-nation CP registers and has existed since 1967 (13). National coverage is assumed to be complete as information on CP is based on reporting from all Danish paediatric departments as well as the Danish National Patient Registry, in which registration is mandatory. The diagnosis was confirmed and data were extracted by chart review by four trained neuropaediatricians before inclusion. Inclusion criteria for the CP register are:

a) Prenatal or perinatal aetiology before 28th day of life, b) Fulfilment of diagnostic criteria according to Surveillance of Cerebral Palsy in Europe (1), c) Born in Denmark and alive at age four to five years. In cases of death before one year of age, cases were only included if the CP-diagnosis was unquestionable. The diagnosis of CP according to the European criteria was fulfilled in only approximately 50% of cases from the Danish National Patient Registry, therefore only 50% of patients with a CP diagnosis in the Danish National Patient Registry was included in the CP register (P Uldall, personal communication). Population data were collected from case notes as well as the Danish Medical Birth Registry (14).

**Assessments**

The age range at ascertainment was 5-6 years. Motor function was based on the gross motor function classification system (GMFCS) level at the time of data extraction (15). Small and large for gestational age (GA) was defined as a birth weight for GA below or above two standard deviations (SD), respectively. The calculation was based on Marsal’s weight curves. DQ (development quotient) was an estimated IQ based on assessment from the medical records at age 5-6 years and divided into 1) normal for children without learning disabilities and expected to start normal school (IQ above 85), 2) abnormal for children with moderate learning disabilities and in need of special education/attendance in special schools and 3) children with a severe intellectual disability (IQ below 50). The description of vision was based on medical charts and in some cases ophthalmological examination. Vision was defined as severely impaired if vision acuity on the best eye was below or at 6/18 after correction with glasses, or a severe visual field reduction. Epilepsy
was defined as more than one non-febrile seizure after the neonatal period. Data on patients with dyskinetic CP was compared to bilateral spastic CP data based on our previous publication (4).

**Neuroimaging**

Magnetic resonance imaging (MRI) was achieved in the majority of dyskinetic and bilateral spastic CP cases. In most of the remaining cases ultrasound or computed tomography (CT) was performed. The choice of modality was based on judgment by the clinician. Information on neuroimaging results was obtained from reports from the local imaging departments.

**Statistical analysis**

Proportions were compared by chi-square test or Fisher’s exact test, when there were less than five cases in a group. Missing data were not included in the analyses.

**RESULTS**

During the period 1999-2007, there were 585,393 live births registered via the Danish Medical Birth Registry. The total number of cases from the Danish national CP register in the period was 1,165. Dyskinetic CP was seen in 92 patients, which was 8.3% of all CP cases. There were 57.6% who were males. The prevalence of dyskinetic CP was 0.16 per 1000 live-born children and the number of children with dyskinetic CP remained stable in the time period (Table 1). Bilateral spastic CP was seen in 540 patients.

**Characteristics of mothers, pregnancy and the neonatal period of cases of dyskinetic CP**

A maternal disorder during pregnancy was present in 15 cases, of whom three had a urinary tract infection and two had an infection with toxoplasmosis, rubella, cytomegalovirus, or herpes virus. Fever during labour was present in 51 mothers and seven had preeclampsia. Delivery was by caesarean section in 41. The majority were born at term (n=69; data missing in 1). None were born post term.

Birth weight was normal in 64 children, below 2500g in 21 and below 1500g in six. Data was missing for birth weight in one case. Ten cases were from twin pregnancies (11.0%). Apgar score at five minutes was below five in one patient (4.5%) born preterm and in 18 patients (27.3%) born at term.

In the neonatal period there was a need for continuous positive airway pressure (CPAP) in 15 children born preterm and in 25 children born at term. Ventilator treatment was needed for three children born preterm were treated with ventilator and eight born at term. There were two children born preterm and eight born at term that needed surfactant treatment and five needed transfusion.
for anaemia. Neonatal seizures occurred in four children born preterm and 35 children born at term. There were nine children born preterm and 42 children born at term who had encephalopathy. None had a verified cerebral infection. A syndrome diagnosis was made in 13 children by the treating paediatrician and the coding neuropaediatrician agreed with the diagnosis. We examined other parameters including presence of multiple pregnancy, caesarean section, Apgar score, hyperbilirubinaemia, sepsis, cardiac disease and necrotizing enterocolitis, treated either with medication or surgery compared to bilateral spastic CP cases (Table 2).

**Impairments and epilepsy in cases of dyskinetic CP**

Distribution of GMFCS levels is shown in Figure 1A. The GMFCS distribution in dyskinetic vs. bilateral spastic was: level I 6.7% versus 21.1%, level II 3.3% versus 12.9%, level III 5.6% versus 8.4%, level IV 22.2% versus 25.8% and level V 62.2% versus 31.8%, implicating that the severity of motor impairment was more pronounced in dyskinetic CP compared to bilateral spastic CP (p<0.01).

Reduced DQ was present in 81 of the 92 children with dyskinetic CP. The DQ was below 50 in 68.1% and between 50 and 85 in 20.9%. Only 10 children had a normal DQ (missing data in 1). The number of cases with a reduced DQ increased with the severity of the motor disability, but it is noteworthy that six of the 76 children with GMFCS IV to V had a normal DQ (7.9%). There were 33 children who had severe visual impairment (39.3%). There were 28 out of the 33 children with a severe visual impairment who were found among those classified in GMFCS level V. Data on vision was missing for eight children. Number of children with epilepsy at some point after the neonatal period was 65.9%, and children who at age 5-6 years still were treated medically for epilepsy was 51.6%.

**Neuroimaging**

MRI was performed before age 5-6 years in 83 of 92 cases of dyskinetic CP and in 383 of 540 cases of bilateral spastic CP. Ultrasound or CT was performed in the majority of the remaining cases. The most frequent abnormalities in dyskinetic CP were: periventricular leukomalacia (n=29), basal ganglia/thalamus lesions (n=23), cortical/subcortical lesions (n=22), periventricular substance loss (n=16), bilateral cerebral malformations or agenesis of the corpus callosum (n=15) and cerebellar malformation or atrophy (n=12). Other MRI findings were described in 24 children. In the nine children where MRI was not performed, there were six who had an ultrasound performed, of whom four were described as normal, one was described with ventriculomegaly, porencephaly and bleeding and one with infarction. One child had a CT scan performed showing atrophy. MRI was normal in 15 and comparison of MRI findings with bilateral spastic CP is depicted in Table 3.
DISCUSSION

The prevalence of dyskinetic CP was 0.16 per 1000 live-births. The 92 children in the present study constituted 8.3% of all children with CP born in Denmark between 1999 and 2007, which is higher than what the majority of others have reported (16,17). A possible explanation to this could be that in other countries the principle of diagnosing CP by the dominant syndrome was applied later (16,18).

The majority of dyskinetic CP cases were born at term (75.6%) and had an appropriate weight for GA (81.3%). Compared to children with bilateral spastic CP there were more cases with Apgar score less than five at five minutes (21.7% versus 11.2%), neonatal seizures (43.5% versus 28.5%), and neonatal cerebral encephalopathy (56.5% versus 42.6%), but less cases with respiratory deficiency, anaemia, hyperbilirubinaemia and sepsis.

Over time the clinical picture of patients with dyskinetic CP has changed, including factors related to known aetiologies and GA. In the study by Kyllerman et al (19), hyperbilirubinaemia was present as a major contributory factor, as they described hyperbilirubinaemia in 32% of cases in 1959-70. With the development of safe routines for testing and the use of phototherapy, hyperbilirubinaemia and kernicterus has decreased markedly as a risk factor for dyskinetic CP. The proportion of children treated for hyperbilirubinaemia in our study was even lower in dyskinetic CP than in other CP types. The proportion of children with dyskinetic CP born preterm has fallen from 35% in Kyllerman et al’s study to 24.2% in our study.

Hypoxic-ischemic encephalopathy in infants born near term is another risk factor for dyskinetic CP. Hypoxic-ischaemic encephalopathy has not decreased in the same way as hyperbilirubinaemia in the last decades, but cooling may impact neurotoxicity in hypoxic-ischaemic encephalopathy and thereby prevent cerebral damage. Cooling therapy was only partly available as part of a research project in Denmark before 2008 and therefore has very limited impact in the studied cohort. Apgar score and the need for neonatal resuscitation was correlated to subsequent risk of CP (20,21). In our cohort 22.7% of dyskinetic CP infants had an Apgar score below five at five minutes as compared to less than 0.5% in the general population (22). This is a considerable difference and higher than what is seen in bilateral spastic CP infants (11.2%). Bilateral spastic CP cases seemed to often present with respiratory problems whereas the dyskinetic CP cases more often presented with neurological problems, such as seizures and encephalopathy. Badawi et al. have reported that 13% of survivors from neonatal encephalopathy had CP (23).
The majority of children in our cohort of dyskinetic CP had a weight at birth appropriate for GA (81.3%). The proportion of small for GA (14.3%) was similar to the series from 1982 (20). On the contrary, Himmelmann et al. (17) found only 2% of the children with dyskinetic CP being small for GA, which was similar to the background population. A correlation between intrauterine growth, either small or large for GA, and CP has been noticed previously, independent of CP type (24). Furthermore, low birth weight for GA was reported to be associated with birth asphyxia in children with dyskinetic CP (19). Our results support the hypothesis that low birth weight for GA may be a risk factor for developing dyskinetic CP.

In the current cohort of children with dyskinetic CP the level of impairment based on GMFCS was mild in nine cases (GMFCS I-II) and severe in 81 (GMFCS level III-V). An equivalent distribution pattern was seen in the study by Sun et al (26), where among 26 cases of dyskinetic CP none had GMFCS levels I-II. The rate of reduced developmental quotient (68.1%), visual impairment (39.3%) and epilepsy after the neonatal period (65.9%) increased with the severity of the motor disability.

We found a high frequency of current epilepsy at age 5-6 years in our cohort (51.6%). Previous reports have been somewhat lower. Carlsson et al have described a cohort with a range of CP types and GMFCS levels where 34 out of 83 had active epilepsy (26). In a survey-based study 43% of children aged six to 14 years with dyskinetic CP designated that they had epilepsy (26). Another approach was used by Reid et al who in a cross-sectional study reported epilepsy in 41.8% across all CP types (9). The different results may be due to methodological variation in definition of epilepsy and possible selection bias.

Neuroimaging with MRI was performed in 74 of the children in our cohort and was normal in 15. Basal ganglia/thalamus lesions were more prevalent in children with dyskinetic CP as compared to spastic bilateral CP, but only occurred in 27.7% of dyskinetic cases. In the remaining cases, a great variety of lesions were found, of which some findings may be related to CP, but not causative.

Several studies have found that insults at different periods of gestation are associated with certain imaging findings and that the lesions match the clinical pictures (28,29). In this study, only a third of the children (32.3%) in the term born group had lesions in the basal ganglia/thalamus in contrast to the majority (77%) in the study of Himmelmann et al. (17). The difference cannot be explained by missing MRI scans in our study, as MRI was available in 89% of cases. In some cases the MRI reports with widespread brain lesions may not specifically have mentioned basal ganglia lesions and therefore may have been missed in our study. In another cohort of 56 patients with dyskinetic CP many different cerebral lesions were likewise seen, and the authors report a normal MRI in 9.8% with the most frequent injuries seen in white matter (26.8%) and grey matter (31.7%) (9). In another
cohort of 39 patients with dyskinetic CP where images were re-evaluated systematically with focus on basal ganglia, brain lesions were most frequently located in the ventral posterior lateral thalamus and frontal lobe (30). It is likely that the use of a standardized imaging protocol and central review of images would have affected the imaging results.

The current study had the strength of being based on the well-established national CP register, where the diagnoses were reliable and extensive data was available. The limitations of the study include that classification of CP has changed slightly over time. Furthermore the predominant expressed type of CP may change over the lifetime of the patient and at some point the patient may encompass more spastic than dyskinetic features or vice versa.

CONCLUSION

Dyskinetic CP was most prevalent in term-born, appropriate for gestational aged children with low Apgar score. Dyskinetic CP had overlapping clinical features as well as MRI presentations with cases of bilateral spastic CP, but differed significantly in several perinatal features and had severe neurodevelopmental impairment. The children had frequently experienced peri- or neonatal neurological adverse events, however, kernicterus was no longer a major factor in the pathogenesis of dyskinetic CP. The strong association between low Apgar and dyskinetic CP could indicate that the injury in many cases occurred intrapartum, and a focus on recent changes in obstetric and early neonatal care including cooling of asphyxiated children to prevent novel cases of dyskinetic CP is warranted.

Conflicts of interest

The authors have no conflicts of interest to declare.

Finance

This study did not receive any specific funding.
**Abbreviations:** CP = cerebral palsy; CPAP = continuous positive airway pressure; CT = computed tomography; DQ = developmental quotient; GA = gestational age; GMFCS = gross motor function classification system; MRI = magnetic resonance imaging.

**References**


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**Tables and figures**

**Table 1.** Distribution of dyskinetic CP cases according to birth year and gestational age (GA) in 1999-2007. Gestational age was unknown in 39 cases and CP type was unknown in 16 cases of total CP.

<table>
<thead>
<tr>
<th>Birth year</th>
<th>Preterm (GA &lt;37 weeks)</th>
<th>Term (GA ≥ 37 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total CP</td>
<td>Dyskinetic CP</td>
</tr>
<tr>
<td>1999-2001</td>
<td>152</td>
<td>5 (3.3%)</td>
</tr>
<tr>
<td>2002-2004</td>
<td>142</td>
<td>9 (6.3%)</td>
</tr>
<tr>
<td>2005-2007</td>
<td>144</td>
<td>8 (5.6%)</td>
</tr>
<tr>
<td>Total</td>
<td>438</td>
<td>22 (5.0%)</td>
</tr>
</tbody>
</table>
Table 2. Comparison of peri- and neonatal features in 92 children with dyskinetic CP and 540 children with bilateral spastic CP. Abbreviations: Apgar=appearance, pulse, grimace, activity, respiration; CPAP=continuous positive airway pressure; CP=cerebral palsy; SD=standard deviation.

<table>
<thead>
<tr>
<th></th>
<th>Dyskinetic CP n (%)</th>
<th>Bilateral spastic CP n (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Twin or multiple pregnancy</td>
<td>10 (11.0%)</td>
<td>83 (15.6%)</td>
<td>0.251</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>41 (45.0%)</td>
<td>232 (44.8%)</td>
<td>0.962</td>
</tr>
<tr>
<td>Apgar at 5 minutes below 5</td>
<td>20 (22.7%)</td>
<td>58 (11.2%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>CPAP treatment</td>
<td>40 (47.1%)</td>
<td>315 (61.1%)</td>
<td>0.02</td>
</tr>
<tr>
<td>Ventilator</td>
<td>11 (12.4%)</td>
<td>101 (19.4%)</td>
<td>0.11</td>
</tr>
<tr>
<td>Surfactant</td>
<td>2 (2.2%)</td>
<td>77 (14.9%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Anaemia treated with transfusion</td>
<td>5 (5.5%)</td>
<td>56 (10.9%)</td>
<td>0.11</td>
</tr>
<tr>
<td>Hyperbilirubinaemia, treated</td>
<td>14 (15.6%)</td>
<td>190 (37.5%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Sepsis</td>
<td>28 (30.4%)</td>
<td>221 (42.9%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Neonatal seizures</td>
<td>40 (44.0%)</td>
<td>148 (28.5%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Cardiac disease in neonatal period</td>
<td>6 (6.5%)</td>
<td>30 (5.7%)</td>
<td>0.75</td>
</tr>
<tr>
<td>Necrotizing enterocolitis, treated</td>
<td>2 (2.2%)</td>
<td>25 (4.8%)</td>
<td>0.27</td>
</tr>
<tr>
<td>Cerebral infection in neonatal period</td>
<td>0</td>
<td>13 (2.5%)</td>
<td>0.13</td>
</tr>
<tr>
<td>Neonatal cerebral encephalopathy</td>
<td>52 (56.5%)</td>
<td>223 (42.6%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Small for gestational age (weight &lt;-2SD)</td>
<td>13 (14.3%)</td>
<td>65 (12.6%)</td>
<td>0.65</td>
</tr>
<tr>
<td>Large for gestational age (weight &gt;+2SD)</td>
<td>4 (4.4%)</td>
<td>22 (4.3%)</td>
<td>0.95</td>
</tr>
<tr>
<td>Appropriate for gestational age</td>
<td>74 (81.3%)</td>
<td>429 (83.1%)</td>
<td>0.67</td>
</tr>
</tbody>
</table>
Table 3. Findings of cerebral magnetic resonance imaging (MRI) in 83 children with dyskinetic CP and in 439 children with bilateral spastic CP.

<table>
<thead>
<tr>
<th>MRI findings</th>
<th>Dyskinetic CP n (%)</th>
<th>Bilateral spastic CP n (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral cerebral malformation (disturbed proliferation, migration or organization)</td>
<td>2 (2.5%)</td>
<td>7 (1.6%)</td>
<td>0.64</td>
</tr>
<tr>
<td>Bilateral cerebral malformations or agenesis of the corpus callosum</td>
<td>15 (18.2%)</td>
<td>56 (12.6%)</td>
<td>0.15</td>
</tr>
<tr>
<td>Unilateral periventricular leukomalacia</td>
<td>4 (4.8%)</td>
<td>17 (3.9%)</td>
<td>0.55</td>
</tr>
<tr>
<td>Bilateral periventricular leukomalacia</td>
<td>25 (30.1%)</td>
<td>172 (39.2%)</td>
<td>0.14</td>
</tr>
<tr>
<td>Periventricular substance loss (local or general enlargement of the ventricular system)</td>
<td>16 (19.3%)</td>
<td>83 (18.9%)</td>
<td>0.83</td>
</tr>
<tr>
<td>Cortical/subcortical lesions, i.e. infarction, porencephaly or atrophy</td>
<td>22 (26.5%)</td>
<td>105 (23.9%)</td>
<td>0.54</td>
</tr>
<tr>
<td>Basal ganglia/ thalamus injury</td>
<td>23 (28.4%)</td>
<td>56 (12.9%)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Cerebellar malformation or atrophy</td>
<td>12 (14.5%)</td>
<td>35 (8.0%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Other malformations, i.e. AV malformations, aneurysms, haemangiomas, atrophy</td>
<td>24 (28.9%)</td>
<td>99 (22.6%)</td>
<td>0.20</td>
</tr>
<tr>
<td>MRI normal</td>
<td>15 (18.1%)</td>
<td>112 (25.5%)</td>
<td>0.15</td>
</tr>
<tr>
<td>MRI not performed</td>
<td>9 (10.8%)</td>
<td>101 (23.0%)</td>
<td>0.04</td>
</tr>
</tbody>
</table>
Figure 1. A. Distribution of GMFCS levels in children with dyskinetic CP and bilateral spastic CP born 1999-2007. X-axis: GMFCS type, Y-axis: axis number of children. B. Focus on neurodevelopmental outcome in children with dyskinetic CP: Developmental quotient, presence of severe visual impairment and epilepsy according to GMFCS score. Abbreviations: GMFCS=gross motor function classification system; DQ=developmental quotient.

A.

B.