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ORIGINAL ARTICLE

High occurrence of perinatal risk factors and more severe impairments in children with postneonatal cerebral palsy in Norway

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Abstract

Aim: To describe causal events, perinatal risk factors and clinical characteristics in children with postneonatal cerebral palsy (PNCP).

Methods: Population-based registry study of Norwegian children born 1999–2013. Prevalence, causal events and clinical characteristics of PNCP were described. The occurrence of perinatal risk factors for CP was compared with the general population.

Results: Among 1710 children with CP, 67 had PNCP (3.9%; 0.75 per 10,000 live-births [95%CI: 0.59–0.96]). The prevalence of PNCP decreased during the study period. Leading causal events were cerebrovascular events (32.8%), head injuries/other accidents (22.4%), infections (19.4%) and hypoxic events (14.9%). Spastic hemiplegic (53.7%) or spastic quadriplegic/dyskinetic CP (31.3%) was more common in children with PNCP than non-PNCP (42.3% and 20.1%, respectively; $p < 0.001$). Children with PNCP had more severe motor and associated impairments. Perinatal risk factors for CP were more common in children with PNCP than in the general population.

Conclusion: The prevalence of PNCP among Norwegian children was low and decreasing. The main causes were cerebrovascular events and head injuries/other accidents. Although spastic hemiplegic CP was the dominating subtype, children with PNCP had more severe motor and associated impairments than children with non-PNCP, as well as a higher occurrence of perinatal risk factors than in the general population.

KEYWORDS
associated impairments, cerebral palsy, motor impairments, perinatal risk factors, postneonatal cerebral palsy

1 | INTRODUCTION

Cerebral palsy (CP) is a term covering a group of disorders affecting muscle tonus, movement and/or posture resulting from a non-progressive injury to the immature brain.[1] Based on whether the assumed brain injury occurred before or after the end of the neonatal period (i.e. 28 days after birth), CP may be divided into non-postneonatal cerebral palsy (non-PNCP) and postneonatal CP (PNCP).[2]

The prevalence of PNCP in high-income countries was reported to vary between 1.26 and 1.90 per 10,000 live births between 1970

Abbreviations: BWsds, birthweight standard deviation score; CI, confidence interval; CP, cerebral palsy; GMFCS, Gross Motor Function Classification System; IQ, intelligence quotient; MACS, Manual Ability Classification System; MBRN, Medical Birth Registry of Norway; NorCP, Norwegian Quality and Surveillance Registry for Cerebral Palsy; PNCP, postneonatal cerebral palsy; SCPE, Surveillance of Cerebral Palsy in Europe; SIDS, sudden infant death syndrome.

and 1999.[3–5] The low prevalence most likely explains why epidemiological studies on PNCP are sparse. These studies reported that the main causes of PNCP were infections, vascular episodes, head injuries, anoxic brain injuries and sequela of brain surgery. Additionally, the children with PNCP had more severe impairments than children with non-PNCP.[3–5] Two of the studies reported a decrease in the prevalence.[3,4]

Since the 1990s, several public health preventive measures have been introduced, such as vaccination against Haemophilus influenzae type b and Streptococcus pneumoniae, advice against infants sleeping in prone position, and better protection of infants and small children against traffic accidents. Thus, updated information on the causes of PNCP in children born in more recent birth cohorts is needed.

In addition, studying associations between the causes and CP subtypes in children with PNCP where the acute event is clearly identified may provide clues to the causes underlying the brain insults leading to non-PNCP. In the latter, the causes are often unknown or debated,[3,6–9] for example when considering if acute hypoxemia during delivery may have caused CP. An international consensus paper concluded that 'spastic quadriplegia and, less commonly, dyskinetic cerebral palsy are the only subtypes of cerebral palsy associated with acute hypoxic intrapartum events'.[10] This statement may be tested in PNCP.

Finally, although PNCP is considered to be caused by a clear event after the neonatal period, it may be speculated whether some of these children could have a minor pre-existing insult of the brain making them more vulnerable to an acute event than other children. Indeed, two previous studies found higher occurrences of some perinatal risk factors in children with PNCP than in the general population.[3,5]

The main aims of this study were to provide updated knowledge on the prevalence, causal events and clinical characteristics in children with PNCP and to study whether and how the clinical characteristics differ from children with non-PNCP. Secondary aims were to study the associations between causal events and clinical characteristics, and whether perinatal risk factors for non-PNCP were more prevalent among children with PNCP than in the general population.

Key Notes

- The prevalence of postneonatal cerebral palsy in Norway was low and decreasing.
- Cerebrovascular events were the leading causes of postneonatal cerebral palsy.
- Children with postneonatal cerebral palsy had more severe motor and associated impairments than children with non-postneonatal cerebral palsy.
- Perinatal risk factors were more common among children with postneonatal cerebral palsy than the general population.

2 | PATIENTS AND METHODS

2.1 | Study population

In this population-based registry study, children born in Norway between 1999 and 2013 were eligible. Data were abstracted from the Medical Birth Registry of Norway (MBRN) and the Norwegian Quality and Surveillance Registry for Cerebral Palsy (NorCP). The MBRN is a mandatory national health registry that has collected information on all births in Norway since 1967.[11,12] The NorCP is a national consent-based medical quality and follow-up registry. Thus, the NorCP comprises both epidemiological and detailed clinical information on children with CP born since 1996. Information is recorded at several points in time, when the diagnosis is first suspected, several times during follow-up, when the diagnosis has been confirmed at age five years, and between ages 15 and 17 years.[13] Information recorded at five years of age was used in this study. Validation studies have shown that approximately 93% of all children with CP in Norway have consented to be registered in NorCP.[13,14] Information in the MBRN and NorCP was linked using the 11-digit unique identifier assigned to each Norwegian resident. The NorCP is a member of the Surveillance of Cerebral Palsy In Europe (SCPE) network, which has previously published two articles on PNCP.[3,4] However, no data used in the present study were included in the two studies.

2.2 | Study design

Data on children with CP were collected from the NorCP 5 Year Registration Form. Cerebral palsy was defined and confirmed at age 5 according to SCPE guidelines.[2] If an acute event was assumed to be the cause of the brain injury between 28 days of life and 24 months of age, it was considered to be PNCP.[2] Conversely, if the brain injury was of unknown origin or assumed to have occurred before 28 days of life, CP was defined as non-PNCP. NorCP collects CP diagnosis codes using the International Statistical Classification of Diseases and Related Health Problems version 10 (ICD-10): G80.2 spastic hemiplegic, G80.1 spastic diplegic, G80.0 spastic quadriplegic, G80.3 dyskinetic, G80.4 ataxic and G80.8/G80.9 other/unspecified. These diagnosis codes, or CP subtypes, were also used in previous studies on PNCP, thereby allowing for comparisons. To test whether the statement "spastic quadriplegia and, less commonly, dyskinetic cerebral palsy are the only subtypes of cerebral palsy associated with acute hypoxic intrapartum events"[10] could be confirmed in cases of PNCP, we combined the spastic quadriplegic and dyskinetic CP subtypes into one group. We categorised causal events associated with PNCP into six main groups based on the previous study by Reid et al.[5]: (1) cerebrovascular events, (2) head injuries/other accidents, (3) infections, (4) hypoxic insults, (5) tumour associated

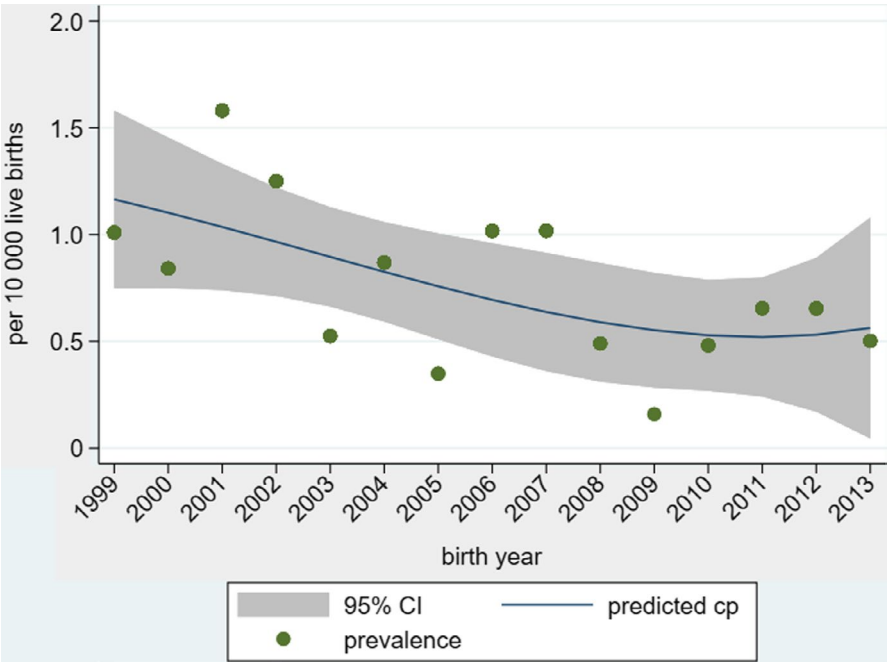

FIGURE 1 Prevalence of postneonatal cerebral palsy per 10,000 live births among children born in Norway from 1999 to 2013. Each point shows the actual prevalence. The solid line represents predicted cerebral palsy (cp) prevalence using logistic regression with fractional polynomials, and the shaded area denotes 95% CI

and (6) other (Table S1). The categorisation of each child was completed in consensus after extensive discussions between all authors. Gross motor impairment was classified according to the Gross Motor Function Classification System (GMFCS),[15] and fine motor impairment was classified according to the Manual Ability Classification System (MACS).[16] Epilepsy was defined as at least two unprovoked seizures after the neonatal period, gastrostomy as presence of gastrostomy for tube feeding. Cognition was classified as normal (IQ above 70), developmental disorder (ICD-10 codes F80–F81 and F83) and intellectual disability (IQ below 70; ICD-10 codes F70–F73 and F79). Speech impairment was classified according to the Viking Speech Scale.[17]

Information on pre-, peri- and neonatal risk factors, hereafter entitled perinatal risk factors, was collected from the MBRN. Preterm birth was defined as a birth before Week 37 of gestation. The 5-min Apgar scores were categorised as low (0–3), moderate (4–6) and normal (7–10).[18] Multiple births included at least two children born to the same mother at the same delivery. A congenital malformation was defined as a structural or functional anomaly that occurred during intrauterine life, recorded at birth. Birthweight standard deviation scores (BWsds) were calculated as the difference between the individual child's birthweight (BW_i) and the mean birthweight of the general population adjusted for sex and gestational age (BW_{mean}) and divided by BW_{mean}.

2.3 | Statistical analyses

Descriptive statistics using either Pearson's chi-square or Fisher's exact test for nominal variables and linear-by-linear association for ordinal variables was used to compare the differences in proportions between children with PNCP and non-PNCP. A *p*-value below 0.05 was considered statistically significant. The Wilson method as recommended by Fagerland et al.[19] was used to estimate prevalence with 95% confidence intervals (CI) of PNCP per 10,000 live births, as well as for the presence of perinatal risk factors in children with PNCP and in the general population. Imputation of missing data was not performed. To study trends in the prevalence of PNCP, we calculated prevalence in three 5-year intervals, as well as estimated changes in the prevalence of PNCP per 10,000 live births during the study period using logistic regression with birth year as covariate. Non-linear trends were accounted for using fractional polynomials with birth years as covariate. Statistical analyses were performed using SPSS version 26 (IBM Corp.) with the exception of logistic regression analyses using STATA version 16 (StataCorp. 2019. Stata Statistical Software: StataCorp LLC).

2.4 | Ethics

The NorCP is governed by the Ministry of Health and Care Services Regulations on Medical Quality Registries. This study was approved by the Regional Committee for Medical and Health Research Ethics in Mid-Norway (18129/2020).

3 | RESULTS

The study population is shown in Figure S1. A total of 889,904 live born children born between 1999 and 2013 were recorded in the MBRN. Among these, 1816 children with a diagnosis of CP were recorded in the NorCP. After exclusion of children who did not meet the PNCP criteria, and children where the 5-year Registration Form had not yet been received by the NorCP, 1710 were included in the study. Among these, 67 children (3.9%, CI: 3.1 to 4.9) had PNCP. The overall prevalence of PNCP was 0.75 per 10,000 live births (CI: 0.59

to 0.96), decreasing from 1.04 (CI: 0.73 to 1.48) among children born 1999–2003 to 0.49 (CI: 0.30 to 0.81) born 2009–2013. Assuming a linear model, the probability of a child born with PNCP was reduced by a factor of 0.937 per year, corresponding to a 6.3% yearly reduction ($p = 0.023$). Figure 1 shows the predicted PNCP prevalence with CIs during the study period.

The median age at PNCP insult was 9 months (interquartile range: 12 months), and 34 (50.7%) of the children with PNCP were boys, compared with 978 (59.5%) in the non-PNCP group.

3.1 | Cerebral palsy diagnoses

Among children with PNCP, a higher proportion were diagnosed with spastic hemiplegic or quadriplegic/dyskinetic CP, while the proportion with diplegic CP was lower compared to children with non-PNCP (Table 1).

3.2 | Motor function and associated impairments

Table 2 shows that children with PNCP had more severe gross and fine motor impairments than children with non-PNCP. Table 2 also shows that a higher proportion of children with PNCP were diagnosed with epilepsy, speech impairment, intellectual disability and more often had a gastrostomy tube compared to children with non-PNCP.

3.3 | Aetiology

The most common causal event of PNCP was attributed to cerebrovascular events, mainly leading to spastic hemiplegic CP (Table 3). This was followed by head injuries/other accidents and infections that mainly caused spastic hemiplegic and quadriplegic CP, as well as dyskinetic CP. Among ten children with hypoxic insults as the causal event, at least two children had either spastic hemiplegic, diplegic or quadriplegic CP, as well as dyskinetic CP.

Twelve (17.9%) children with PNCP had a congenital malformation, compared with 306 (18.6%) children with non-PNCP. However, nine children (13.4%) with PNCP had a cardiac malformation compared with 47 (2.9%) children with non-PNCP ($p < 0.001$). Among the nine children with PNCP and a cardiac malformation, seven had a cerebrovascular event, while two had a cardiac arrest as the assumed cause of CP.

Infection was the dominating cause in 10 of the 23 (34%) children with PNCP on GMFCS levels IV–V who also had severe fine motor impairment (21 (91%) MACS levels IV–V; two unknown) and multiple associated impairments (18 (78%) gastrostomy tube feeding, 19 (83%) Viking Speech Scale level IV and 16 (84%) epilepsy (four unknown)). Additionally, acute hypoxic events and head injuries/other accidents were identified as causes in five of the children.

3.4 | Perinatal risk factors for CP among children with PNCP

Compared with the general population, a higher proportion of children with PNCP were born preterm, had a lower Apgar score (4–6) at 5 min, were more often born as a multiple and more often had a congenital malformation (Table 4).

Forty-five children (67.2%) with PNCP had a BWsds below zero, and the mean BWsds was −0.43 (CI: −0.17 to −0.69) (Figure S2). The mean BWsds in the general population born during the same time period was marginally higher than zero: 0.005 (CI: 0.002 to 0.007).

4 | DISCUSSION

We found that less than one (0.7) in 10,000 children born in Norway between 1999 and 2013 had PNCP and that the prevalence decreased during these years. The main causes of PNCP were cerebrovascular events followed by head injuries/other accidents. Children with PNCP were more likely to be diagnosed with spastic hemiplegic or spastic quadriplegic/dyskinetic CP than children with non-PNCP. They also had more severe gross and fine motor impairments and were more likely to have one or more associated impairments. Infections were the dominating cause in more than every third child with PNCP who had multiple severe motor and associated impairments. A higher proportion of children with PNCP were born as a multiple, had some evidence of suboptimal growth in utero, a complicated delivery, or were diagnosed with a congenital anomaly, compared to the general population.

TABLE 1 Distribution of cerebral palsy (CP) subtypes among children with postneonatal (PNCP) and non-postneonatal CP (non-PNCP) born in Norway between 1999 and 2013

		PNCP		Non-PNCP		
ICD-10-code	CP subtype	n	(%)	n	(%)	*p*-value
G80.2	Spastic hemiplegic	36	(53.7)	695	(42.3)	
G80.1	Spastic diplegic	5	(7.5)	515	(31.3)	
G80.0	Spastic quadriplegic	15	(22.4)	223	(13.6)	
G80.3	Dyskinetic	6	(9.0)	108	(6.6)	
G80.4	Ataxic	3	(4.5)	69	(4.2)	
G80.8/G80.9	Other/unspecified	2	(3.0)	33	(2.0)	
	Total	67	100.0	1643	100.0	0.012

TABLE 2 Motor function and associated impairments in children with postneonatal cerebral palsy (PNCP) compared with children with non-postneonatal cerebral palsy (non-PNCP) born in Norway between 1999 and 2013

	PNCP		Non-PNCP		
	n	(%)	n	(%)	*p*-value
GMFCS[a]					0.037
Level I	33	(50.8)	833	(52.8)	
Level II	4	(6.2)	262	(16.6)	
Level III	5	(7.7)	112	(7.1)	
Level IV	5	(7.7)	148	(9.4)	
Level V	18	(27.7)	224	(14.2)	
MACS[b]					0.008
Level I	16	(27.1)	572	(39.4)	
Level II	16	(27.1)	435	(30.0)	
Level III	10	(16.9)	170	(11.7)	
Level IV	4	(6.8)	102	(7.0)	
Level V	13	(22.0)	172	(11.9)	
Epilepsy present[c]	34	(53.1)	461	(29.9)	<0.001
Gastrostomy present[d]	18	(28.6)	183	(12.4)	<0.001
Viking speech scale[e]					0.003
Level I	26	(42.6)	791	(52.4)	
Level II	5	(8.2)	257	(17.0)	
Level III	9	(14.8)	206	(13.6)	
Level IV	21	(34.4)	257	(17.0)	
Cognition[f]					0.015
Normal	22	(47.8)	771	(64.8)	
Developmental disorder	8	(17.4)	164	(13.8)	
Intellectual disability	16	(34.8)	255	(21.4)	

Abbreviations: GMFCS, Gross Motor Function Classification System; MACS, Manual Ability Classification System.

[a]Missing data in 2 (3.0%) children with PNCP and in 64 (3.9%) children with congenital CP.

[b]Missing data in 8 (11.9%) children with PNCP and in 192 (11.7%) children with congenital CP.

[c]Missing data in 3 (4.5%) children with PNCP and in 100 (6.1%) children with congenital CP.

[d]Missing data in 4 (6.0%) children with PNCP and in 171 (10.4%) children with congenital CP.

[e]Missing data in 6 (9.0%) children with PNCP and in 132 (8.0%) children with congenital CP.

[f]Missing data in 21 (31.3%) children with PNCP and in 453 (27.6%) children with congenital CP cases.

4.1 | Strength and limitations

A strength of this study is the prospective recording of data in both the MBRN and NorCP and that the CP diagnosis was confirmed at age five years, in accordance with SCPE guidelines.[2] Furthermore, validation studies have confirmed that more than 90% of children with CP during the study period were recorded in the NorCP.[13,14] In one of these studies, we found no difference in CP subtypes, severity of motor or associated impairments between children with CP included/not included in the NorCP.[14] When invited to participate, only 0.6% of parents declined to have information on their child registered. This was most likely due to work overload by collaborating professionals or parents of children who recently immigrated to Norway. Thus, it is unlikely that the differences between children with PNCP and non-PNCP are explained by selection bias due to the possibility that parents of children with PNCP are more reluctant to give consent than parents of children with non-PNCP.

Another strength is that the main results regarding group comparisons are unlikely to be due to chance as indicated by the low p-values ($p \leq 0.01$) and the non-overlapping confidence intervals. Yet, due to the relatively low number of children with PNCP, some of the results should be interpreted with caution. This applies in particular to the results GMFCS ($p = 0.037$) and cognition ($p = 0.015$). Another limitation is the missing data regarding cognition and MACS. Again, in previous studies, we reported that missing information in the NorCP was most likely due to work overload by the reporting clinicians, at random, and therefore, the results are most likely not explained by selection bias.[14,20] Typically, registers depend on information recorded by clinicians, and there are limits to how much detailed information can be requested. This is also a limitation of this study. Moreover, information collected on children with PNCP was limited to an ICD-10 code and the age when the event occurred, as assessed by a paediatrician. However, some misclassifications cannot be excluded, as it is possible that some children had a pre-existing brain injury not identified at birth (e.g. antenatally), when an acute event occurred thought to be the cause of CP. This potential misclassification would be expected to dilute the differences between children with PNCP and non-PNCP regarding subtypes, motor and associated impairments. However, the higher proportion of perinatal risk factors observed among children with PNCP compared to the general population should be interpreted with caution.

4.2 | Comparison with other studies

The proportion of children with PNCP in our study (3.9%) was lower than in previous studies.[3–5,21] Two studies in Europe by Cans et al. and Germany et al. reported that the proportion of children with PNCP was 7.7% and 5.5%, respectively,[3,4] while a study in Canada by Robertson et al. found that 5.2% of children with CP had PNCP.[21] A fourth study by Reid et al. in Australia reported that the percentage of children with PNCP among all children with CP was 10.7%.[5] The main difference between our study and the other studies is that we included a more recent birth cohort. Therefore, the lower proportion of children with PNCP in our more recent study on children in Norway may reflect the fact that PNCP was already reported to be decreasing in the four aforementioned studies, and the trend has continued to decrease in our study. This decrease may be explained by improvements in general paediatric medicine and public health

TABLE 3 Proportions of cerebral palsy (CP) subtypes according to categories of assumed causes among children with postneonatal CP born in Norway between 1999 and 2013

	Spastic					
CP subtypes	**Hemiplegic**	**Diplegic**	**Quadriplegic**	**Dyskinetic**	**Ataxic**	**Other/unspecified**
Causes	%	%	%	%	%	%
Cerebrovascular events	90.9		4.5			4.5
Head injuries/other accidents	40.0		33.3	6.7	13.3	6.7
Infections	23.1	15.4	46.2	15.4		
Hypoxic insults	20.0	20.0	20.0	30.0	10.0	
Tumour associated	100.0					
Other		50.0	50.0			

TABLE 4 Presence of perinatal risk factors among children in Norway born between 1999 and 2013 with postneonatal cerebral palsy (PNCP) and the general population

	PNCP			General population(live births)		
Perinatal risk factors	**n**	**(%)**	**[95% CI]**	**n**	**(%)**	**[95% CI]**
Prematurity[a]	13	(20.3)	[12.3; 31.7]	63,712	(7.1)	[7.1; 7.2]
5-min Apgar scores[b]						
0–3	0	(0.0)	[0; 5.7]	7499	(0.8)	[0.8; 0.9]
4–6	3	(4.7)	[1.6; 12.9]	8861	(1.0)	[1.0; 1.0]
7–10	61	(95.3)	[87.1; 98.4]	873,946	(97.5)	[97.5; 97.6]
Multiple births	6	(9.0)	[4.2; 18.2]	31,765	(3.5)	[3.5; 3.6]
Congenital malformation	12	(17.9)	[10.6; 28.7]	39,813	(4.4)	[4.4; 4.5]

[a]Missing in 3 (4.5%) children with PNCP and in 6257 (0.7%) in the general population.

[b]Missing in 3 (4.5%) children with the PNCP and in 3576 (0.4%) in the general population.

measures, including the decrease in the incidence of SIDS,[22] reduction of severe infections due to the introduction of new vaccines[23] and a decrease in the prevalence of traumatic head injuries in children born (ie motor vehicle-pedestrian accidents).[5] A further significant difference between our study and the study by Reid et al. is that the upper age limit for the injury causing CP was age five years in that study, while it is age two years in Norway.

Compared with three other studies[3,4,24] where infections were the leading cause of PNCP, cerebrovascular insults and head injuries/other accidents were more common in Norway. This finding may reflect the effect of the introduction of vaccination against Haemophilus influenzae type b and Streptococcus pneumoniae. Consistent with our study, previous studies have found that spastic unilateral and spastic quadriplegic/dyskinetic CP are the predominant subtypes in PNCP.[3,5] The relationship between assumed causes and CP subtypes is also consistent with other studies,[3,5] although the strong association between infections and more severe motor and associated impairments has to our knowledge not been described previously.

Our findings of more severe impairments among children with PNCP than among children with non-PNCP are also reported in three previous studies.[3,4,21] However, the study by Reid et al. did not find any differences in the distributions of motor impairment severity between non-PNCP and PNCP.[5] These different results may also be due to the different definition of PNCP.[3,4,25,26]

Our finding of a higher prevalence of perinatal risk factors for CP among children with PNCP than in the general population is also partially consistent with the study by Reid et al., where the authors reported that a higher proportion of children with PNCP were born preterm than expected.[5] There have also been reports showing some evidence for an increased risk of PNCP among children with a low birthweight[3] or other neonatal risk factors for CP.[22]

A recent multicentre study of 468 children, including data from the NorCP, studied the association between congenital anomalies and PNCP in children born between 1999 and 2009.[24] The authors found that 25.6% of children had congenital anomalies, in which cardiac anomalies were the most common.[25] These findings are in line with our findings.

4.3 | Interpretation of the findings

The proportion of children with PNCP was lower than in previous studies, and the prevalence has continued to decrease. This may be due to the introduction of the vaccines against Haemophilus influenzae type b (1992) and pneumococcal disease (2006) in Norway,[23,27]

as well as the preventive measures against Reye's syndrome since the 1980s.[28] In addition, there has been recommendations to change sleeping position after the SIDS epidemic in the 1980s[22] and a decrease in traffic accidents among children between zero and five years has been reported in Norway since 1999.[29]

The high proportion of children with PNCP with spastic hemiplegia compared to children with non-PNCP is reasonable since cerebrovascular events were the major causal event. Moreover, seven of the 20 children in this group had a cardiac malformation, and it is most likely that an infarction or bleeding occurred during or after surgery. However, we do not have sufficient data to explore this further. Also, we assume that brain tumours are most likely localised in one hemisphere resulting in spastic hemiplegic CP. Whether the CP diagnosis in these cases was due to damage caused by the tumour itself before surgery, or whether it was due to a complication of surgery is unknown.

Moreover, it is also reasonable that head injuries/other accidents, near drowning and near miss-SIDS more commonly resulted in spastic quadriplegic/dyskinetic CP than in other CP subtypes since such injuries are likely to result in more global insults to the brain (ie the cortex, the basal ganglia and the thalamus).[30]

A higher proportion of severe motor and associated impairments was found among children with PNCP than among children with non-PNCP. We assume that the high proportion of children with severe motor and associated impairments was a result of the higher proportion of spastic quadriplegic/dyskinetic CP.[25,26]

We found that known perinatal risk factors for CP were more common among children with PNCP than in the general population. Even though misclassification could in part explain this finding, we speculate that the association may have at least three further plausible explanations. First, children born premature with a low birthweight and Apgar score may be less likely to tolerate critical events after the neonatal period, such as infections, accidents and hypoxia than children born term without the other risk factors. Secondly, it has been documented that children born preterm or small for gestational age are at a higher risk of attention and hyperactivity problems and may therefore be more prone to be exposed to accidents during the first two years of life.[31] Finally, the postneonatal event may not be the cause of the CP diagnosis. Instead, the child may already have a brain injury at birth, which is the cause of the CP while the postneonatal event is just coincidental.

5 | CONCLUSION

The prevalence of PNCP in Norway is low and decreasing. Children who were diagnosed with PNCP were more likely to have spastic hemiplegic CP or spastic quadriplegic/dyskinetic CP than children with non-PNCP, as well as with more severe associated impairments. An interesting finding was that a number of perinatal risk factors for non-PNCP were more commonly present among children with PNCP than in the general population. We suggest that future research should include more detailed clinical information about children with PNCP, including perinatal risk factors, other factors related to causal pathways and the timing of the brain injury.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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