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CEREBRAL PALSY IN CHILDREN: CLINICAL PROFILE, RISK FACTORS, AND ASSESSMENT COMPARED WITH OTHER NEUROLOGICAL DISORDERS

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Abstract

This case-control study compared clinical, perinatal, and neuroimaging characteristics between 18 children with cerebral palsy (CP) and 18 children with other, heterogeneous pediatric neurological disorders evaluated at the same clinic. Children with CP had lower gestational age (34.2 ± 4.1 vs 37.9 ± 2.3 weeks) and birth weight (2210 ± 780 vs 2980 ± 540 g), and more often had a history of perinatal asphyxia (50.0% vs 11.1%), abnormal brain MRI (88.9% vs 33.3%), and abnormal muscle tone or spasticity (94.4% vs 27.8%; all $p<0.05$). Comorbid epilepsy (38.9% vs 22.2%) and developmental delay (66.7% vs 50.0%) were more frequent in the CP group but did not reach statistical significance given the small sample. Motor severity varied across Gross Motor Function Classification System levels I–V. These findings, interpreted cautiously given the limited sample size, support perinatal history and neuroimaging as key discriminators between CP and other pediatric neurological conditions.

Keywords: cerebral palsy; risk factors; Gross Motor Function Classification System; neuroimaging; perinatal asphyxia; differential diagnosis; pediatric neurology

Introduction

Cerebral palsy (CP) remains the most common cause of childhood physical disability, arising from a non-progressive injury to the developing brain occurring from the embryonic period through early childhood [2]. A recently updated Chinese expert consensus reaffirmed three diagnostic pillars for CP — non-progressive brain injury, absence of motor regression, and, in selected cases, an underlying genetic contribution — and highlighted cerebral MRI, the General Movements Assessment (GMA), and the Hammersmith Infant Neurological Examination (HINE) as the principal tools for early, accurate diagnosis [2]. A recent feasibility study demonstrated that a structured early-detection pathway combining GMA, HINE, and neuroimaging can bring the mean age of CP diagnosis down to 4.3 months, regardless of a family's region of residence or ethnicity [3]. National CP registers, such as the newly proposed Vietnam Cerebral Palsy Register, continue to be established to systematically capture incidence, risk factors, and motor severity using the Gross Motor Function Classification System (GMFCS) [4].

Perinatal risk factors remain central to the etiology of CP. Cohort data from a tertiary neonatal unit in Pakistan found that placental abruption, prematurity, and low birth weight independently increased the hazard of later neurodevelopmental impairment, including cerebral palsy [5]. Similarly, early hyponatremia following perinatal asphyxia has been shown to markedly increase the odds of a later CP diagnosis (adjusted OR 8.94) [6], and, in children who already carry a CP diagnosis, a low 5-minute Apgar score is strongly associated with more severe comorbid epilepsy on neuroimaging-correlated severity scales [7].

Despite this progress, CP can still be difficult to distinguish clinically from other non-progressive or slowly evolving pediatric neurological conditions, particularly when perinatal history

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is not immediately available; a growing body of genetic evidence shows that a meaningful proportion of children with a presumptive CP diagnosis instead carry an unrelated monogenic disorder or CP-mimicking condition, underscoring the importance of careful phenotyping before the label is applied [1]. Few small-sample clinical studies have directly compared the perinatal, clinical, and neuroimaging features of children with confirmed CP against children with other neurological diagnoses evaluated in the same clinical setting. This study aimed to compare the clinical profile, risk factors, and assessment findings of children with CP against a comparison group of children with other pediatric neurological disorders.

Methods

This case-control study was conducted in a pediatric neurology clinic between March 2025 and August 2026. Eighteen children aged 1–12 years with a confirmed diagnosis of CP, established according to standard non-progressive brain injury criteria and classified by the Gross Motor Function Classification System (GMFCS I–V), formed the index group. Eighteen children of comparable age evaluated in the same clinic for other, non-CP neurological diagnoses (including idiopathic epilepsy without motor impairment, hereditary neuromuscular disease, isolated global developmental delay, and hereditary ataxia) formed the comparison group. Data on gestational age, birth weight, perinatal history, muscle tone and spasticity, comorbid epilepsy, developmental delay, and brain MRI findings were extracted from structured clinical assessment and medical records. In a younger subset of each group (CP, n=6; comparison, n=5), the Hammersmith Infant Neurological Examination (HINE) global score was additionally recorded.

Continuous variables are reported as mean ± standard deviation (M ± SD) and compared with the independent-samples t-test; categorical variables were compared with the chi-square test or Fisher's exact test, as appropriate for small cell counts. Given the small sample size (n=18 per group), statistical power to detect moderate effect sizes was limited; results are therefore interpreted cautiously, and comparisons that did not reach conventional significance are reported as such rather than as evidence of no difference. A two-tailed $p<0.05$ was considered statistically significant.

Results

The CP and comparison groups were similar in age at assessment (5.8 ± 2.6 vs 6.2 ± 2.9 years; p=0.68) and sex distribution (61.1% vs 50.0% male; p=0.51). Children with CP had significantly lower gestational age at birth (34.2 ± 4.1 vs 37.9 ± 2.3 weeks; p=0.004) and lower birth weight (2210 ± 780 vs 2980 ± 540 g; p=0.006) than the comparison group (Table 1).

Characteristic	CP group (n=18)	Comparison group (n=18)	p value
Gestational age, wk (M ± SD)	34.2 ± 4.1	37.9 ± 2.3	0.004
Birth weight, g (M ± SD)	2210 ± 780	2980 ± 540	0.006
Perinatal asphyxia / HIE, n (%)	9 (50.0%)	2 (11.1%)	0.012
Abnormal muscle tone/spasticity, n (%)	17 (94.4%)	5 (27.8%)	<0.001
Abnormal brain MRI, n (%)	16 (88.9%)	6 (33.3%)	<0.001
Comorbid epilepsy, n (%)	7 (38.9%)	4 (22.2%)	0.29
Developmental delay, n (%)	12 (66.7%)	9 (50.0%)	0.31

Table 1. Comparison of perinatal, clinical, and neuroimaging characteristics between the cerebral palsy and comparison groups

A history of perinatal asphyxia or hypoxic-ischemic encephalopathy was present in 9/18 children with CP (50.0%) compared with 2/18 in the comparison group (11.1%; p=0.012). Abnormal brain MRI findings were documented in 16/18 children with CP (88.9%) versus 6/18 in the

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comparison group (33.3%; p<0.001), and abnormal muscle tone or spasticity was present in 17/18 children with CP (94.4%) compared with 5/18 in the comparison group (27.8%; p<0.001).

Within the CP group, motor severity by GMFCS was distributed across all five levels: level I in 3 children (16.7%), level II in 5 (27.8%), level III in 4 (22.2%), level IV in 4 (22.2%), and level V in 2 (11.1%). Comorbid epilepsy was more frequent in the CP group (7/18, 38.9%) than in the comparison group (4/18, 22.2%), but this difference did not reach statistical significance (p=0.29). Developmental delay was likewise numerically more common in the CP group (12/18, 66.7%) than in the comparison group (9/18, 50.0%), again without reaching significance (p=0.31); both comparisons should be interpreted cautiously given the limited sample size.

In the younger subset assessed with the HINE (CP, n=6; comparison, n=5), the mean global HINE score was markedly lower in children with CP (38.2 ± 9.4) than in the comparison subgroup (54.6 ± 6.1; p=0.004), consistent with the tool's established discriminative value, although this subgroup comparison is based on very few children and should be treated as preliminary.

Discussion

In this small case-control comparison, children with confirmed CP differed most clearly from children with other pediatric neurological disorders in perinatal history, neuroimaging findings, and motor tone — precisely the domains emphasized by the current diagnostic framework for CP, which anchors diagnosis in a non-progressive brain injury demonstrable on MRI together with a persistent motor abnormality [2]. The lower gestational age, lower birth weight, and higher rate of perinatal asphyxia observed in the CP group are consistent with cohort evidence identifying prematurity, low birth weight, and placental abruption as independent risk factors for later neurodevelopmental impairment [5], and with data showing that early hyponatremia after perinatal asphyxia substantially raises the odds of a later CP diagnosis [6].

The comorbid epilepsy rate in our CP group (38.9%) is broadly in line with, if numerically lower than, rates reported in larger cross-sectional cohorts of children with CP and epilepsy, where seizure severity has been linked to grey-matter MRI injury and low 5-minute Apgar scores [7]; our study was not designed or powered to examine this perinatal-severity relationship directly. That comorbid epilepsy and developmental delay were only numerically, not statistically, more frequent in our CP group illustrates a real limitation of comparing 18 children against 18 children: modest but potentially clinically meaningful differences can fail to reach significance purely because of sample size, and these findings should not be read as evidence that CP and other neurological disorders carry a similar comorbidity burden.

The markedly lower HINE global score in the small CP subgroup mirrors the tool's intended role as a discriminative early-assessment instrument, and aligns with recent evidence that combining HINE, GMA, and neuroimaging in a structured early-detection pathway can meaningfully reduce the age at CP diagnosis [3]. Because a meaningful minority of children who appear to have CP instead have an unrelated genetic or CP-mimicking disorder [1], the clearer perinatal and neuroimaging differences identified here reinforce the practical value of thorough perinatal history-taking and MRI review before finalizing a CP diagnosis, rather than relying on motor examination alone. Assessment tools developed for children already diagnosed with CP — including GMFCS-stratified gait analysis [9] and GMFCS-linked measures such as diaphragmatic thickness as a marker of systemic muscle and nutritional status [8] — may have a role in further characterizing disease severity once the diagnosis is secure, though such tools were outside the scope of the present comparison.

This study's principal limitation is its small sample size (18 per group), which limits statistical power, widens confidence intervals, and precludes multivariable adjustment for confounders such as age or comorbidity; the comparison group was also clinically heterogeneous rather than a single diagnostic entity, which may obscure differences specific to any one alternative diagnosis. Larger,

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prospective, multicentre studies — ideally incorporating standardized registries such as those now being developed regionally [4] — are needed to confirm these preliminary findings and to refine the clinical and neuroimaging criteria that best distinguish CP from its mimics.

Conclusion

Even at a modest scale, this comparison points to a consistent pattern: children with cerebral palsy carry a perinatal and neuroimaging signature — lower gestational age and birth weight, more frequent asphyxia, and far more frequent MRI abnormality and spasticity — that sets them apart from children with other neurological diagnoses seen in the same clinic, while comorbidities such as epilepsy and developmental delay overlap more than they diverge. That overlap is itself a clinically useful reminder: a motor examination alone should never carry the full weight of a CP diagnosis. Pairing careful perinatal history-taking with structured early assessment tools and neuroimaging remains the most reliable way to tell cerebral palsy apart from its mimics, and to do so as early in a child's life as possible.

References

1. D.-H. Jang, J. Kim, A. L. Schwabe, and T. E. Lotze, “Genetics of Cerebral Palsy: Diagnosis, Differential Diagnosis, and Beyond,” Ann. Rehabil. Med., vol. 48, no. 6, pp. 369–376, 2024.
2. W. Jiang, D. Zhu, X. Tang, K. Xu, C. Zhu, and N. Xiao, “Chinese expert consensus on the diagnostic definition of cerebral palsy,” BMJ Paediatr. Open, vol. 10, no. 1, 2026.
3. A. A. Fletcher et al., “Equitable access to cerebral palsy assessments and diagnosis through an early detection Hub: a feasibility study,” Pediatr. Res., 2026.
4. T. H. H. Khuc et al., “Vietnam Cerebral Palsy Register: protocol for co-design of a national register with people with lived experience of cerebral palsy,” BMJ Open, vol. 16, no. 7, p. e116376, 2026.
5. S. K. Jafri et al., “Surveillance for neurodevelopmental impairment (NDI) in high-risk neonates: a cohort study at a tertiary care hospital in Pakistan,” BMJ Open, vol. 16, no. 8, p. e121569, 2026.
6. N. Viitaharju, K. Helenius, E. Löyttyniemi, B. Singh, and V. Parikka, “Association between early hyponatremia and neurological diagnoses in infants with perinatal asphyxia,” Eur. J. Pediatr., vol. 185, no. 9, 2026.
7. A. Wander et al., “Epilepsy in children with cerebral palsy: perinatal, clinical and neuroimaging correlates — a cross-sectional study from a tertiary care centre in North India,” BMJ Paediatr. Open, vol. 10, no. 1, 2026.
8. M. B. M. Sobreira et al., “Diaphragmatic Thickness as a Marker of Systemic Muscle Depletion in Non-Ambulatory Children with Cerebral Palsy: Implications for Integrated Neurorehabilitation Assessment,” NeuroSci, vol. 7, no. 5, 2026.
9. L. Donno et al., “Effects of Lokomat-based robotic-assisted gait training and physical therapy on gait quality in children with cerebral palsy: A severity-stratified analysis,” Gait Posture, vol. 131, p. 110628, 2026.