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Clinical Correlates of the Onodera Prognostic Nutritional Index in Children with Cerebral Palsy: A Cross-Sectional Study

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Abstract

Background.

The clinical relevance of the Onodera Prognostic Nutritional Index (PNI) in children with cerebral palsy remains unclear. We aimed to characterize PNI in children with cerebral palsy and examine its relationships with anthropometry, motor function, epilepsy, and blood count–derived inflammatory indices.

Methods.

This retrospective, single-center, cross-sectional study screened 215 children and included 171 with complete data. PNI was compared across body mass index (BMI)-, weight-, and height-based categories, Gross Motor Function Classification System (GMFCS) groups, and epilepsy status. Group comparisons used Mann–Whitney U tests, and associations with anthropometric z scores were assessed using Spearman correlations. BMI-specific analyses included a location shift, directional area under the curve (AUC), and adjusted logistic regression. Inflammatory indices were exploratory.

Results.

Median PNI was 58.00 (IQR 53.02–62.83). PNI was lower in children with epilepsy (q = 0.006) but did not differ by underweight status or GMFCS group and was not correlated with anthropometric z scores. Fifty-one children (29.8%) had BMI-for-age z score ≤ − 2. For the location-shift analysis, the BMI-for-age z score ≤ − 2 group was compared with the > − 2 group. The PNI location shift was 1.10 points (95% confidence interval [CI] − 1.55 to 3.75), the directional AUC, with lower PNI indicating BMI-for-age z score ≤ − 2, was 0.459 (95% CI 0.363–0.553), and the adjusted odds ratio per 5-point PNI reduction was 0.86 (95% CI 0.70–1.06). Older age (adjusted odds ratio per year 1.09, 95% CI 1.01–1.18) and GMFCS IV–V were associated with BMI-for-age z score ≤ − 2 (adjusted odds ratio 3.90, 95% CI 1.65–9.21). Inflammatory indices were not correlated with anthropometric z scores.

Conclusions

. PNI was associated with epilepsy status but showed little relationship with anthropometric measures or motor-function group. It should not be interpreted as a direct measure of body-size-based nutritional status in cerebral palsy. Longitudinal studies should determine whether it provides prognostic information beyond routine clinical and anthropometric assessment.

Background

Cerebral palsy describes a group of permanent disorders affecting movement and posture that result from non-progressive disturbances in the developing brain [1]. Its effects extend beyond motor function and may include impaired oromotor control, feeding difficulties, gastrointestinal symptoms, altered growth, and epilepsy. Together with reduced mobility, altered energy requirements, and differences in body composition, these features contribute to substantial nutritional risk [2, 3].

Assessing nutritional status in children with cerebral palsy can be challenging. Weight and height remain the basis of routine assessment, but obtaining an accurate standing height is often difficult in children with contractures, scoliosis, or marked motor impairment. BMI may also give an incomplete picture of body composition when low muscle mass coexists with relatively preserved fat mass [4, 5]. These limitations do not lessen the value of anthropometry, but they make it reasonable to examine complementary clinical and laboratory measures.

The Onodera Prognostic Nutritional Index (PNI) combines serum albumin and absolute lymphocyte count. It was developed to estimate perioperative risk in malnourished patients undergoing gastrointestinal surgery [6]. Later studies have used it mainly for prognostic and immunonutritional risk stratification rather than as a stand-alone test for malnutrition. Neither component is specific to nutritional status: albumin is influenced by inflammation, hydration, hepatic and renal function, and illness severity, while lymphocyte counts vary with age and immune activity [7, 8]. Pediatric work has likewise focused largely on disease severity or prognosis [9, 10]. The meaning of PNI in children with neurodisability therefore remains uncertain.

We aimed to characterize the distribution of PNI in children with cerebral palsy and examine its relationships with anthropometric status, motor function, epilepsy, and routinely available blood count–derived inflammatory indices. BMI-, weight-, and height-based measures were treated as anthropometric correlates, and the inflammatory analyses were exploratory.

Methods

Study design, reporting and ethical approval

This exploratory retrospective cross-sectional study used routine-care records of children with cerebral palsy evaluated at a tertiary pediatric neurology outpatient clinic between 15 June 2020 and 15 June 2026. Reporting was guided by the STROBE statement and the RECORD extension for studies using routinely collected health data [11, 12].

The local Clinical Research Ethics Committee approved the study (approval no. 244–2026) and waived individual informed consent because the analysis used de-identified information collected during routine care. The study was conducted in accordance with the Declaration of Helsinki.

Participants and record selection

Medical records and hospital information system data were reviewed for children with a documented diagnosis of cerebral palsy who attended during the study period. Identifier-level checks ensured that each child was represented once. Entry into the analytic cohort required the clinical, anthropometric, and laboratory information needed to calculate PNI and anthropometric z scores and to make the planned group comparisons.

Of 215 screened records, 43 lacked both weight and height and one lacked height. These 44 records were excluded, leaving 171 children in the complete-case analysis. Missing values were not imputed. Characteristics available for both included and excluded records are reported with variable-specific denominators in Additional file 1.

Clinical and anthropometric variables

The extracted clinical variables were age, sex, motor function, and epilepsy status. Motor function was grouped as Gross Motor Function Classification System (GMFCS) levels I–III or IV–V using the assessment recorded by the treating pediatric neurologist [13]. Epilepsy status was based on the documented clinical diagnosis. Cerebral palsy subtype, feeding route, seizure characteristics, and detailed treatment information were not available consistently and were not analyzed.

Weight and standing height or recumbent length were taken from routine clinical records. Anthropometry and laboratory results from the same clinical assessment were selected. BMI was calculated as weight in kilograms divided by height in meters squared. Weight-, height-, and BMI-for-age z scores were recalculated by age and sex using the national reference values reported by Neyzi et al. [14]. Lambda–mu–sigma values were linearly interpolated between available age points.

A BMI-for-age z score ≤ − 2 was classified as low BMI-for-age and used as the study's BMI-based definition of undernutrition. This threshold represents at least a moderate BMI deficit in the Academy/ASPEN framework for children aged 2 years or older [15]. Weight- and height-for-age z scores ≤ − 2 defined underweight and short stature. Analyses were repeated in children aged 2 years or older because BMI-for-age is less well established below that age.

Laboratory measures and derived indices

Serum albumin and complete blood count results from the selected clinical assessment were extracted. PNI was calculated in SI units as serum albumin (g/L) + 5 × absolute lymphocyte count ($\times 10^9$/L). This is equivalent to the original expression of 10 × albumin (g/dL) + 0.005 × lymphocyte count/mm^3 [6].

Four blood count–derived indices were calculated: neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII; platelet count × absolute neutrophil count / absolute lymphocyte count).

Sample size and statistical analysis

The sample size was determined by the eligible records available during the study period; no a priori calculation was performed. All complete records were included, and effect estimates are reported with 95% confidence intervals.

Continuous variables are summarized as medians with IQRs and categorical variables as counts and percentages. Continuous and categorical variables were compared with the Mann–Whitney U test and Fisher's exact test, respectively. PNI was compared across BMI-for-age, underweight, short-stature, GMFCS, and epilepsy groups. Spearman coefficients described its correlations with continuous anthropometric z scores.

The BMI-for-age comparison was examined in greater detail because it was supported by a defined z-score threshold. We estimated the Hodges–Lehmann shift for the group with BMI-for-age z score ≤ − 2 relative to the group with a z score > − 2. Its 95% confidence interval was obtained from 10,000 stratified bootstrap resamples. Directional receiver operating characteristic analysis treated lower PNI, albumin, and lymphocyte count as corresponding to BMI-for-age z score ≤ − 2. This analysis quantified correspondence with the BMI category and was not treated as validation against a diagnostic gold standard.

A multivariable logistic model examined BMI-for-age z score ≤ − 2 in relation to PNI, age, sex, and GMFCS group. PNI was scaled per 5-point reduction. To limit multiplicity, Benjamini–Hochberg correction was applied to the four other PNI group comparisons—underweight, short stature, GMFCS, and epilepsy—and separately to the three anthropometric correlations [16]. Group comparisons and correlations involving NLR, PLR, MLR, and SII were considered exploratory and are reported in Additional file 1.

Sensitivity analyses were restricted to children aged 2 years or older, excluded BMI-for-age z scores outside − 5 to 5, excluded selected laboratory extremes (white blood cell count > 15×10^9/L, absolute lymphocyte count > 8×10^9/L, or albumin < 30 g/L), and applied both sets of restrictions. These restrictions assessed the influence of extreme observations and possible acute illness; they were not clinical exclusion criteria. All tests were two-sided. Unadjusted p values and false-discovery-rate-adjusted q values are reported as appropriate. Analyses used Python 3.13.14 with pandas 2.2.3, NumPy 2.3.5, SciPy 1.17.1, statsmodels 0.14.6, and scikit-learn 1.6.1. The bootstrap seed was 20260917.

Generative AI tools were used for language revision and statistical quality control; all analytical decisions, interpretation, and final verification remained the responsibility of the authors.

Results

Study population and anthropometric profile

Of 215 children screened, 171 entered the analysis (Fig. 1). Their median age was 7.0 years (IQR 4.27–11.0); 98 (57.3%) were boys, 114 (66.7%) had GMFCS levels IV–V, and 116 (67.8%) had epilepsy. Fifty-one children (29.8%) had a BMI-for-age z score ≤ − 2, 80 (46.8%) were underweight, and 81 (47.4%) had short stature. Children with BMI-for-age z score ≤ − 2 were older (median 8.0 vs 7.0 years; p = 0.038) and more often had GMFCS IV–V (84.3% vs 59.2%; p = 0.001); sex and epilepsy status were similar between BMI groups (Table 1).

Table 1
Cohort characteristics overall and by BMI-for-age category

Characteristic	All (n = 171)	BMI z > − 2 (n = 120)	BMI z ≤ − 2 (n = 51)	p
Age, years	7.00 (4.27–11.00)	7.00 (4.00–10.25)	8.00 (5.50–13.00)	0.038
Male sex	98 (57.3)	69 (57.5)	29 (56.9)	1.000
GMFCS IV–V	114 (66.7)	71 (59.2)	43 (84.3)	0.001
Epilepsy	116 (67.8)	77 (64.2)	39 (76.5)	0.152
Weight-for-age z ≤ − 2	80 (46.8)	33 (27.5)	47 (92.2)	< 0.001
Height-for-age z ≤ − 2	81 (47.4)	47 (39.2)	34 (66.7)	0.001
BMI, kg/m²	15.50 (13.54–17.58)	16.62 (15.15–18.67)	12.35 (11.72–13.18)	—
BMI-for-age z score	−0.65 (− 2.17–0.65)	0.10 (− 0.78–0.87)	−3.11 (− 4.54–−2.35)	—
Serum albumin, g/L	44.00 (40.00–46.00)	44.00 (40.75–46.00)	43.00 (39.50–46.00)	0.635
Absolute lymphocytes, ×10⁹/L	2.80 (2.04–3.77)	2.71 (2.01–3.70)	3.01 (2.18–3.85)	0.342
PNI	58.00 (53.02–62.83)	57.77 (52.89–62.51)	59.15 (53.77–63.43)	0.394

Values are median (IQR) or n (%). Continuous variables were compared with the Mann–Whitney U test and categorical variables with Fisher's exact test. Comparisons were not performed for BMI and BMI-for-age z score because these variables define the displayed groups. BMI, body mass index; GMFCS, Gross Motor Function Classification System; IQR, Interquartile range; PNI, Prognostic Nutritional Index

PNI distribution and clinical correlates

Median PNI was 58.00 (IQR 53.02–62.83), with median albumin 44.00 g/L (IQR 40.00–46.00) and absolute lymphocyte count 2.80×10⁹/L (IQR 2.04–3.77). Children with epilepsy had lower PNI than those without epilepsy (57.10 [IQR 52.46–61.83] vs 59.50 [IQR 56.33–66.20]; p = 0.001, q = 0.006). PNI was

also lower in children with short stature before multiplicity correction (p = 0.045), but the adjusted result was not significant (q = 0.089). No difference was found by underweight status or GMFCS group (Table 2; Additional file 1, Fig. S1).

Table 2
Onodera PNI across anthropometric and clinical profiles

Variable	Group 1	PNI, median (IQR)	Group 2	PNI, median (IQR)	p	q
BMI-for-age z score	>−2 (n = 120)	57.77 (52.89–62.51)	≤−2 (n = 51)	59.15 (53.77–63.43)	0.394	—
Weight-for-age z score	>−2 (n = 91)	57.75 (53.05–62.52)	≤−2 (n = 80)	58.17 (52.81–63.14)	0.784	0.784
Height-for-age z score	>−2 (n = 90)	58.48 (54.04–65.01)	≤−2 (n = 81)	57.00 (52.20–61.75)	0.045	0.089
GMFCS level	I–III (n = 57)	58.45 (54.00–65.50)	IV–V (n = 114)	57.77 (52.50–62.33)	0.104	0.138
Epilepsy	Absent (n = 55)	59.50 (56.33–66.20)	Present (n = 116)	57.10 (52.46–61.83)	0.001	0.006

p values are from Mann–Whitney U tests. q values are Benjamini–Hochberg adjusted across the four non-BMI comparisons; the BMI comparison was reported separately with additional location-shift, AUC, and regression analyses. GMFCS, Gross Motor Function Classification System; IQR, interquartile range; PNI, Prognostic Nutritional Index.

PNI was not significantly correlated with BMI-, weight-, or height-for-age z scores. The anthropometric categories themselves agreed only partly: BMI-for-age z score ≤ − 2 showed moderate agreement with underweight (κ = 0.556) and fair agreement with short stature (κ = 0.235; Additional file 1).

Detailed analysis by BMI-for-age category

Median PNI was 59.15 (IQR 53.77–63.43) in children with BMI-for-age z score ≤ − 2 and 57.77 (IQR 52.89–62.51) in those above − 2. The Hodges–Lehmann shift was 1.10 PNI points (95% CI − 1.55 to 3.75; p = 0.394; Table 2 and Fig. 2). With lower PNI specified as the direction corresponding to BMI-for-age z score ≤ − 2, the AUC was 0.459 (95% CI 0.363–0.553). The corresponding AUCs were 0.523 (95% CI 0.425–0.618) for albumin and 0.454 (95% CI 0.357–0.551) for absolute lymphocyte count (Table 3).

Table 3
Directional AUCs of PNI and its components by BMI-for-age category

Index	Direction corresponding to BMI z ≤ − 2	AUC	95% CI
PNI	Lower	0.459	0.363–0.553
Serum albumin	Lower	0.523	0.425–0.618
Absolute lymphocyte count	Lower	0.454	0.357–0.551

AUCs were calculated in the specified direction, with lower values treated as corresponding to BMI-for-age z score ≤ − 2. Confidence intervals were obtained from 10,000 stratified bootstrap resamples. The analysis quantifies correspondence with the BMI category and is not a validation against a diagnostic gold standard. AUC, area under the receiver operating characteristic curve; CI, confidence interval; PNI, Prognostic Nutritional Index.

In the adjusted model, PNI was not associated with BMI-for-age z score ≤ − 2 (odds ratio per 5-point reduction 0.86, 95% CI 0.70–1.06; p = 0.150). GMFCS IV–V was associated with higher odds of BMI-for-age z score ≤ − 2 (odds ratio 3.90, 95% CI 1.65–9.21; p = 0.002), as was increasing age (odds ratio per year 1.09, 95% CI 1.01–1.18; p = 0.025; Table 4).

Table 4
Multivariable model for BMI-for-age z score ≤ − 2

Variable	Adjusted OR	95% CI	p
PNI, per 5-point reduction	0.86	0.70–1.06	0.150
Age, per year	1.09	1.01–1.18	0.025
Male vs female sex	0.90	0.44–1.82	0.765
GMFCS IV–V vs I–III	3.90	1.65–9.21	0.002

The model included 171 children and 51 events (McFadden pseudo-R^2=0.084). It examines association with the BMI-derived category and is not a diagnostic model. CI, confidence interval; GMFCS, Gross Motor Function Classification System; OR, odds ratio; PNI, Prognostic Nutritional Index.

Additional file 1 (.docx). Supplementary methods and results. Contains Tables S1–S5 and Fig. S1.

Exploratory and sensitivity analyses

Among the blood count–derived indices, NLR was higher in children with epilepsy and SII was higher in the GMFCS IV–V group after false-discovery-rate correction. None of the four indices correlated significantly with the anthropometric z scores. Other group differences were not significant after correction (Additional file 1).

The BMI-related PNI estimates changed little after restriction to children aged 2 years or older or after anthropometric and laboratory extreme-value restrictions. Directional AUCs ranged from 0.462 to 0.499, and adjusted PNI estimates remained non-significant (Additional file 1).

Discussion

This study describes PNI across several clinical and anthropometric profiles in children with cerebral palsy. Children with epilepsy had lower PNI values. In contrast, PNI varied little across BMI-, weight-, height-, or GMFCS-based groups. The BMI-specific analyses supported this finding: the difference between BMI categories was small, the AUC was near 0.50, and PNI was not associated with a BMI-for-age z score ≤ − 2 after adjustment for age, sex, and GMFCS group. In the same model, GMFCS IV–V was associated with a BMI-for-age z score ≤ − 2.

PNI was developed by Onodera and colleagues to estimate perioperative risk in malnourished surgical patients [6]. Most subsequent studies have examined complications, survival, or disease severity rather than anthropometric malnutrition [17]. In this cohort, PNI did not closely follow body-size-based classifications; this does not address its original prognostic purpose.

The weak association with anthropometry may reflect the different components of these measures. BMI is determined by weight and height, whereas PNI combines albumin and lymphocyte count. Both laboratory measures can change with inflammation, acute illness, hydration, organ function, and age [7, 8]. Lark et al. similarly reported that albumin and prealbumin did not reflect anthropometric nutritional status in children with cerebral palsy [18]. Group-level differences in albumin or PNI reported in other clinical settings do not establish that either measure accurately classifies the nutritional status of an individual child.

Anthropometry has its own limitations in cerebral palsy. Contractures, scoliosis, and postural abnormalities make height difficult to measure, and BMI does not distinguish fat mass from fat-free mass [4, 5]. The incomplete agreement among BMI-, weight-, and height-based categories in our data illustrates this problem. Current guidance recommends that nutritional assessment in children with neurological impairment draw on feeding history, examination, serial anthropometry, and body-composition measures when available [3]. PNI should not replace that assessment.

GMFCS IV–V was independently associated with low BMI-for-age. Feeding and swallowing problems become more common with increasing motor impairment and may occur alongside altered energy requirements, reduced mobility, and greater dependence at mealtimes [19, 20]. The available data did not allow us to determine why this association occurred, because detailed information on dysphagia, feeding route, energy intake, and body composition was unavailable.

The lower PNI in children with epilepsy was the most consistent clinical association. Epilepsy in cerebral palsy often accompanies a greater overall neurological burden [21], but the present data cannot explain the finding. Seizure frequency, seizure control, and treatment details were unavailable. The result is therefore hypothesis-generating and should not be interpreted as evidence that epilepsy itself lowers PNI.

The exploratory inflammatory analyses were mixed. NLR was higher in children with epilepsy and SII in the GMFCS IV–V group, but none of the indices was consistently related to anthropometric measures. A single blood count may reflect transient immune activity or recent illness. These exploratory findings require confirmation.

We recalculated all z scores from the recorded measurements using one national reference and reported effect estimates with confidence intervals. Multiplicity correction was applied to the secondary and exploratory families, and the BMI-related PNI estimate was examined under several data restrictions.

This retrospective single-center design prevents causal inference and may limit generalizability. We excluded 44 of the 215 screened records because of incomplete data, creating a possible source of selection bias. GMFCS could be analyzed only as two broad groups, I–III and IV–V. Several clinically relevant factors were not documented consistently, including cerebral palsy subtype, feeding route, dysphagia, gastrointestinal symptoms, body composition, acute infection, hydration status, liver or kidney dysfunction, and treatments that could influence albumin or blood-cell counts. Weight and height came from routine care, and detailed measurement protocols or repeatability data were unavailable. PNI was based on one laboratory assessment, has no validated pediatric threshold, and was not tested against longitudinal outcomes. This study therefore cannot establish the prognostic value of PNI in cerebral palsy.

Conclusions

In this cohort, PNI differed according to epilepsy status but showed no consistent association with anthropometric measures or GMFCS group. Our data do not support using PNI to infer nutritional status from weight, height, or BMI, or to replace routine nutritional assessment. Prospective longitudinal studies are needed to determine whether PNI predicts clinical outcomes in children with cerebral palsy.

Abbreviations

AUC
area under the receiver operating characteristic curve
BMI
body mass index
CI
confidence interval
GMFCS
Gross Motor Function Classification System
IQR
interquartile range
MLR
monocyte-to-lymphocyte ratio

NLR
neutrophil-to-lymphocyte ratio
PLR
platelet-to-lymphocyte ratio
PNI
Prognostic Nutritional Index
SII
systemic immune-inflammation index

Declarations

Running title

Clinical correlates of PNI in cerebral palsy

Declarations

Ethics approval and consent to participate:

Approved by the Haseki Training and Research Hospital Clinical Research Ethics Committee (Approval number. 244–2026). The requirement for informed consent was waived owing to the retrospective design.

Consent for publication:

Not applicable.

Competing interests:

The authors declare that they have no competing interests.

Clinical trial number

Not applicable.

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Author Contribution

S.B.Y.: Methodology, Project administration, Visualization, Writing – original draft, Writing – review and editing. G.B. and R.A.: Investigation, Data curation. S.M.D. and U.P.: Formal analysis, Methodology, Validation.P.G.: Conceptualization, Supervision, Methodology, Writing – review and editing. All authors read and approved the final manuscript and agreed to be accountable for all aspects of the work.

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Not applicable.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available due to privacy and ethical restrictions.

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Figures

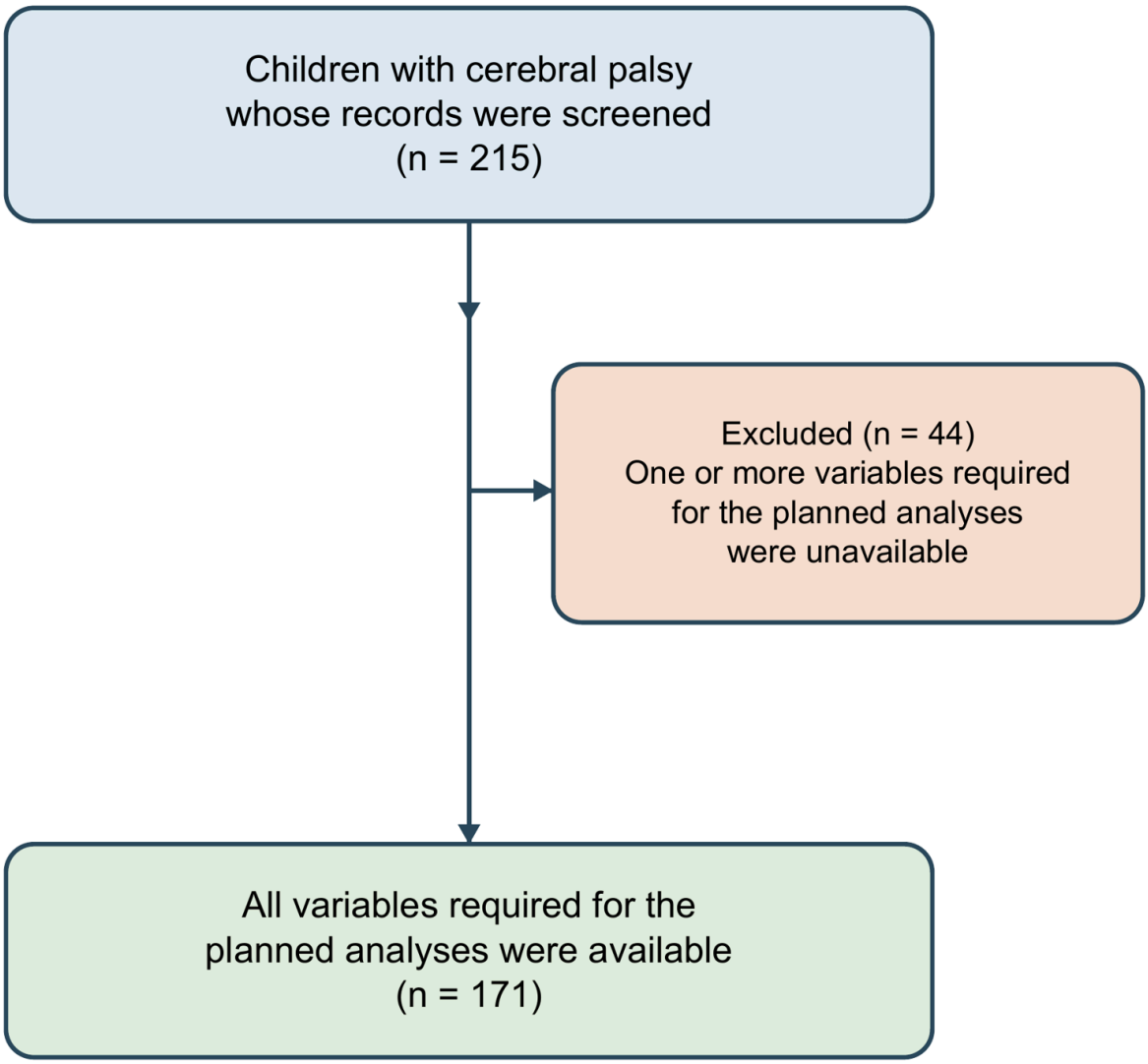


Figure 1

Study Flow

Fig. 1. Of 215 children screened, 44 were excluded because required data were incomplete, leaving 171 children in the analytic cohort.

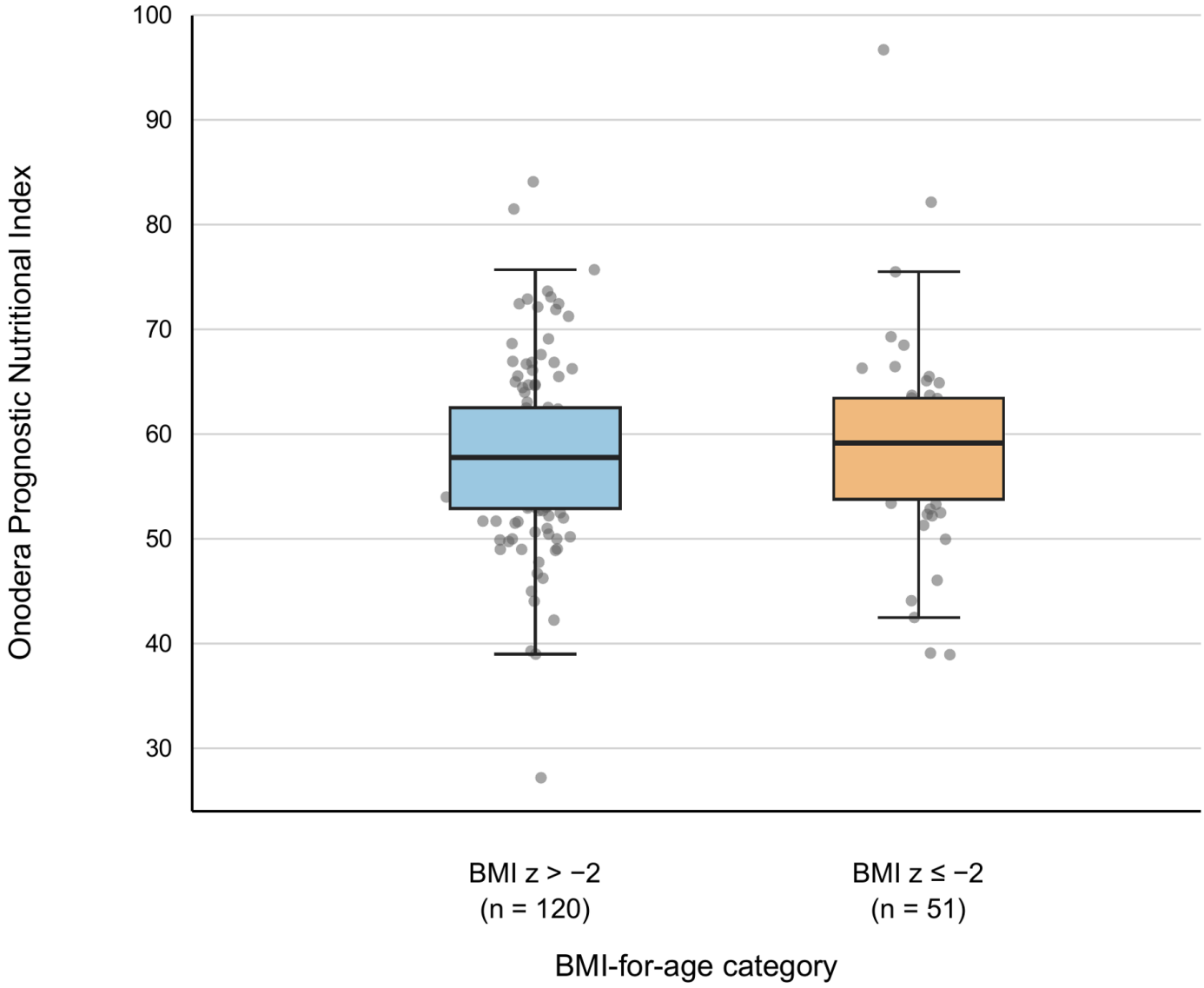


Figure 2

Distribution of the Onodera Prognostic Nutritional Index by BMI-for-age category

Boxes show medians and IQRs; whiskers extend to 1.5 times the IQR and points represent individual children. PNI distributions did not differ between the BMI-for-age z score >−2 and ≤−2 groups (Mann–Whitney U test, p=0.394).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Additionalfile.docx