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Research Article

Research on soft neurological signs obese children

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ABSTRACT

Objective: Soft neurological signs (SNS) are subtle indicators detected during neurological examination in the absence of an overt neurological disorder. This study aimed to investigate the presence of SNS in obese children.

Methods: A total of 61 obese children and 57 healthy controls (HCs) were evaluated using the Neurological Evaluation Scale (NES) to assess the presence of SNS.

Results: There were no significant differences between the obese and HC groups regarding age and sex ($p>0.05$). The sensory integration, motor coordination, complex sequential motor acts, other NES, and total NES scores were significantly higher in the obese group compared with HCs ($p=0.005$, $p=0.009$, $p=0.004$, $p=0.002$, and <0.001, respectively).

Conclusion: This is the first study to demonstrate a significant presence of SNS in obese children using the NES. Incorporating SNS assessment into routine obesity evaluations may help identify children at risk for early neurodevelopmental changes.

Keywords: obesity, soft neurological signs, neurological evaluation scale, children

INTRODUCTION

Obesity is defined as the abnormal or excessive accumulation of body fat.[1] Subclinical inflammatory processes and white matter demyelination, which are related to the duration and severity of obesity, are believed to contribute to cognitive dysfunction.[2]

In particular, chronic inflammation in the developing brain has been suggested to exert neurotoxic effects. In this context, proinflammatory cytokines such as TNF-α and IL-6 have been shown to impair synaptic plasticity and myelination.[3,4] These alterations may lead to deficits in motor coordination, sensory integration, and attention regulation, potentially contributing to the emergence of soft neurological signs (SNS).[5]

Soft neurological signs are defined as subtle abnormalities in motor, sensory, cognitive, and memory-related functions that cannot be attributed to an overt brain lesion. They are often undetectable during routine neurological examinations but can be revealed through careful and detailed assessment.[6] SNS are believed to reflect functional immaturity of specific brain regions, particularly the cerebellum, basal ganglia, and prefrontal cortex. Clinically, they are important because of their association with a range of cognitive and neuropsychiatric conditions, including

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attention deficits, poor academic performance, and autism spectrum disorders.[7-9]

The most comprehensive tool for assessing SNS in children is the Neurological Evaluation Scale (NES), developed by Buchanan and Heinrichs. This scale evaluates SNS across four main domains: sensory integration (SI), motor coordination (MC), complex sequential motor acts (SCMA), and other neurological findings.[10,11]

The present study aimed to investigate the presence of SNS in obese children using the NES and to compare the results with those of healthy controls. To the best of our knowledge, this is the first study to evaluate SNS in obese children using the NES.

MATERIALS and METHODS

Study patients

This prospective study was conducted between September 2021 and June 2022. A total of 61 obese children, aged 8 to 16 years, diagnosed in the general pediatrics outpatient clinic, and 57 age-and-sex matched healthy controls (HCs) were enrolled. SNS were assessed in all participants using the NES.

Children with a body mass index (BMI) at or above the 95th percentile for age and sex were classified as obese. Those with a BMI greater than 120% of the 95th percentile or ≥35 kg/m^2, whichever was lower, were classified as morbidly obese.[12]

Children with obesity secondary to genetic syndromes, monogenic or endocrine disorders, as well as those with diagnosed psychiatric conditions (in themselves or their parents), chronic diseases (e.g., cyanotic congenital heart disease, chronic lung disease, immunodeficiency, epilepsy, hypoxic-ischemic encephalopathy, intracranial lesions, neurodegenerative diseases) were excluded. Participants taking medications that could affect body weight or those who regularly engaged in sports activities were also excluded from the study.

Similarly, children with any neurological, psychiatric, or chronic diseases; those taking medications that could affect the central nervous system; or those who regularly participated in physical exercise were excluded from the HCs.

Measurements

Body weight was measured using a digital electronic scale (SECA®, Hamburg, Germany) with a sensitivity of 0.1 kg, and height was measured using a Harpenden stadiometer with a sensitivity of 0.1 cm. BMI was calculated as weight (kg) divided by height squared (m^2). Standard deviation scores (SDS) for weight, height, and BMI were calculated using the Child Metrics online calculator for pediatric endocrinologists, based on the reference data for the Turkish population published by Neyzi et al.[13,14]

Pubertal development was assessed according to the Tanner and Whitehouse staging system. Testicular volume ≥4 mL in males and breast development at stage ≥2 in females were considered indicative of pubertal onset.[15]

Blood samples were collected from obese participants after 10-12 hours of overnight fasting. Serum glucose, lipid profile, aspartate transaminase (AST), and alanine transaminase (ALT) levels were measured using a spectrophotometric method. Insulin concentrations were determined using a chemiluminescent microparticle immunoassay (Abbott Architect i2000SR). Complete blood count parameters were analyzed using an automated hematology analyzer (Mindray BC-6800, Shenzhen, China) based on hydrodynamic focusing flow cytometry.

Neurological evaluation scale (NES)

The NES was administered to all participants.[10] The scale consists of 26 items, one of which assesses hand preference and is not scored; therefore, the total score is calculated based on the remaining 25 items. Except for the sucking and snout reflexes—each scored as 0 (absent) or 2 (present)—all other items are rated on a 3-point scale: 0 (no impairment), 1 (mild but definite impairment), and 2 (marked impairment). Fourteen of the items were assessed bilaterally.

Validity and reliability

The scale used in this study did not require separate validity and reliability analyses, as it evaluates objective neurobiological parameters rather than subjective or culturally sensitive constructs. Furthermore, all items were administered and scored by the clinician according to standardized instructions, thereby minimizing inter-rater variability and eliminating participant bias.[16]

Ethics

Ethical approval for the study was obtained from the local ethics committee in accordance with the principles of the Declaration of Helsinki (Approval No: 2021/129). Written informed consent was obtained from all participants and their parents prior to enrollment.

Statistical method

The research data were analyzed using the IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). The normality of distribution for continuous variables was assessed using both visual methods (histograms and probability plots) and analytical tests (Kolmogorov-Smirnov and Shapiro-Wilk tests). Descriptive statistics for normally distributed variables were expressed as mean ± standard deviation, whereas non-normally distributed variables were presented as median (25^{th} –75^{th} percentile). Categorical variables were summarized as frequencies and percentages. The independence of categorical variables was tested using the chi-square test. Continuous variables with parametric distribution were compared between independent groups using the Student's t-test or one-way ANOVA. For non-parametric variables, the Mann-Whitney U test or Kruskal-Wallis test was applied. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1 compares the demographic characteristics and body measurements of obese children and healthy controls. A total of 118 participants were included in the study, of whom 37 (31.4%) were classified as obese, 24 (20.3%) as morbidly obese, and 57 (48.3%) as HCs. There were no statistically significant differences between the obese and HCs with respect to age or sex ($p > 0.05$). In contrast, height percentile and SDS, body weight percentile and SDS, and BMI, BMI percentile, and BMI SDS were significantly higher in obese children than in HCs ($p < 0.001$).

Table 2 compares the laboratory parameters of obese and morbidly obese children. The median neutrophil count and insulin level were significantly higher in the morbidly obese group ($p = 0.022$ and $p < 0.001$, respectively), while no significant differences were observed in the other laboratory parameters ($p > 0.05$).

Pairwise comparisons revealed that both obese and morbidly obese children had significantly higher total and subscale NES scores than HCs (all $p < 0.05$), whereas no significant differences were observed between the obese and morbidly obese groups ($p > 0.05$) (Table 3).

No significant differences in NES scores were observed based on sex, place of residence, family income level, or maternal education in either the obese or HCs ($p > 0.05$; data not shown).

Table 4 compares NES scores across age groups in obese children and HCs. No statistically significant differences in NES scores were observed among the age groups within the obese group. However, in the HC group, prepubertal children demonstrated significantly higher total NES, SCMA, and other subscale scores compared with those in the early adolescent period. Similarly, mid-adolescent children exhibited significantly higher total NES, SI, SCMA, and other subscale scores than early adolescents ($p < 0.005$).

DISCUSSION

To the best of our knowledge, this is the first study to investigate SNS in obese children through the NES. Our results revealed that obese children exhibited significantly higher total NES scores compared with HCs.

Obesity is known to promote an increase in inflammatory cells through cytokine activation.[17,18] Marginean et al. reported higher levels of leukocytes, lymphocytes, and platelets in obese children.[19] Similarly, Anık et al. found that leukocyte, neutrophil, and lymphocyte counts were significantly higher in obese children with insulin resistance compared to those without, and that insulin levels increased with the severity of obesity.[20] Consistent with these findings, our study demonstrated significantly higher neutrophil counts and insulin levels in morbidly obese children compared with both obese and HCs. These results support the presence of systemic inflammation in morbid obesity, which may contribute to the pathophysiology underlying increased SNS.

Yau et al. reported that as obesity severity increases, children exhibit poorer arithmetic and reading performance, shorter attention spans, and reduced overall cognitive functioning.[21] In addition, several studies have suggested that weight loss may improve obesity-related neurological impairments.[22,23] In our study, although higher NES scores were expected in morbidly obese children due to more severe and prolonged inflammation, no significant differences were observed between the obese and morbidly obese groups. This may be partly attributed to the limited sample size of the morbidly obese subgroup

Table 1. Comparison of demographic data and body measurements of obese children and healthy control

Parameter	Obese Children	Healthy Controls	p-value
	n=61	n=57	
Age (years), median (IQR)	13 (10-15)	13 (10-15)	0.648
Sex (n, %)			
Female	36 (59%)	30 (52.6%)	0.485
Male	25 (41%)	27 (47.4%)	
Height (cm), median (IQR)	158 (152-165)	156 (146-162)	0.104
Height p, median (IQR)	81.9 (61.8-97.6)	40 (24.2-69.2)	**<0.001**
Height SDS, median (IQR)	0.9 (0.3-2.2)	-0.2 [(-0.7)-0.5]	**<0.001**
Body Weight (kg), median (IQR)	76 (61-84)	48 (36.0-53.5)	**<0.001**
Body Weight p, median (IQR)	99.7 (98.6-100)	36.3 (20.6-64.4)	**<0.001**
Body Weight SDS, median (IQR)	2.7 (2.2-3.4)	-0.3 [(-0.7)-0.4]	**<0.001**
BMI (kg/m²), median (IQR)	29 (26.3-32)	19.5 (17.7-21.3)	**<0.001**
BMI p, median (IQR)	99.1 (97.9-99.7)	46.0 (21.8-68.1)	**<0.001**
BMI SDS, median (IQR)	2.4 (2.0-2.8)	-0.1 [(-0.7)-0.5]	**<0.001**
Birth Length (cm), median (IQR)	51 (50-52)	50 (50-52)	0.494
Birth Weight (kg), median (IQR)	3.4 (3.0-3.7)	3.2 (3.0-3.5)	0.276
Educational Status, median (IQR)			
Primary School	16 (26.2)	12 (21.1)	0.459
Middle School	18 (29.5)	23 (40.4)	
High School	27 (44.3)	22 (38.6)	
Screen Time (n, %)			
≤ 4 h	33 (54.1%)	35 (61.4%)	0.422
> 4 h	28 (45.9%)	22 (38.6%)	
Residence (n, %)			
Urban	45 (73.8%)	46 (80.7%)	0.370
Rural	16 (26.2%)	11 (19.3%)	
Income Level (n, %)			
Minimum wage or less	25 (41%)	16 (28.1%)	0.141
Above minimum wage	36 (59%)	41 (71.9%)	
Mother Educational Status (n, %)			
High School or less	58 (95.1%)	48 (84.2%)	0.051
Above High School	3 (4.9%)	9 (15.8%)	
Father Educational Status (n, %)			
High School or less	57 (93.4%)	37 (64.9%)	**<0.001**
Above High School	4 (6.6%)	20 (35.1%)	
Number of Siblings, median (IQR)	2 (1-3)	1 (1-2)	0.007
Mother's Age, median (IQR)	40 (35.0-45)	39 (36.0-42.0)	0.604
Father's Age, median (IQR)	43 (38.5-48)	43 (41.0-45.0)	0.894
Pubertal status (n, %)			
Prepubertal	11 (18%)	13 (22.8%)	0.520
Pubertal	50 (82%)	44 (77.2%)	

IQR, interquartile range; n, number; cm, centimeter; kg, kilogram; p, percentile; SDS, standard deviation score; m², square meter; BMI, body mass index. Data are presented as n (%) for categorical variables and as median (IQR) for continuous variables.

Table 2. Comparison of laboratory parameters in obese and morbidly obese patients

Parameter	Groups		p-value
	Obese children (n=37)	Morbidly Obese children (n=24)	
	Median (25-75 p)	Median (25-75 p)	
Hb (g/dL)	13.2 (12.6-13.8)	13.2 (12.4-13.7)	0.989
Leukocyte (10^3 /uL)	8050 (6950-9210)	9190 (7380-9860)	0.224
Neutrophil (mm^3)	3975 (3295-4665)	5220 (4010-5980)	0.022
Lymphocyte (mm^3)	2860 (2475-3545)	2930 (2665-3485)	0.958
PLT (10^9 /l)	361 (286-407)	327 (283-371)	0.174
Ferritin (ng/mL)	36.4 (15.2-51.1)	39.3 (21.3-52.2)	0.800
Vitamin B_{12} (pg/mL)	342 (235.5-402)	306 (231-344)	0.233
25-Hydroxy Vitamin D (ng/mL)	13.3 (10.9-21.3)	13.1 (9.7-17.9)	0.407
Glucose (mg/dL)	90 (86.0-94)	90 (85-95)	0.700
AST (U/L)	21 (16-27)	20.5 (16.5-27.5)	0.945
ALT (U/L)	19 (16-28)	21 (16-27)	0.552
Urea (mg/dL)	21.5 (17.5-24)	18 (15-22)	0.072
Creatinine (mg/dL)	0.6 (0.6-0.7)	0.6 (0.6-0.7)	0.320
Triglyceride (mg/dL)	112 (71.0-136)	99.5 (77-120.5)	0.466
Total Cholesterol (mg/dL)	159 (143.0-181)	159.5 (128-174.5)	0.338
HDL (mg/dL)	46.6 (41.9-53.3)	43.8 (37.3-53.1)	0.128
LDL (mg/dL)	82 (69.5-102)	92 (60-107)	0.853
HbA1c (%)	5.3 (5.1-5.4)	5.3 (5.2-5.8)	0.562
Insulin (µU/mL)	12.4 (8.5-16.1)	21.5 (15.2-25.5)	<0.001

IQR, interquartile range; Hb, hemoglobin; PLT, platelet; AST, aspartate aminotransferase; ALT, alanine aminotransferase; HDL, high-density lipoprotein; LDL, low-density lipoprotein; HbA1c, Hemoglobin A1c; g, gram; mg, milligram; mm^3, cubic millimeter; dL, deciliter; ng, nanogram; uL, microliter; L, liter; pg, pictogram; mL, milliliter. All data are presented as median (IQR).

Table 3. Comparison of neurological evaluation scale scores between healthy, obese and morbidly obese controls

Parameter	Healthy Children (1) (n=57)	Obese Children (2) (n=37)	Morbidly Obese Children (3) (n=24)	p-value
SI score	0 (0–1)	1 (0–2)	1 (0–2.5)	1–2: 0.005 ‖ 1–3: 0.003 † 2–3: 0.713 †
MC score	0 (0–0)	0 (0–1)	0 (0–2)	1–2: 0.009 ‖ 1–3: 0.001 † 2–3: 0.222 †
SCMA score	0 (0–1)	1 (0–2)	0 (0–1)	1–2: 0.004 ‖ 1–3: 0.401 † 2–3: 0.131 †
Other NES score	1 (0–2)	2 (1–4)	2 (0–3.5)	1–2: 0.002 ‖ 1–3: 0.097 † 2–3: 0.485 †
Total NES score	2 (0–4)	5 (2–8)	4.5 (1.5–8.5)	1–2:<0.001 ‖ 1–3:0.008 † 2–3: 0.853 †

QR, interquartile range; n, number; SI, sensory integration; MC, motor coordination; SCMA, sequencing of complex motor acts; NES, neurological evaluation scale. Values are presented as median (IQR). Pairwise comparisons: 1 = healthy controls, 2 = obese children, 3 = morbidly obese children. Tests: Wilcoxon signed-rank (‖, 1 vs 2); Mann–Whitney U (†, 1 vs 3; 2 vs 3). p<0.05 was considered statistically significant.

Table 4. Comparison of neurological evaluation scale scores by age group in obese and healthy controls

Parameter	Obese Children			p-value	Healthy Controls			p-value
Age Groups	Prepubertal (8–10 yr)	Early Adolescence (11–14 yr)	Middle Adolescence (15–16 yr)		Prepubertal (8–10 yr)	Early Adolescence (11–14 yr)	Middle Adolescence (15–16 yr)	
SI score [2]	2 (0–3)	1 (0–3)	1 (0–1)	0.243	1 (0–1)	0 (0–1)	0 (0–0)	0.040
MC score	1 (0–1)	0 (0–0.5)	0 (0–2)	0.220	0 (0–1)	0 (0–0)	0 (0–0)	0.133
SCMA score [1, 2]	1 (0–2)	1 (0–1)	0 (0–2)	0.509	1 (0–2)	0 (0–0)	0 (0–0)	<0.001
Other NES score [1, 2]	3 (1–4)	2 (0.5–4)	2 (1–3)	0.130	2 (1–3)	1 (0–1)	0 (0–0.5)	0.001
Total NES score [1, 2]	6 (3–9)	4.5 (2–8.5)	4 (1–6)	0.206	4 (3–6)	1 (0–3)	0 (0–1.5)	<0.001

Abbreviations: IQR, interquartile range; SI; sensory integration; MC, motor coordination; SCMA,sequencing of complex motor acts;NES,neurological evaluation scale.

[1] In the healthy group, there is a statistically significant difference between the prepubertal group and the early adolescent group.

[2] In the healthy group, there is a statistically significant difference between the prepubertal group and the mid-adolescent group.

and, more importantly, to the absence of data regarding obesity duration. The chronicity of inflammation may play a critical role in the extent of neurological dysfunction. These findings indicate that obesity, even at moderate levels, is associated with higher NES scores compared to healthy peers, while further increases in BMI do not appear to result in additional neurological impairment. Future studies should incorporate obesity duration as a variable to better elucidate this relationship.

Previous studies have reported the presence of SNS in healthy individuals.[24-27] A longitudinal study of 48 healthy boys demonstrated that SNS increased over one year among those who initially exhibited such signs.[28] In our study, SNS were detected in both obese and healthy children; however, NES scores were significantly higher in the obese group. Beyond inflammation, physical inactivity and consequent immobility in obese children may also have contributed to these findings. Physical activity is known to exert neuroprotective effects by enhancing neuroplasticity, motor coordination, and cognitive performance.[29] In our study, children who regularly engaged in physical exercise were excluded to minimize potential confounding; however, objective measures of physical activity were not recorded. Therefore, the lack of quantitative assessment of physical activity represents a limitation, and future studies should incorporate comprehensive evaluations to better elucidate its role.

Several studies have suggested that SNS tend to decrease with age. For instance, Martins et al. reported a decline in SNS scores among healthy children over a five-year period. Their study found higher SNS scores in boys aged 11–14 years, with this sex difference diminishing between 15 and 17 years of age, which was interpreted as reflecting neurodevelopmental maturation.[27] Similarly, Patankar et al.[30] and Ardila et al.[31] also observed age-related reductions in SNS. A separate study involving 101 children aged 4–11 years demonstrated a comparable trend.[27] In our study, within the healthy group, children aged 8–11 years exhibited significantly higher total NES scores, as well as higher scores in the SCMA and other subscales, compared with older age groups (11-14 and 15-16 years). Conversely, no significant age-related differences in NES scores were observed among obese children. These findings support the notion that SNS decline with age in healthy children as a consequence of brain maturation[32], whereas in obese children, this developmental trajectory may be disrupted by persistent chronic inflammation affecting all age groups.

This study has several limitations. First, the relatively small number of morbidly obese children reduces statistical power for subgroup analyses. Second, the cross-sectional design precludes causal inference and evaluation of temporal changes in SNS. Third, the absence of data on obesity duration limits the understanding of cumulative inflammatory exposure. Fourth, although children who regularly engaged in physical exercise were excluded, objective quantification of physical activity was not performed. Finally, as this was a single-center study, the generalizability of the findings may be limited.

Despite these limitations, the study has several notable strengths. These include its prospective design, the

objective and standardized assessment of SNS using the NES, and the exclusion of potential confounding effects of regular physical activity. Moreover, this is among the first studies to investigate SNS in obese children using a validated neurological assessment tool.

CONCLUSION

In conclusion, childhood obesity may influence neurodevelopmental processes and be associated with SNS. The underlying mechanisms linking obesity to these findings, as well as their potential long-term implications, remain to be clarified. Future studies with larger cohorts and longitudinal designs are needed to better elucidate these relationships. Such research could facilitate earlier identification and prevention strategies aimed at supporting healthy neurodevelopment in obese children.

Author contributions

Conception and design: T.D., E.C.; Data acquisition: T.D.; Data analysis: T.D., E.C.; Data interpretation: T.D., E.C.; Drafting of the manuscript: T.D., E.C., A.A., A.T. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

This study was approved by the Aydın Adnan Menderes University Faculty of Medicine Ethics Committee (Decision/ Protocol No: 2021/129). Informed consent was obtained from all participants involved in this study.

Conflict of interest

The authors declare that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this study.

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