ORIGINAL ARTICLE ÖZGÜN ARAŞTIRMA

Systemic Inflammatory Indices (SII and SIRI) in Obese Pediatric Patients: A Retrospective Study

Obez Çocuk Hastalarda Sistemik Enflamasyon Göstergeleri: SII ve SIRI İndekslerinin Retrospektif Değerlendirmesi

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Abstract

Introduction: Childhood obesity is associated with systemic inflammation and metabolic disturbances. This study aimed to investigate the relationships between obesity, systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI) in children and adolescents.

Materials and Methods: A total of 217 children (126 controls, 91 obese; mean age 12.2 ± 3.4 years) were included. Anthropometric measurements, laboratory parameters, and inflammatory indices (SII, SIRI) were assessed. Multiple linear regression analyses were performed to examine associations between BMI SD, age, gender, pubertal status, and inflammatory indices.

Results: Obese children had significantly higher weight SD, height SD, BMI SD, NEU, MPV, glucose, total cholesterol, LDL-C, TG, ALT, GGT, SII, and SIRI compared to controls ($p<0.05$), while HDL-C was lower ($p=0.041$). Regression analyses showed that BMI SD was a significant positive predictor of both SII and SIRI ($p<0.001$), whereas age, gender, and pubertal status were not significant. The models explained 8.6% and 7.2% of the variance in SII and SIRI, respectively.

Conclusion: Our findings indicate that childhood obesity is associated with elevated systemic inflammatory markers and that BMI SD is a strong predictor of systemic inflammation. These results highlight the importance of early weight management to reduce inflammatory risk in children and adolescents.

Keywords
Childhood obesity, NLR (Neutrophil-to-lymphocyte ratio), PLR (Platelet-to-lymphocyte ratio) MPV (min platelet volume), puberty SII (Systemic immune inflammatory index), SIRI (Systemic Inflammatory Response Index)

Öz

Giriş: Çocukluk çağında obezite, sistemik inflamasyon ve metabolik bozukluklarla ilişkilidir. Bu çalışmada, çocuk ve ergenlerde obezite ile sistemik immün-inflamasyon indeksi (SII) ve sistemik inflamasyon yanıt indeksi (SIRI) arasındaki ilişkiler araştırılmıştır.

Gereç ve Yöntem: Çalışmaya toplam 217 çocuk (126 kontrol, 91 obez) dahil edildi; yaş ortalaması 12,2 ± 3,4 yıl idi. Antropometrik ölçümler, laboratuvar parametreleri ve inflamasyon indeksleri (SII, SIRI) değerlendirildi. Çoklu doğrusal regresyon analizleri ile BMI SD, yaş, cinsiyet ve pubertal durum ile inflamasyon indeksleri arasındaki ilişkiler incelendi.

Bulgular: Obez çocuklarda, kontrol grubuna kıyasla ağırlık SD, boy SD, BMI SD, NEU, MPV, glukoz, total kolesterol, LDL-C, TG, ALT, GGT, SII ve SIRI seviyeleri anlamlı olarak yüksekti ($p<0,05$); HDL-C seviyeleri ise düşüktü ($p=0,041$). Regresyon analizleri, BMI SD'nin hem SII hem de SIRI için anlamlı pozitif bir belirleyici olduğunu gösterdi ($p<0,001$), yaş, cinsiyet ve pubertal durum ise anlamlı etkiler göstermedi. Modeller sırasıyla SII ve SIRI varyansının %8,6 ve %7,2'sini açıkladı.

Anahtar kelimeler
Çocukluk çağı obezitesi, NLR (Nötrofil-lenfosit oranı), PLR (Trombosit-lenfosit oranı), MPV (Ortalama trombosit hacmi), puberte, SII (Sistemik immun-inflamatuar indeks), SIRI (Sistemik inflamatuar yanıt indeksi)

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Sonuç: Çalışmamız, çocukluk çağında obezitenin sistemik inflamasyon ile ilişkili olduğunu ve BMI SD'nin inflamasyonun güçlü bir belirleyicisi olduğunu göstermektedir. Bulgular, erken dönemde kilo yönetimi ve inflamatuvar riskin azaltılmasının önemini vurgulamaktadır.

Introduction

Childhood obesity is one of the most common chronic health problems today. According to the 2013 Turkey Demographic and Health Survey (TNSA), the prevalence of overweight or obesity among children under the age of five is 10.9% (1). The results of the 2009 Study on Monitoring Growth in School-Age Children in Turkey (TOÇBİ) indicate that among children aged 6–10, 14.3% are overweight and 6.5% are obese. The obesity rate is 7.5% in boys and 5.4% in girls, while the rate of being overweight is 15.1% in boys and 13.5% in girls (2).

Furthermore, according to the 2009–2010 Health Behaviour in School-Aged Children (HBSC) survey, 7% of 11-year-old girls and 16% of boys are classified as overweight or obese. Among 13-year-olds, the rates are 10% for girls and 18% for boys, while in the 15-year age group, 6% of girls and 17% of boys are considered overweight or obese (3).

It has become clear that obesity is not only a condition related to energy imbalance, but is also associated with chronic low-grade systemic inflammation. Adipose tissue synthesizes and secretes a large number of hormones, cytokines, extracellular matrix proteins, growth factors and vasoactive factors that we call adipokines. These cytokines and adipokines induce leukocytosis in obese individuals by increasing neutrophils (NEU) in the intravascular space and accelerating bone marrow production (4).

Hematologic parameters such as white blood cell (WBC) count, NEU and lymphocyte counts (LYM), NEU to LYM ratio (NLR), platelet count (PLT), PLT to LYM ratio (PLR), and mean platelet volume (MPV) have been suggested to be associated with systemic inflammation and increased cardiometabolic risk (5).

WBC, NEU and LYM, along with NLR, may serve as markers of systemic inflammation. NLR has recently emerged as a potential prognostic indicator and may be associated with carotid intima-media thickness in children aged 7 to 9 years (6).

MPV is a marker of PLT function. MPV greater than normal (above 10 femtoliters) indicates that PLT WBC are more active and is associated with an increased risk of cardiovascular disease (7).

Systemic Immune-Inflammation Index (SII) and Systemic Inflammation Response Index (SIRI), which include four basic hematologic cell types (NEU, Monocyte (MON), LYM and PLT), are the next generation of objective inflammation indicators developed based on peripheral blood parameters, which can provide more clinical information and have practical use. They have been reported to be associated with the prognosis and development of diseases that are positively associated with coronary artery stenosis, cancer and obesity in adult studies (8-10). Although the prognostic and diagnostic value of these indices in various diseases in the adult population has been investigated, their role in childhood obesity is still unclear (8).

Therefore, the aim of this retrospective case-control study is to evaluate the utility of SII and SIRI indices as markers of systemic inflammation in obese pediatric patients.

Materials and Methods

Study Sample

This retrospective case-control study included data from 91 obese patients aged 5 to 18 years who were admitted to the Pediatric Endocrinology Outpatient Clinic of Kayseri City Training and Research Hospital between April and December 2023 due to excess weight. All patients had a body mass index (BMI)-for-age > +2 SD (Standart deviation score), were diagnosed with simple (primary) obesity, and had no chronic diseases.

As the control group, 126 non-obese subjects aged 5 to 18 years without chronic disease were randomly selected from patients attending the same clinic.

Exclusion criteria: Malignancies, infection, bone marrow suppressive disorder, malabsorptive disorder, medication use, history of lung, liver or kidney disease, including any inflammatory disorder; c-reactive protein more than 10 mg/L, WBC count less than $4x10^3$ µ/ml or more than $13.5x10^3$ µ/ml, PLT less than $150x10^3$ µ/ml or more than $450x10^3$ µ/ml and anemia.

Age, gender, anthropometric measurements, puberty, complete blood count and biochemical test results were analyzed.

Body weights and heights were measured with Seca digital scale and Harpenden Stadiometer during the initial examination. Height, weight and BMI SD were evaluated according to age and gender appropriate charts. A BMI of over 2 SD according to age and gender was considered obesity (11). Puberty status of the patients was determined

according to Tanner Marshall pubertal staging system. Stage 1 was considered prepubertal according to Tanner Marshall staging (12).

Complete blood count, blood glucose, Gamma Glutamyl Transferase (GGT, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), Total cholesterol, triglyceride (TG)), liver function tests (alanine aminotransferase (ALT), aspartate aminotransferase (AST)) were evaluated after at least 10 hours of fasting. Venous samples for complete blood count were collected in potassium EDTA tubes and analyzed within 2 hours. Blood cell count was performed with an automated cell counter (LH 780, Beckman Coulter, Krefeld, Germany) using the impedance method. LYM, NEU and PLT (expressed as $\times 10^3$ cells/µl) were measured using automated hematology analyzers. Index calculations:

Neutrophil-Lymphocyte Ratio (NLR): NEU/LYM

Platelet-Lymphocyte Ratio (PLR): PLT /LYM

Systemic Immune-inflammation Index (SII): (PLT × NEU) / LYM (9).

Systemic Inflammatory Response Index (SIRI): (NEU × MON) /LYM

Statistical Analysis

Mean, standard deviation, median, minimum-maximum values, ratio, frequency values were used in descriptive statistics of the data. Qualitative data were analyzed with chi-square test. The distribution of the data was analyzed by Kolmogorov Simirnov test. Mann-Whitney U test was used to compare groups for variables that did not show normal distribution; Student t test was used to compare groups for variables that showed normal distribution. Spearman correlation analysis was used to determine the relationship between variables that did not show normal distribution, and Pearson correlation analysis was used for variables with normal distribution. A value of $p<0.05$ was considered statistically significant.

In the study, multiple lineer regression analysis was performed to evaulate the effects of SII and SIRI indices on the other variables. Because SII and SIRI showed right-skewed distributions, $\log_{10}$-transformed values (log-SII, log-SIRI) were additionally used for regression analyse.

A priori power analysis was conducted assuming a moderate effect size (Cohen's d = 0.5), a significance level of 0.05, and desired power of 0.95. The calculated sample size per group to detect differences in SII and SIRI was approximately 88 participants. Given that the study included 126 controls and 91 obese participants, the sample size was sufficient to detect moderate effect sizes with high power.

Approval for this study was obtained from the Kayseri City Hospital Clinical Research Ethics Committee of decision number: 1009, date: 12.12.2023).

Results

General Characteristics of the Study Groups

A total of 217 children were included in the study, comprising 126 in the control group and 91 in the obese group. The mean age was 12.2 ± 3.4 years. There were no statistically significant differences between the groups in terms of age and gender distribution ($p>0.05$). Weight SD, height SD, and BMI SD were significantly higher in the obese group compared to the control group (all $p<0.001$) (Table 1).

Comparison of inflammatory and metabolic parameters between obese and control groups

Among laboratory parameters, NEU, MPV, glucose, total cholesterol, LDL-C, TG, ALT, GGT, SII, and SIRI levels were significantly higher in the obese group ($p<0.05$). HDL-C levels were significantly lower in the obese group compared to the control group ($p=0.041$). No significant differences were observed between the groups regarding LEU, MON, PLT, hemoglobin, AST, NLR, and PLR levels ($p>0.05$) (Table 1).

In terms of inflammatory indices, both SII and SIRI were significantly elevated in obese children. Mean SII was 528.5 ± 194.3 in the obese group versus 448.1 ± 209.6 in controls ($p=0.008$), and median SIRI was 1.00 (0.70–1.43) in obese subjects and 0.79 (0.49–1.10) in controls ($p<0.001$ (Table 1).

Comparision by Pubertal Status

In the pubertal group, obese children had significantly higher weight SD, height SD, BMI SD, NEU and erythrocyte counts, glucose, total cholesterol, HDL-C, LDL-C, TG, ALT, GGT, and SIRI levels compared to controls ($p<0.05$) . Pubertal sub-analysis results are summarized in Supplementary Table 1.

Association Between BMI-SD and Inflammatory Indices: Regression Analysis

Multiple linear regression analyses were conducted to examine the relationship between SIRI and SII as dependent variables and several predictors, including BMI SD, age, gender, and pubertal status. The models explained 8.6% of the variance in SII ($R^2 = 0.086$, Adjusted $R^2 = 0.066$) and 7.2% of the variance in SIRI ($R^2 = 0.072$, Adjusted $R^2 = 0.052$). Among the predictors, BMI SD was a significant positive predictor for both SII and SIRI ($p <0.001$), indicating that for each one-unit increase in BMI SD, SII increased by 38.16

units and SIRI increased by 0.103 units. Age, gender, and pubertal status showed no significant effects in either model. Regression assumptions, including multicollinearity and independence of residuals, were satisfied (VIF <2; Durbin–Watson ≈ 2) (Tables 2 and 3).

Table 1. General characteristics and laboratory findings of obese and control subjects

	Control group (n:126)	Obese group (n:91)	p value
Age years*	11.93 (8.64-15.44)	13 (10.12-15.24)	0.142
Gender male*	44 (34%)	41 (45%)	0.131
Weight SD	-0.05 ± 1.32	2.66 ±0.93	<0.001
Height SD	-0.27 ± 1.41	0.53 ± 1.39	<0.001
BMI SD kg/m^{2}*	0.26 (-0.67-1.21)	2.43 (2.14-2.85)	<0.001
NEU 10^{3}/µ/l*	3.83 (2.57-4.77)	4.57 (3.59- 5.86)	<0.001
LYM 10^{3}/µ/l*	2.88 (2.25-3.32)	2.85 (2.54-3.30)	0.349
Hb g/dl	13 (12-14)	13.7 (13-14)	0.060
Erythrocytes 10^{6}/L	4.96 ± 0.39	5.17 ± 0.42	<0.001
MON 10^{3}/L*	0.55 (0.45-0.67)	0.62(0.51-0.73)	0.066
PLT 10^{3}/µ/l*	315 (263-346)	327 (286-366.5)	0.064
MPV fl*	9.6 (9-10)	9.8 (9.2-10)	0.009
Glucose mg/dl*	84 (78.7-87)	87(82-92.75)	0.001
Total-C mg/dl	133.56 ±18.72	156.01 ±30.21	<0.001
HDL-C mg/dl	49.4 ± 9.68	44.86 ± 8.87	0.041
LDL-C mg/dl	80.12 ±15.59	96.23 ± 25.67	0.001
TG mg/dl*	76 (64.75-92.75)	102 (76-163)	0.001
AST U/L*	23 (18-26.5)	21 (17-27)	0.607
ALT U/L*	14 (11-17)	20 (14-27)	<0.001
GGT U/L*	12 (9-14)	17 (13-24)	<0.001
NLR*	1.36 (0.97-1.98)	1.56 (1.14-2.04)	0.063
PLR*	108 (90-144.5)	111.5 (93.25-128.75)	0.680
SII, 10^{3}/µ/l	448.07 ± 209.61	528.54 ± 194.26	0.008
Log SII	5.98 ±0.46	6.20 ± 0.37	0.002
SIRI, 10^{3}/µ/l*	0.79 (0.49-1.10)	1.00 (0.70-1.43)	<0.001
Log SIRI	-0.32 ± 0.58	-0.01 ± 0.56	<0.001

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, BMI: Body mass index, GGT: Gamma glutamyl transferase, Hb: Hemoglobi, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, LYM: Lymphocyte, MON: Monocyte, MPV: Mean platelet volume, NEU: Neutrophil, NLR: Neutrophil/lymphocyte ratio, N: Number, PLT: Platelet, PLR: Platelet/lymphocyte ratio, SD: Standard deviation score, SII: Systemic immune-inflammatory index, SIRI: Systemic inflammatory response index, TG: Triglyceride, Total-C: Total-cholesterol, bold represents statistically significant values (p<0.05). Data are presented as mean ± standard deviation for normally distributed variables and median (interquartile range) for non-normally distributed variables. Student's t-test was used to compare groups for variables with normal distribution. *Mann-Whitney U test was applied. **Chi-square test was applied

Table 2. Multiple linear regression analysis predicting SII

Variable	B	Std. Error	Beta	t	VIF	95% CI for B	p
Constant	464.78	55.81	—	8.33	—	354.6-574.88	<0.001
BMI SD	38.16	10.30	0.272	3.71	1.08	17.84- 58.47	<0.001
Age	-1.98	5.09	-0.033	-0.39	1.49	-12.02- 8.07	0.698
Gender	-49.19	30.82	-0.116	-1.60	1.05	-110.00- 11.62	0.112
Puberty	19.38	45.71	0.037	0.42	1.56	-7 0.80- 109.56	0.672

CI: Confidence Interval. Note: Gender coded as 0 = Male, 1 = Female. Pubertal stage indicates whether puberty has started (1 = Yes, 0 = No). Model summary: R = 0.294, R^2 = 0.086, Adjusted R^2 = 0.066, Standard Error = 199.55, Durbin-Watson = 1.49. No multicollinearity detected (VIF<2)

Table 3. Multiple linear regression analysis predicting SIRI

Variable	B	Std. Error	Beta	t	VIF		95% CI for B	p
Constant	0.879	0.175	—	5.02	—		0.533-1.224	<0.001
BMI SD	0.103	0.032	0.235	3.25	1.08		0.041-0.166	0.001
Age	-0.014	0.016	-0.074	-0.87	1.50		-0.046-0.018	0.388
Gender	-0.017	0.095	-0.012	-0.17	1.05		-0.200-0.172	0.863
Puberty	0.198	0.141	0.122	1.40	1.56		-0.081-0.477	0.163

CI: Confidence Interval. Note: Gender coded as 0 = Male, 1 = Female. Pubertal stage indicates whether puberty has started (1 = Yes, 0 = No). Model summary: R = 0.268, R^2 = 0.072, Adjusted R^2 = 0.052, Standard Error = 0.639, Durbin-Watson = 1.79. No multicollinearity detected (VIF<2).

Discussion

In this study, obese children and adolescents were found to differ from their normal-weight peers in terms of hematological parameters and inflammation markers. Overall, the obese group showed significantly higher levels of NEU, MPV, glucose, total cholesterol, LDL-C, TG, ALT, GGT, SII, and SIRI. These findings support the association between obesity and systemic inflammation as well as metabolic disorders.

Adipose tissue functions as an inflammatory organ by continuously secreting various cytokines, adipokines, and immune cells into the systemic circulation. Studies have shown that these bioactive substances are valuable indicators for diagnosing and monitoring systemic inflammation. In chronic inflammation, increases in NEU and PLT and decreases in LYM are often observed. Especially, NLR and PLR are inexpensive and accessible markers that reflect the clinical manifestations of metabolic inflammation (13). In our study, obese children and adolescents were found to have significantly higher NEU, MPV, SII, and SIRI compared to their normal-weight peers. These findings support the close association between obesity and inflammatory processes, highlighting the systemic inflammation originating from adipose tissue and its clinical reflections. Interestingly, parameters such as LEU, MON, PLT, hemoglobin, AST, NLR, and PLR did not show significant differences between the groups. This may suggest that while some classic inflammatory markers remain within normal ranges in early or moderate stages of obesity, composite indices like SII and SIRI may offer greater sensitivity in detecting subclinical inflammation. Additionally, the elevated MPV values in the obese group could reflect enhanced platelet activation, which has been linked to endothelial dysfunction and cardiovascular risk in obese children (14).

Increased leptin in obesity may increase the number of circulating NEU by triggering NEU chemotaxis and activation. Furthermore, obesity-induced hypoxia is another factor that increases NEU infiltration in adipose tissue. Several studies in recent years have found a positive correlation between BMI and NEU. Increased NLR is used as an indicator of inflammatory status in obese individuals and is associated with cardiometabolic risk factors (15,16). On the other hand, there are studies reporting that no difference was found in obese and normal weight children and that it cannot be considered as a marker of metabolic syndrome compared to adults (4,17). In the study, NEU and NLR were higher in obese patients but no difference was found in NLR when patients were classified according to puberty. While erythrocytes increase with increased erythropoietin due to hypoxia in obese patients, anemia due to chronic inflammation is also observed (18). Increased erythrocyte in obese people has been associated with increased BMI, diabetes risk, and central obesity (19). Similarly, in our study, erythrocyte count was found to be high in obese pubertal subjects.

Obesity has been associated with thrombocytosis and thrombosis, while hyperinsulinemia, hypertriglyceridemia, and hypertension are independently associated with thrombosis, regardless of obesity. In addition, increased leptin levels in obesity may increase the risk of PLT aggregation and cardiovascular disease (20). In the prepubertal group, higher PLT counts were observed, highlighting the need for further studies on this topic. MPV is a common indicator of PLT activity. Increased MPV has been associated with increased waist circumference, increased risk of diabetes (21,22). In our study, MPV was found to be higher in obese subjects, both in the pubertal group and in the prepubertal group.

In pubertal children, the metabolic and inflammatory burden of obesity appears to intensify. Obese individuals in this group showed significantly higher levels of erythrocyte counts, glucose, total cholesterol, LDL-C, HDL-C, triglycerides, ALT, GGT, Log SII and Log SIRI than controls. These alterations reflect the compounded effects of hormonal changes during

puberty on obesity-related pathophysiology. The decrease in HDL-C and increase in triglycerides and liver enzymes are particularly concerning due to their association with future cardiovascular risk. Overall, the findings indicate that while obesity-related complications begin in the prepubertal period, they become more pronounced and widespread during puberty, emphasizing the critical importance of age- and stage-specific interventions for effective obesity management (23).

In our study, children with and without puberty were compared. Adolescence is a complex developmental stage in which sex steroids and metabolic processes change significantly in the transition from childhood to adulthood. Physiologic insulin resistance, fluctuations in growth hormone and increased sex steroids, coupled with obesity, can have a synergistic effect on inflammation. Increased growth rate and cellular renewal processes can also increase the sensitivity of the immune system (23).

SII quantifies the relationship between PLT, NEU and LYM. It is a relatively stable marker despite physiological conditions. SII has been found to be associated with other inflammatory diseases, including heart failure, rheumatoid arthritis, acute pulmonary embolism and chronic kidney disease (24). SII has a role as an additional measure of cardiometabolic instability in predicting metabolic syndrome in obese children (25). In the study by Zhang et al. (24), although SII was found to be high in obese children, there was no linear relationship between SII and obesity. Increased SII levels were positively correlated with obesity up to a certain threshold (threshold log SII: 6.410), but negatively correlated with obesity after crossing the threshold. Increased peripheral proinflammatory cytokines in systemic inflammation may lead to weight loss due to increased energy expenditure, lipolysis, weight loss and decreased appetite due to the inflammatory environment around the vagus (24). In our study, Log SII was found to be higher in the both pubertal and prepubertal obese groups.

SIRI is a new index used to reflect systemic inflammation and immune response. SIRI has been used in past studies to predict and assess the prognosis of diseases such as pneumonia, rheumatoid arthritis and acute pancreatitis. Inflammation is directly associated with insulin resistance and chronic metabolic disorders as well as increased cardiovascular risk (26). In our study, it was observed to increase in obese children.

Multiple linear regression analyses in our study revealed significant positive associations between BMI SD and both the SII and SIRI. In contrast, age, sex, and pubertal status did not have significant effects in either model. These findings indicate that, as reported in the literature, increased body weight in children and adolescents is associated with a marked impact on systemic inflammation (27).

In contrast to several previous studies that have reported significant associations between SII and SIRI with metabolic parameters such as glucose levels, lipid profiles, and liver enzymes, our findings did not reveal such correlations in the pediatric population studied (8,24,28). This discrepancy may be attributed to differences in study populations, particularly age, pubertal status, and the pathophysiological progression of obesity-related complications in children versus adults.

Study Limitations

The limitations of my study include the limited number of patients. Confounding factors such as screen time, activity status, parental weight and socioeconomic status, which have a role in the development of obesity in children, were not included in the study. The volume of PLT distribution may vary, especially in samples taken in EDTA tubes (29). The strength of the study is that a wide age range was included and the cases were classified according to puberty.

Conclusion

In conclusion, NEU, erythrocytes and MPV were increased in obese children compared to their healthy peers. SIRI index was higher in adolescent obese children compared to their peers. Obesity in adolescence, in combination with hormonal and metabolic changes, triggers inflammatory processes and poses a significant threat in terms of long-term cardiometabolic risks. Indices such as SII and SIRI can be used as valuable biomarkers for monitoring this inflammatory response and early intervention. Therefore, monitoring the level of inflammation should be addressed with multidisciplinary approaches as well as early diagnosis and treatment of obesity in adolescence.

Ethics

Ethical Approval: Approval for this study was obtained from the Kayseri City Hospital Clinical Research Ethics Committee of decision number: 1009, date: 12.12.2023).

Footnotes

Conflict of Interest: No conflict of interest was declared by the authors.

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References

1. Hacettepe Üniversitesi Nüfus Etütleri Enstitüsü. (2014). 2013 Türkiye nüfus ve sağlık araştırması (TNSA). Ankara: HÜNEE, T.C. Kalkınma Bakanlığı ve TÜBİTAK.
2. T.C. Sağlık Bakanlığı. (2011). Türkiye okul çağı çocuklarında (6-10 yaş grubu) büyümenin izlenmesi projesi (TOÇBİ) 2009 yılı araştırma sonuçları. Ankara: T.C. Sağlık Bakanlığı Temel Sağlık Hizmetleri Genel Müdürlüğü.
3. Currie, C., and World Health Organization. health behaviour in school-aged children (HBSC) study: international report from the 2001/2002 survey. (2004).
4. Mărginean CO, Meliţ LE, Ghiga DV, Mărginean MO. Early inflammatory status related to pediatric obesity. Front Pediatr. 2019;7:241.
5. Aypak C, Türedi O, Bircan MA, Yüce A. Could mean platelet volume among complete blood count parameters be a surrogate marker of metabolic syndrome in pre-pubertal children? Platelets. 2014;25:393-8.
6. Prats-Puig A, Gispert-Saüch M, Díaz-Roldán F, et al. Neutrophil-to-lymphocyte ratio: an inflammation marker related to cardiovascular risk in children. Thromb Haemost. 2015;114:727-34.
7. Nkambule BB, Mxinwa V, Nyambuya TM, Dludla PV. The mean platelet volume and atherosclerotic cardiovascular-risk factors in adults with obesity: a systematic review and meta-analysis of observational studies. BMC Nutr. 2022;8:47.
8. Zhou Y, Wang Y, Wu T, Zhang A, Li Y. Association between obesity and systemic immune inflammation index, systemic inflammation response index among US adults: a population-based analysis. Lipids Health Dis. 2024;23:245.
9. Hu B, Yang XR, Xu Y, Sun YF, Sun C, Guo W, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. Clin Cancer Res. 2014;20:6212-22.
10. Ma J, Li K. Systemic immune-inflammation index is associated with coronary heart disease: a cross-sectional study of NHANES 2009-2018. Front Cardiovasc Med. 2023;10:1199433.
11. Neyzi O, Gunoz H, Furman A, Bundak R, Gokcay G. Türk çocuklarında vücut ağırlığı, boy uzunluğu, baş çevresi ve vücut kitle indeksi referans değerleri. Çocuk Sağlığı ve Hastalıkları Dergisi. 2008.
12. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child. 1969;44:291-303.
13. Ren Y, Zhao H, Yin C, Lan X, Wu L, Du X, et al. Adipokines, hepatokines and myokines: focus on their role and molecular mechanisms in adipose tissue ınflammation. Front Endocrinol (Lausanne). 2022;13:873699.
14. Coban E, Ozdogan M, Yazicioglu G, Akcit F. The mean platelet volume in patients with obesity. Int J Clin Pract. 2005;59:981-2.
15. Gummlich L. Obesity-induced neutrophil reprogramming. Nat Rev Cancer. 2021;21:412.
16. Gomez-Casado G, Jimenez-Gonzalez A, Rodriguez-Muñoz A, Tinahones FJ, González-Mesa E, Murri M, et al. Neutrophils as indicators of obesity-associated inflammation: a systematic review and meta-analysis. Obes Rev. 2025;26:e13868.
17. Marra A, Bondesan A, Caroli D, Grugni G, Sartorio A. The neutrophil to lymphocyte ratio (NLR) positively correlates with the presence and severity of metabolic syndrome in obese adults, but not in obese children/adolescents. BMC Endocr Disord. 2023;23:121.
18. Zhao L, Zhang X, Shen Y, Fang X, Wang Y, Wang F. Obesity and iron deficiency: a quantitative meta-analysis. Obes Rev. 2015;16:1081-93.
19. Christakoudi S, Tsilidis KK, Evangelou E, Riboli E. Associations of obesity and body shape with erythrocyte and reticulocyte parameters in the UK Biobank cohort. BMC Endocr Disord. 2023;23:161.
20. Rafail S, Ritis K, Schaefer K, Kourtzelis I, Speletas M, Doumas M, et al. Leptin induces the expression of functional tissue factor in human neutrophils and peripheral blood mononuclear cells through JAK2-dependent mechanisms and TNFalpha involvement. Thromb Res. 2008;122:366-75.
21. Zhou Z, Chen H, Sun M, Ju H. Mean Platelet volume and gestational diabetes mellitus: a systematic review and meta-analysis. J Diabetes Res. 2018;2018:1985026.
22. Furman-Niedziejko A, Rostoff P, Rychlak R, Golinska-Grzybala K, Wilczynska-Golonka M, Golonka M, et al. Relationship between abdominal obesity, platelet blood count and mean platelet volume in patients with metabolic syndrome. Folia Med Cracov. 2014;54:55-64.
23. Montero D, Walther G, Perez-Martin A, Roche E, Vinet A. Endothelial dysfunction, inflammation, and oxidative stress in obese children and adolescents: markers and effect of lifestyle intervention. Obes Rev. 2012;13:441-55.
24. Zhang YZ, Ma RW, Bhandari S, Xie J, Zhang XY, Xie C, et al. Association between systemic immune inflammation index and adolescent obesity in a cross-sectional analysis. Sci Rep. 2025;15:6439.
25. Nicoară DM, Munteanu AI, Scutca AC, Mang N, Juganaru I, Brad GF, et al. Assessing the relationship between systemic immune-inflammation index and metabolic syndrome in children with obesity. Int J Mol Sci. 2023;24:8414.
26. Wang P, Guo X, Zhou Y, Li Z, Yu S, Sun Y, et al. Monocyte-to-high-density lipoprotein ratio and systemic inflammation response index are associated with the risk of metabolic disorders and cardiovascular diseases in general rural population. Front Endocrinol (Lausanne). 2022;13:944991.
27. Su Z, Cao L, Chen H, Zhang P, Wu C, Lu J, et al. Obesity indicators mediate the association between the aggregate index of systemic inflammation (AISI) and type 2 diabetes mellitus (T2DM). Lipids Health Dis. 2025;24:176.
28. Xie R, Xiao M, Li L, Ma N, Liu M, Huang X, et al. Association between SII and hepatic steatosis and liver fibrosis: A population-based study. Front Immunol. 2022;13:925690.
29. J Jackson SR, Carter JM. Platelet volume: laboratory measurement and clinical application. Blood Rev. 1993;7:104-13.