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The role of fetuin-A in adolescent obesity: Association with anthropometric and metabolic parameters

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ABSTRACT

Objective: Childhood obesity is a growing global health problem linked to chronic inflammation and metabolic complications, including dyslipidemia, insulin resistance, type 2 diabetes mellitus, nonalcoholic fatty liver disease, and hypertension. Fetuin-A, a hepatokine involved in insulin signaling and inflammation, has been studied in adults, but its role in pediatric obesity is unclear. This study evaluated fetuin-A levels in obese adolescents and their association with anthropometric and metabolic parameters.

Materials and Methods: This cross-sectional study included 40 obese adolescents (BMI > 95th percentile) and 30 healthy controls (BMI between the 15th and 85th percentiles) who had no additional systemic diseases and were not receiving any medication. Anthropometric measurements and fasting glucose, insulin, high-sensitivity C-reactive protein, and fetuin-A were assessed. The presence of dyslipidemia, hepatic steatosis, and hypertension was evaluated.

Results: Hs-CRP levels were significantly higher in obese patients than in controls (p=0.015) However, fetuin-A levels did not differ significantly between the groups. Fetuin-A showed no correlation with insulin resistance, lipid parameters, hepatic steatosis, or hypertension.

Conclusion: Obesity-related complications (insulin resistance, dyslipidemia, fatty liver, and hypertension) were observed in more than half of our group. Although hs-CRP levels of obese cases were high, fetuin-A concentrations were observed to be similar in all cases and in subgroup analyzes. It may be due to factors affecting fetuin-A levels or its posttranslational modification. Further large-scale longitudinal studies are required to elucidate its contribution to metabolic inflammation in children.

Keywords: Adolescent, children, α2- Heremans-Schmid glycoprotein, obesity

Introduction

Childhood obesity is rising daily due to contemporary lifestyles globally. This condition is associated with several metabolic and cardiovascular consequences, including dyslipidemia, insulin resistance, type 2 diabetes mellitus (T2DM), metabolic dysfunction-associated steatotic liver disease (MASLD), and hypertension. Obesity is thought to initiate systemic inflammatory pathways, and obesity-induced endotoxemia may exacerbate associated comorbidities. Chronic inflammation is proposed to contribute to the etiopathogenesis of insulin resistance (1, 2).

Fetuin-A, also known as α2 -Heremans-Schmid glycoprotein, is a glycoprotein that inhibits the endogenous insulin receptor tyrosine kinase and is mostly produced in the liver (3). This hepatokin possesses various roles owing to its intricate structure and its interaction with distinct toll-like receptors (TLR) across diverse tissues (4). Numerous studies have shown that TLR4 is involved in obesity-related inflammation and insulin resistance, with fetuin-A serving as the endogenous ligand of TLR4 (4,5). Furthermore, it disrupts insulin signaling in skeletal muscle and adipose tissue by inhibiting the autophosphorylation of the insulin receptor (3,4). Another potential mechanism is the elevation of inflammatory cytokines and suppression of adiponectin (4,6). Fetuin-A is recognized as a mediator of inflammation. In adult research, fetuin-A is linked to various diseases, including infections, renal and cardiovascular disorders, cirrhosis, cancer, insulin resistance and metabolic syndrome (3,7,8,9). There exists a finite quantity of studies concerning children (5, 6, 10-15).

Investigating fetuin-A, a multifunctional protein, across several disease cohorts will enhance our comprehension of

its pathogenesis. This study aimed to assess fetuin-A levels in obese adolescents and their correlation with anthropometric measurements, insulin levels, and high-sensitive C-reactive protein (hsCRP).

Materials and Methods

The research was performed at the pediatric endocrinology outpatient clinic of Dr. Sami Ulus Training and Research Hospital between January and December 2017. Informed consent was acquired from the parents of all participants.

The study comprised 40 obese adolescents (BMI>95th percentile) and 30 healthy controls (BMI between the 15th and 85th percentiles) all of whom had no other systemic diseases and were not undergoing any pharmacological treatment. The study included cases aged 10 to 19 who were undergoing puberty. Anthropometric measures, fasting glucose, insulin, hsCRP, and fetuin-A levels were assessed in both groups. Information concerning obesity-related comorbidities, such as dyslipidaemia and hepatosteatosis, was obtained from hospital records. In the healthy control group, dyslipidemia, hypertension, and steatosis were not assessed. A SECA scale (SECA, Hamburg, Germany) was utilised to measure weight, while a Harpenden stadiometer (Holtain Ltd., Crymych, UK) was employed to assess height. Anthropometric reference data for the Turkish population, encompassing height, weight, and body mass index (BMI), were sourced from an internet database (www.ceddcozum.com) (16). Venous blood samples were obtained from both the patient and control groups prior to 9:00 AM following an overnight fast. Serum glucose, HbA1c, total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride levels were assessed using the Architect C16000 auto-analyzer system, whereas insulin concentrations were measured via the chemiluminescence method (Advia Centaur XP). Hs-CRP levels were quantified by a cassette-based assay with Getein 1600 immunofluorescence quantitative analyser (Getein Biotech, Nanjing, China). Serum fetuin-A levels were measured using a commercially available sandwich enzyme-linked immunosorbent test (ELISA) kit from Elabscience Biotechnology Inc., Houston, TX, USA.

Insulin resistance was assessed using the homeostatic model assessment (HOMA-IR), calculated as follows: HOMA-IR= [fasting insulin (μU/ml) × fasting glucose (mg/dl)]/405 (the cut-off was taken as 3.82 for pubertal girls and 5.22 for pubertal males) (17). Dyslipidemia was defined based on the American Academy of Pediatrics criteria, including total cholesterol ≥ 200 mg/dl, LDL cholesterol ≥ 130 mg/dl, triglycerides ≥ 130 mg/dl, HDL cholesterol < 40 mg/dl (18). Ambulatory blood pressure monitoring (ABPM) was applied to cases with three high blood pressure (BP) readings measured after at least 10 minutes of rest. An oscillometric monitor (Mobil-OGraph®NG, v.20.0, Stolber, Germany) and a correctly sized cuff were utilized on the non-dominant arm for ABPM. The monitor recorded BP measurements every 20 minutes from 08:00 to 20:00, then every 30 minutes from 20:00 to 08:00. Parameters for daytime, nocturnal, and 24-hour ABPM were documented. ABPM was deemed dependable if above 70% of the measurements were valid. The diagnosis of hypertension with ABPM was predicated on a mean systolic and/or diastolic BP exceeding the 95th percentile or a BP load beyond 25% (19).

Statistics analysis:

The Shapiro-Wilk test was used to assess the normality of data distribution. Continuous variables were expressed as mean and standard deviation or median (minimum-maximum), as appropriate. For variables adhering to a normal distribution, group comparisons were conducted using the Student t-test; otherwise, the Mann-Whitney U test was utilised. Group comparisons were performed using Chi-square or Fisher's exact test for categorical variables. Correlations between variables were analyzed using Spearman's rank correlation coefficient, as the data did not meet the assumption of normal distribution. Simple linear regression analysis was performed to evaluate the independent effects of metabolic parameters on serum fetuin-A levels. A p-value of less than 0.050 was deemed statistically significant. All analyses were conducted using SPSS software (version 22.0; IBM Corp., Armonk, NY, USA).

Results

Forty pubertal obese and 30 healthy adolescents were included in the study. Anthropometric and biochemical measurements are displayed in Table I, while the results of the subgroup analysis are shown in Table II. In our study, insulin resistance was observed in 65%, dyslipidaemia 77.5%, fatty liver 84%, and hypertension 35.9% of obese patients.

There was no difference in fetuin-A levels between obese and healthy cases in the whole group; however, obese patients had significantly higher hs-CRP levels than healthy individuals (p=0.015). Fetuin-A concentrations were comparable across boys and girls in the obese cohort. Although the fasting glucose levels of the patients were similar, the insulin and HOMA-IR levels in obese patients were considerably elevated compared to those in healthy individuals (p=0.127; p=0.000; p=0.000, respectively). No significant variations in fetuin-A and hs-CRP levels were identified between the insulin resistant and insulin non-resistant groups classified by HOMA-IR. Oral glucose tolerance test (OGTT) was conducted on 32 patients with additional risk factors within the obese cohort. In OGTT, the plasma glucose concentration at 0 minutes was 88.2±7.7 (72-106) mg/dl, and at 120 minutes, it was 116.7±25.6

Table I: Comparison of antropometric and laboratory findings between obese and control groups

	Obese	Normal-weight	p
Total number of patients	40	30	-
Gender (Female)*	65%	73.3%	0.463
Age (years)†	15.3±2.1	14.3±2.1	0.063
Height SDS†	0.33±1.4	0.145±0.7	0.073
BMI (kg/m^2)†	33.1±4.2	21.5± 2.2	0.001
BMI SDS†	2.8±0.55	0.4±0.7	0.001
Fasting glucose (mg/dl)†	95.1±8.8	91.4±8	0.127
Fasting insulin (μIU/ml)‡	21.4 (8.7-56.9)	11.4 (4.6-30.8)	0.001
HOMA-IR‡	5.01 (1.7-14.7)	2.5 (1.0-7.2)	0.001
Fetuin-A (ng/ml)‡	418.2 (255.9-638.6)	448.8 (232.9-694.2)	0.151
HSCRP (mg/dl)‡	0.23 (0-0.5)	0.11 (0-0.5)	0.015

**: n(%) (Chi-square test), †: mean±SD (Independent samples t-test), ‡: median (min-max) (Mann-Whitney U test)*

Table II: Comparison of laboratory findings according to insulin resistance, dyslipidemia, hepatic steatosis, and hypertension

	Insulin Resistance			Dyslipidemia			Steatosis			Hypertension		
	Yes	No	p	Yes	No	p	Yes	No	p	Yes	No	p
Total number of patients	26 (65%)	14 (35%)	-	31 (77.5%)	9 (22.5)	-	32 (84%)	6 (15.7%)	-	14 (35.9%)	25 (64.1%)	-
Gender(F)*	69.3%	57.2%	0.295	64.5%	66.7%	1.000	68.8%	33.3%	0.321	50%	72%	0.163
BMI (kg/m^2)†	34.1±4.2	31.7±4.2	0.386	33.6±4.4	31.5±3.6	0.103	33.9±4.3	29.8±3.3	0.049	36.2±4.7	31.6±3.1	<0.001
BMI SDS†	2.9±0.5	2.6±0.6	0.017	2.9±0.5	2.6±0.6	0.055	2.9±0.5	2.5±0.6	0.108	3.0±0.7	2.8±0.5	0.222
Fasting glucose (mg/dl)†	95±7.2	95.4±11.2	0.864	95.3±8.2	94.7±11.3	0.854	94.3±8.6	100.8±8.3	0.125	96.4±9.5	94.0±8.5	0.442
Fasting insulin (µIU/ml)‡	33.4 18.1-81.5	11.6 8.7-23.8	0.001	24. 8.7-56.9	12.8 9.3-23.8	0.002	21.2 8.7-42.9	12.1 11.4-12.8	0.985	22.6 9.9-81.5	27.2 8.7-51.3	0.917
HOMA-IR ‡	7.4 4.1-17.5	2.7 1.7-5.3	0.001	5.7 1.8-14.7	3.5 1.7-5.5	0.004	4.6 1.7-9.6	3.2 2.8-3.6	0.955	5.15 2.6-17.5	6.2 1.75-13.68	0.964
Fetuin-A (ng/ml) ‡	403.4 256-655.7	403 256.8-634.8	0.231	410.8 256-655.7	341 256.9-634.8	0.296	395.6 256-655.7	436.4 313.6-559	0.967	418.2 256.8-655.7	313.6 255.9-634.8	0.731
HSCRP (mg/dl) ‡	0.28 0.0-0.5	0.23 0.03-0.5	0.910	0.2 0.0-0.5	0.5 0.2-0.5	0.019	0.26 0.0-0.5	0.34 0.18-0.5	0.733	0.5 0.0-0.5	0.23 0.0-0.5	0.424

**: n(%) (Chi-square test), †: mean±SD (Independent samples t-test), ‡: median (min-max) (Mann-Whitney U test). Missing data: Insulin resistance group (n=38) and hypertension group (n=39); other groups included 40 patients. Analyses were performed using available data.*

(75-205) mg/dl. Glucose intolerance was identified in four cases and diabetes mellitus in one case. No significant association was seen between plasma glucose levels during the OGTT at 0,30,60,90 and 120 minutes and fetuin-A levels (correlation coefficients: r=-0.02; p=0.880; r=0.16, p=0.360; r=0.15, p =0.390; r=0.19, p=0.280; r=-0.06, p=0.710).

Eight of the obese patients exhibited elevated cholesterol levels, 15 presented with excessive triglycerides, eight had increased LDL, and 20 demonstrated low HDL. There was no significant difference in fetuin-A levels between dyslipidemic and non-dyslipidemic groups (p=0.296), however hs-CRP levels were considerably elevated in the non-dyslipidemic group (p=0.019). No association existed between lipid metrics and fetuin-A levels.

In simple linear regression analysis, no significant association was found between serum fetuin-A levels and lipid parameters in the obese group. Total cholesterol showed borderline but non-significant positive relationship with fetuin-A (B=1.397, p=0.086, R2=0.078), while LDL cholesterol (B=0.606, p=0.503), HDL cholesterol (B=2.288, p=0.318), and triglycerides (B=0.315, p=0.300) were not significant predictors of fetuin-A levels.

Thirty-two obese cases had fatty liver (Grade 1; 10 cases, Grade 2; 13 cases, Grade 3; 9 cases), with two cases showing elevated SGOT-SGPT levels. No significant difference was observed in fetuin-A and hs-CRP levels between cases with and without fatty liver (p=0.967, p=0.733, respectively). Hypertension was identified in 14 obese patients with 24-hour blood pressure monitoring. The fetuin-A levels in hypertension patients were 418.2 (256.8-655.7) ng/ml, compared to 313.6 (255.9-634.8) ng / ml in normotensive individuals, with no statistically significant difference (p = 0.731). Furthermore, there was no significant difference between the two groups regarding hs-CRP (p=0.424).

Discussion

The present study investigated the association between serum fetuin-A levels and anthropometric and metabolic parameters in obese adolescents. Our principal finding was that fetuin-A concentrations did not differ significantly between obese and healthy-weight pubertal adolescents, despite the presence of substantially elevated hs-CRP levels (p= 0.015) and a high prevalence of obesity-related metabolic complications, including insulin resistance (65%), dyslipidemia (77.5%), hepatic stetosis (84%), and hypertension (35.8%) in the obese group. Furthermore, fetuin-A showed no significant correlation with insulin resistance, OGTT glucose values at any time point, lipid parameters, hepatic steatosis grade, or hypertension status in subgroup analyses.

The absence of fetuin-A elevation in our obese adolescent cohort stands in marked contrast to the well- established association between fetuin-A and obesity in adult populations. In a large study of 345 adults with type 2 diabetes and 300 normoglycemic controls stratified by BMI (cut off 25 kg/m^2), demonstrated that obese individuals with type 2 diabetes had the highest fetuin-A concentrations, and that independent associations of fetuin-A with free fatty acids, HOMA-IR, CRP, and adiponectin were exclusively observed in the obese subgroups. The odds ratio for obesity increased progressively with fetuin-A quartiles (p for trend <0.001), leading the authors to conclude that fetuin-A functions as a molecular bridge connecting obesity and type 2 diabetes (3). In the context of MASLD, Elhoseeny et al.(8) reported that fetuin-A was markedly elevated in adult MASLD patients compared to controls, with a diagnostic cut off of 702.5 ng/mL yielding 82% sensitivity and 90% specificity. In another study, however, it has been showed that fetuin-A concentrations in type 2 diabetic adults were significantly influenced by total and LDL cholesterol levels, though no direct correlation with hepatic steatosis was observed. These adult findings collectively support a role for fetuin-A in mediating obesity-related metabolic dysfunction (9).

However, pediatric literature reveals a considerably more heterogeneous picture, with several studies reporting findings consistent with our results. Notably, in a pediatric study, evaluated fetuin-A in 81 obese children with biopsy-proven MASLD, 79 obese children with ultrasonographically detected MASLD, and 23 lean controls (10). They found virtually identical fetuin-A concentrations between obese and lean children (694.6±221.9

vs. 695.0±220.9 ng/mL, p=0.996). Although fetuin-A was higher in the ultrasound-detected MASLD group than in those without steatosis. There was no significant difference in fetuin-A between children biopsy-confirmed MASLD and those with simple steatosis, nor did fetuin-A differ across histological grades of steatosis, inflammation, ballooning, or fibrosis. The authors concluded that fetuin-A is not a reliable marker of liver damage severity in obese children. Conversely, other pediatric studies have reported elevated fetuin-A in association with obesity. In a study, evaluating 45 obese and 30 healthy children, they found significantly higher fetuin-A in obese children compared to controls (11). The MASLD subgroup (n=19) had the highest fetuin-A, though no significant difference existed between obese children with and without MASLD, and fetuin-A did not correlate with HOMA-IR, lipid parameters, or intima-media thickness (11). In a different study, significant correlations has been found between serum fetuin-A and BMI, waist and hip circumferences, subcutaneous fat thickness, and HOMA-IR in the 42 obese Turkish children compared to 40 controls (12). In a prepubertal cohort, Selvaraju et al. (14) demonstrated that salivary fetuin-A was markedly elevated in overweight and obese children aged 6-10 years compared to normal-weight peers, with strong positive correlations between fetuin-A and BMI z-score (r= 0.538), fetuin-A and insulin (r= 0.875), and negative correlation with adiponectin (r= 0.467). The authors have suggested that salivary fetuin-A could be valuable as a non-invasive marker. These conflicting findings across pediatric studies underscore the complexity of fetuin-A biology in the growing child.

Several mechanistic and methodological factors may explain the discordance in fetuin-A findings across pediatric studies. First, the developmental biology of fetuin-A must be considered. Fetuin-A is a multifunctional protein with significant biological variation across the lifespan (20). Serum concentrations are highest in neonates, gradually declining through childhood and stabilizing by late adolescence. On the other hand, posttranslational modifications of fetuin-A may play a critical role. Fetuin-A exists in two distinct compartments in the circulation: as a soluble plasma protein and within colloidal calciprotein particles, each with different functional implications (7). Most commercial ELISA kits, including the one used in our study, measure total fetuin-A without distinguishing between phosphorylated and non-phosphorylated forms. This methodological limitation could account for the apparent discrepancy between elevated hs-CRP and unchanged total fetuin-A levels in our obese cohort. Indeed, as Trepanowski et al. (4) emphasized, future studies must measure phosphorylated fetuin-A specifically to accurately assess its metabolic impact.

The complex organokine crosstalk network may differentially regulate fetuin-A across age groups and metabolic states. As a hepatokine, fetuin-A is primarily produced by the liver, but emerging evidence indicates that it also functions as an adipokine, being secreted from adipose tissue (4,7). Its expression is therefore influenced by adipose tissue inflammation, skeletal muscle metabolism, and systemic factors (21,22). Recent studies have highlighted the multifaceted endocrine role of hepatokines, including fetuin-A, noting that their clinical effects involve complex regulatory feedback loops (23). Kim et al. (22) examined the progression of metabolic syndrome, noting that it involves temporal activation of various adipokines and hepatokines, with the pattern of their dysregulation potentially differing between children and adults. These data suggests the organokine environment in adolescent obesity may fundamentally different from that in adult obesity, which could explain why fetuin-A does not behave as expected in our cohort.

Furthermore, differences in study populations-including age, pubertal status, ethnicity, degree and duration of obesity, genetic background, dietary habits, and physical activity levels-may all influence fetuin-A concentrations (20,24). A meta-analysis has been shown that exercise significantly modulates hepatokine levels in obese adults, with different exercise modalities (aerobic, resistance, and high-intensity interval training) producing varying effects on fetuin-A (24). Robinson et al. (20) reported that just seven days of aerobic exercise reduced fetuin-A 11% in adults. These observations suggest that even habitual physical activity levels in our adolescent cohort could have influenced the results, a variable we did not assess. The significantly elevated hs-CRP levels in our obese cohort (p=0.015) confirm the presence of systemic low-grade inflammation, validating our study's ability to detect obesity-related inflammatory changes. The dissociation between hs-CRP elevation and unchanged fetuin-A levels is particularly noteworthy. This pattern may reflect the activation of different inflammatory pathways at different stages of obesity progression. While CRP is an acute-phase reactant produced in response to IL-6 and IL-1b, fetuin-A's role in inflammation operates through a distinct mechanism involving TLR4 activation and the formation of the fetuin-A-free fatty acid complex (4).

Limitations

This study has several limitations. First, the relatively small sample size may have resulted in limited statistical power for fetuin-A comparison, which could explain the lack of statistically significant differences between groups despite a clinically relevant trend. Larger prospective studies are needed to confirm these findings. In addition, physical activity levels were not assessed. Given that exercise may influence circulating fetuin-A concentrations, the lack of adjustment for physical activity may have confounded the observed results.

Conclusion

In conclusion, although obese adolescents demonstrated significant metabolic and inflammatory alterations, fetuin-A levels were comparable to those of normal-weight peers and showed no association with metabolic risk parameters. This may indicate that fetuin-A has limited utility as a biomarker of early metabolic dysfunction in pediatric obesity. Longitudinal studies are needed to determine whether its clinical significance emerges only in more advanced stages of disease.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Ankara Numune Training and Research Hospital (25.01.2017, reference number: E-16–1134).

Contribution of the authors

Study conception and design: GKK, SÇ, ZA; data collection: GKK, SÇ, EK, EB, ŞÖ; analysis and interpretation of results: GKK, SÇ, GD, HSÖ; draft manuscript preparation GKK, SÇ, ŞSE, ZA. All authors reviewed the results and approved the final version of the article.

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Conflict of interest
The authors declare that there is no conflict of interest.

References

1. Leung AKC, Wong AHC, Hon KL. Childhood Obesity: An Updated Review. Curr Pediatr Rev. 2024;20(1):2-26. https://doi.org/10.2174/1573396318666220801093225
2. Podeanu MA, Ionele CM, Sandu RE, Rogoveanu I, Stepan MD, Niculescu CE, Cazacu SM, Vintilescu ŞB. Obesity, Metabolic Syndrome and MASLD in Children: Inflammation as the Missing Link-A Short Narrative Review. Life (Basel). 2026;16(2):310. https://doi.org/10.3390/life16020310
3. Zhou ZW, Ju HX, Sun MZ, Chen HM, Fu QP, Jiang DM. Serum fetuin-A levels in obese and non-obese subjects with and without type 2 diabetes mellitus. Clin Chim Acta. 2018;476:98-102. https://doi.org/10.1016/j.cca.2017.11.023
4. Trepanowski JF, Mey J, Varady KA. Fetuin-A: a novel link between obesity and related complications. Int J Obes (Lond). 2015;39:734-41. https://doi.org/10.1038/ijo.2014.203
5. Reinauer C, Reinehr T, Baechle C, et al. Relationship of serum Fetuin A with metabolic and clinical parameters in German children and adolescents with type 1 diabetes. Horm Res Paediatr. 2018;89:73-81. https://doi.org/10.1159/000484896
6. Ahn MB, Kim SK, Kim SH, et al. Clinical Significance of the Fetuin-A-to-Adiponectin Ratio in Obese Children and Adolescents with Diabetes Mellitus. Children (Basel). 2021;8(12):1155. https://doi.org/10.3390/children8121155
7. Jirak P, Stechemesser L, Moré E, et al. Clinical implications of fetuin-A. Adv Clin Chem. 2019;89:79-130. https://doi.org/10.1016/bs.acc.2018.12.003
8. Elhoseeny MM, Abdulaziz BA, Mohamed MA, Elsharaby RM, Rashad GM, Othman AAA. Fetuin-A: a relevant novel serum biomarker for non-invasive diagnosis of metabolic dysfunction-associated steatotic liver disease (MASLD): a retrospective case-control study. BMC Gastroenterol. 2024;24(1):226. https://doi.org/10.1186/s12876-024-03310-y
9. Gavril OI, Adam CA, Stamate TC, et al. Fetuin-A as a Link Between Dyslipidemia and Cardiovascular Risk in Type 2 Diabetes: A Metabolic Insight for Clinical Practice. Biomedicines. 2025;13(9):2098. https://doi.org/10.3390/biomedicines13092098
10. Pampanini V, Inzaghi E, Germani D, et al. Serum Fetuin-A levels in obese children with biopsy proven nonalcoholic fatty liver disease. Nutr Metab Cardiovasc Dis. 2018;281:71-6. https://doi.org/10.1016/j.numecd.2017.09.008
11. Lebensztejn DM, Białokoz-Kalinowska I, Kłusek-Oksiuta M, Tarasów E, Wojtkowska M, Kaczmarski M. Serum fetuin A concentration is elevated in children with non-alcoholic fatty liver disease. Adv Med Sci. 2014;59:81-4. https://doi.org/10.1016/j.advms.2013.08.003
12. Hızlı Ş, Abacı A, Özdemir O, Akelma Z, Akın O. Relation of fetuin A levels with cardiac, subcutaneous lipid accumulation and insulin resistance parameters in Turkish obese children. J Pediatr Endocrinol Metab. 2016;29:669-73. https://doi.org/10.1515/jpem-2015-0430
13. Manco M, Nobili V, Alisi A, Panera N, Handberg A. Arterial stiffness, thickness and association to suitable novel markers of risk at the origin of cardiovascular disease in obese children. Int J Med Sci. 2017;14:711-20. https://doi.org/10.7150/ijms.20126
14. Selvaraju V, Babu JR, Geetha T. Multiplexed measurements of salivary fetuin-A, insulin, and adiponectin as potential non-invasive biomarkers in childhood obesity. Cytokine. 2022;153:155843. https://doi.org/10.1016/j.cyto.2022.155843
15. Kącka A, Charemska A, Jarocka-Cyrta E, Głowińska-Olszewska B. Comparison of novel markers of metabolic complications and cardiovascular risk factors between obese non-diabetic and obese type 1 diabetic children and young adults. Front Endocrinol (Lausanne). 2022;13:1036109. https://doi.org/10.3389/fendo.2022.1036109
16. Demir K, Konakçı E, Özkaya G, et al. New features for child metrics: Further growth references and blood pressure calculations. J Clin Res Pediatr Endocrinol. 2020;12:125-9. https://doi.org/10.4274/jcrpe.galenos.2019.2019.0127
17. Kurtoğlu S, Hatipoğlu N, Mazıcıoğlu M, Kendirici M, Keskin M, Kondolot M. Insulin resistance in obese children and adolescents: HOMA-IR cut-off levels in the prepubertal and pubertal periods. J Clin Res Pediatr Endocrinol. 2010; 2:100-6. https://doi.org/10.4274/jcrpe.v2i3.100
18. Hampl SE, Hassink SG, Skinner AC, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity. Pediatrics. 2023;151(2):e2022060640. https://doi.org/10.1542/peds.2022-060640
19. Flynn JT, Daniels SR, Hayman LL, et al. American Heart Association Atherosclerosis, Hypertension and Obesity in Youth Committee of the Council on Cardiovascular Disease in the Young. Update: ambulatory blood pressure monitoring in children and adolescents: a scientific statement from the American Heart Association. Hypertension. 2014;63(5):1116-35. https://doi.org/10.1161/HYP.0000000000000007
20. Robinson KN, Teran-Garcia M. From infancy to aging: Biological and behavioral modifiers of Fetuin-A. Biochimie. 2016;124:141-9. https://doi.org/10.1016/j.biochi.2015.12.016
21. Ren Y, Zhao H, Yin C, Lan X, Wu L, Du X, Griffiths HR, Gao D. Adipokines, Hepatokines and Myokines: Focus on Their Role and Molecular Mechanisms in Adipose Tissue Inflammation. Front Endocrinol (Lausanne). 2022;13:873699. https://doi.org/10.3389/fendo.2022.873699
22. Kim JE, Kim JS, Jo MJ, Cho E, Ahn SY, Kwon YJ, Ko GJ. The Roles and Associated Mechanisms of Adipokines in Development of Metabolic Syndrome. Molecules. 2022;27(2):334. https://doi.org/10.3390/molecules27020334
23. Iglesias P. The endocrine role of hepatokines: implications for human health and disease. Front Endocrinol (Lausanne). 2025;16:1663353. https://doi.org/10.3389/fendo.2025.1663353
24. Ataeinosrat A, Abednatanzi H, Hajesfandiari S, Al Nasser MN, Dara MM, Farzanegi P, Nahand MS, Almaqhawi A, Karimi N, Bagherzadeh-Rahmani B, Saeidi A, Laher I, Zouhal H. Hepatokines modulation in obesity: which exercise training model is better in men with obesity? Front Endocrinol (Lausanne). 2025;16:1560453. https://doi.org/10.3389/fendo.2025.1560453