Ibrahim Card Med J 2025; 15 (2): 43-48

Correlation of Hepatic Enzymes with Lipid Profile in Obese Adolescents

Sumaiya Khan,[1] S. M. Ahshanul Kabir Al Aziz,[2] Nahid Yeasmin,[3] Shameema Akhter,[4] Tamanna Hasan[5]

ABSTRACT

Background & objective: Childhood and adolescent obesity have emerged as significant global health challenges, serving as major risk factors for non-alcoholic fatty liver disease (NAFLD) and dyslipidemia. In Bangladesh, the rising prevalence of adolescent obesity necessitates early screening markers for hepatic and metabolic complications. This study aimed to assess the relationship between serum hepatic enzymes (ALT and AST) and lipid profile parameters in obese adolescents.

Methods: This case-control study was conducted at the Department of Physiology, Dhaka Medical College, involving 52 obese adolescents (BMI ≥ 95th percentile for age and sex) aged 10–19 years. Biochemical analysis included serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), and a full lipid profile (TC, TG, LDL, and HDL). Data were analyzed using SPSS version 25.0, employing Pearson's correlation coefficient to determine the correlation between variables. A p-value of < 0.05 was considered statistically significant.

Results: The mean age of participants was 14.5 ± 2.6 years. Elevated ALT (≥ 40 U/L) was observed in 30.8% of subjects, while hypertriglyceridemia (≥ 130 mg/dl) was highly prevalent (76.9%). Correlation analysis revealed that serum ALT had a significant positive correlation with total cholesterol ($r = +0.365$, $p = 0.008$) and triglycerides ($r = +0.464$, $p < 0.001$). Serum AST showed weak, non-significant correlations with lipid parameters ($p > 0.05$).

Conclusion: Serum ALT and AST levels are significantly elevated in obese adolescents and ALT shows a strong positive correlation with atherogenic lipid fractions (TC and TG). These findings suggest that monitoring hepatic enzymes, particularly ALT, alongside lipid profiles may serve as a crucial tool for the early prediction and management of future NAFLD risk in the obese pediatric population.

Keywords: Hepatic Enzymes, Alanine Aminotransferase (ALT), aspartate aminotransferase (AST), Adolescent Obesity, Lipid Profile, etc.

ORIGINAL ARTICLE

INTRODUCTION

Childhood obesity has emerged as one of the most critical global health challenges of the 21st century.[1] The escalation of obesity rates is currently rising faster in children and adolescents than in the adult population.[2] This surge is particularly concerning as adolescent obesity serves as a significant predictor for adulthood obesity & the subsequent development of non-communicable diseases, including type 2 diabetes mellitus, hypertension, and dyslipidemia.[3] Furthermore, obesity is a primary driver of non-alcoholic fatty liver disease (NAFLD), a condition that can progress from simple steatosis to inflammatory steatohepatitis, fibrosis, and eventual cirrhosis.[3,4]

Authors' information:

[1] ***Dr. Sumaiya Khan***, *Assistant Professor (Physiology), Dhaka Dental College, Dhaka, Bangladesh.*

[2] ***Dr. S. M. Ahshanul Kabir Al Aziz***, *Assistant Professor (Paediatrics), Mugda Medical College Hospital, Dhaka, Bangladesh.*

[3] ***Professor Dr. Nahid Yeasmin***, *Department of Physiology, Dhaka Medical College, Dhaka, Bangladesh.*

[4] ***Dr. Shameema Akhter***, *Assistant Professor (Physiology), Dhaka Medical College, Dhaka, Bangladesh.*

[5] ***Dr. Tamanna Hasan***, *Lecturer (Microbiology), Shaheed Tajuddin Ahmad Medical College, Gazipur, Bangladesh.*

Correspondence: ***Dr. Sumaiya Khan***, *Mobile: 01718187323, E-mail: drsumaiyakhan123@gmail.com*

The World Health Organization (WHO) estimates that over 340 million children and adolescents aged 5-19 is affected by obesity worldwide.[5] While historically viewed as a problem of affluent nations, the majority of overweight or obese children now reside in developing countries[1]. In Bangladesh, where children and adolescents constitute over 60% of the 164 million population, recent trends indicate a rising prevalence of overweight status even in rural areas.[6,7] Projections suggest that by 2030, approximately 30% of the world's children will be affected by obesity.[8] The liver serves as the central hub for lipid homeostasis, processing large quantities of free fatty acids (FFAs). Under physiological conditions, hepatic triglyceride (TG) storage remains below 5%.[9] However, in obese individuals, visceral adipose tissue a metabolically active site undergoes high rates of lipolysis. This releases an excess of FFAs into the portal vein, leading to increased hepatic TG synthesis.[10] This surplus lipid not only disrupts liver anatomy and physiology but is also released into the bloodstream as very-low-density lipoproteins (VLDL), contributing to systemic hyperlipidemia.[11,12]

Clinically, liver biopsy remains the gold standard for assessing hepatic damage; however, its invasive nature makes it unsuitable for routine screening in pediatric populations.[13] Consequently, serum levels of hepatic enzymes specifically alanine aminotransferase (ALT), aspartate aminotransferase (AST), & alkaline phosphatase (ALP) are utilized as surrogate markers for hepatic dysfunction and injury.[14,15] While several international studies have explored the link between these enzymes and lipid profiles, there is a paucity of data specifically addressing these correlations within the adolescent population of Bangladesh.

Given that childhood obesity is a preventable condition, early identification of at-risk individuals is paramount. This study aims to assess the relationship between serum hepatic enzymes (ALT and AST) and lipid profiles in obese adolescents. By clarifying these correlations, we hope to provide clinicians and parents with better predictive tools for identifying early hepatic involvement, thereby facilitating timely lifestyle interventions to reduce the future burden of chronic liver disease.

METHODS:

Study Design and Setting

This case-control study was conducted in the Department of Physiology at Dhaka Medical College, Bangladesh, between July 2022 and June 2023. Ethical approval was obtained from the Ethical Review Committee (ERC) of Dhaka Medical College prior to the commencement of the study.

Participant Selection

A total of 52 obese adolescents, aged 10–19 years, were recruited. Obesity was defined as a Body Mass Index (BMI) ≥ 95th percentile for age and sex according to the Center for Disease Control and Prevention (CDC) growth charts. However, participants excluded if they:

- Were taking medications known to affect lipid or glucose metabolism (e.g., systemic steroids, estrogen, progesterone, insulin, diuretics, anticonvulsants, or antipsychotics).
- Suffering from genetic or endocrine disorders (e.g., Cushing's syndrome, hypothyroidism).
- Had a history of chronic renal, cardiac, or hepatic diseases.
- Had physical or mental disabilities.
- Reported a history of smoking or alcohol consumption.

Biochemical Analysis

Venous blood samples were collected after an overnight fast. Hepatic enzyme assessment focused on serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Lipid profile parameters included total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL).

Statistical Analysis

Data were processed and analyzed using SPSS version 25.0. Qualitative variables are presented as frequencies and percentages, while quantitative data are expressed as mean ± standard deviation (SD). Pearson's correlation coefficient (r) was employed to determine the relationship between serum hepatic

enzymes and lipid profile parameters. A p-value <0.05 was considered statistically significant.

RESULTS:

Participant Characteristics & Biochemical Profiles

The study included 52 obese adolescents with a mean age of 14.5 ± 2.6 years. The cohort was predominantly male (n = 32, 61.5%), with a male-to-female ratio of approximately 3:2. Biochemical analysis revealed that 30.8% of participants had elevated ALT (≥ 40 U/L) and 7.7% had elevated AST (≥ 38 U/L). Regarding lipid profiles, three qurters (76.9%) of the subjects exhibited hypertriglyceridemia (≥ 130 mg/dl), and 57.7% had low HDL levels (< 40 mg/dl). Hypercholesterolemia and elevated LDL were less prevalent, affecting 25.0% and 9.6% of the participants respectively (Table I).

Correlation Between Hepatic Enzymes & Lipid Profile

Pearson's correlation analysis showed that serum ALT had a significant positive correlation with total cholesterol (r = +0.365, p = 0.008) and triglycerides (r = +0.464, p < 0.001). Although ALT showed a positive correlation with LDL and a negative correlation with HDL, these did not reach statistical significance (p > 0.05). Serum AST demonstrated weak positive correlations with TC, TG, and LDL, and a negative correlation with HDL; however, none of these correlations were statistically significant (Table II, Fig 1 to 8).

Table I: Demographic and biochemical characteristics of the subjects (n = 52)

Parameters	Frequency %	Percentage %
Age (years)	---	14.5 ± 2.6
Gender		
Female	20(38.5)	---
Male	32(61.5)	---
Hepatic Enzymes		
Serum ALT (≥ 40 U/L)	16(30.8)	37.87 ± 3.17
Serum AST (≥ 38 U/L)	4(7.7)	31.3 ± 5.0
Lipid Profile		
Serum TC (≥ 200 mg/dl)	13(25.0)	154.2 ±22.5
Serum Tg (≥130 mg/dl)	40(76.9)	168.4 ± 43.2
Serum LDL (≥130 mg/dl)	5(9.6)	86.9±19.0
Serum HDL (< 40 mg/dl)	30(57.7)	33.7 ± 6.2

Table II: Correlation of liver enzymes with lipid profile in obese adolescents

Liver enzyme	Lipid profile	Pearson Correlation	
		r-value	p-value
S.ALT (U/L) with	S.TC (mg/dl)	+0.365*	0.008
	S.TG (mg/dl)	+0.464*	<0.001
	S.LDL (mg/dl)	+0.230	0.101
	S.HDL (mg/dl)	-0.239	0.088
S.AST (U/L) with	S.TC (mg/dl)	+0.081	0.569
	S.TG (mg/dl)	+0.258	0.064
	S.LDL (mg/dl)	+0.152	0.283
	S.HDL (mg/dl)	-0.204	0.146

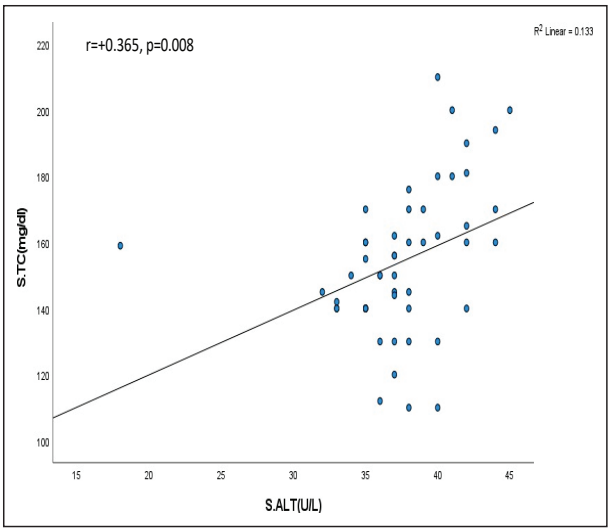

Figure 1. Correlation of S. ALT with S. TC

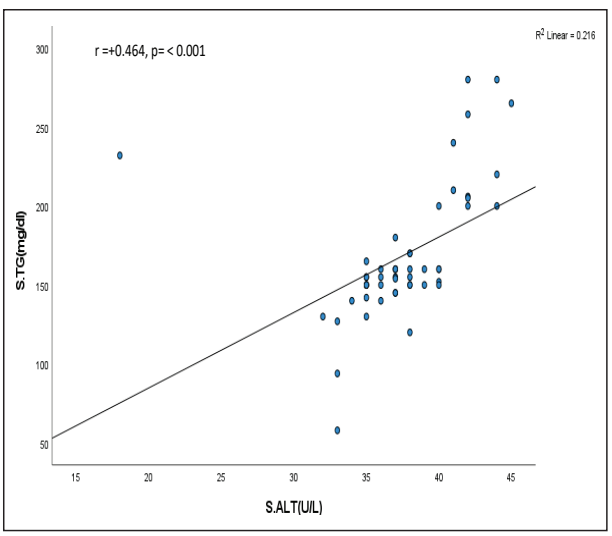

Figure 2. Correlation of S. ALT with S. TG

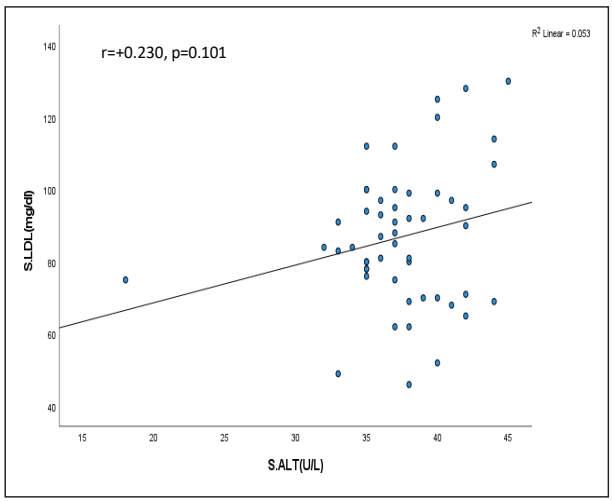

Figure 3. Correlation of S. ALT with S. LDL

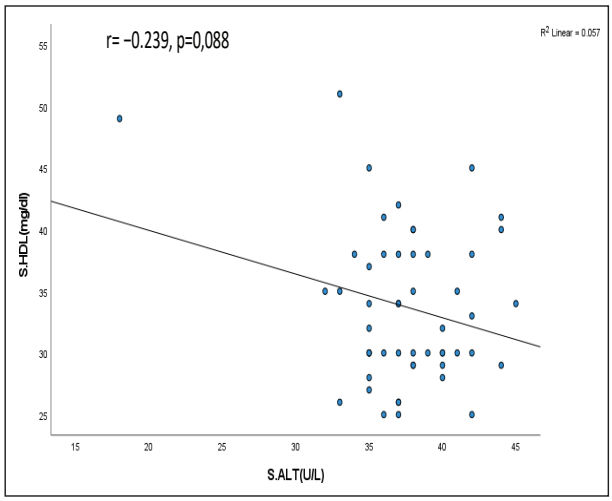

Figure 4. Correlation of S. ALT with S. HDL

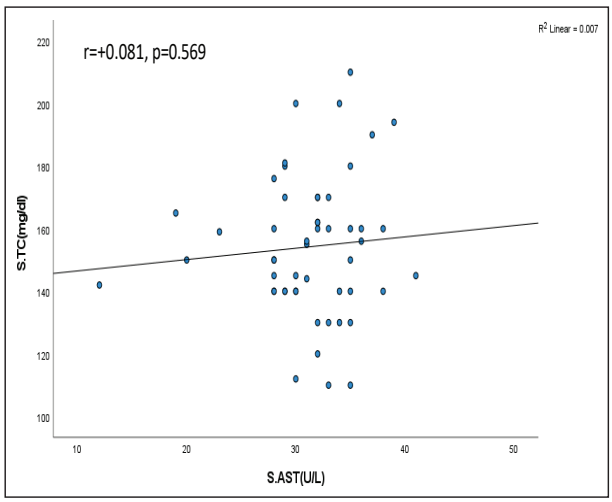

Figure 5. Correlation of S. AST with S. TC

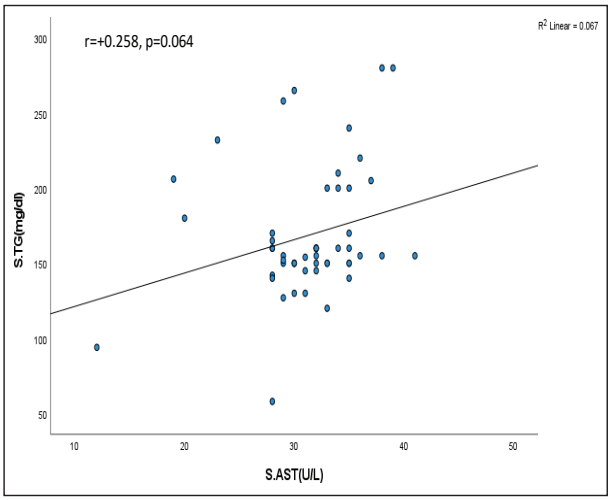

Figure 6. Correlation of S. AST with S. TG

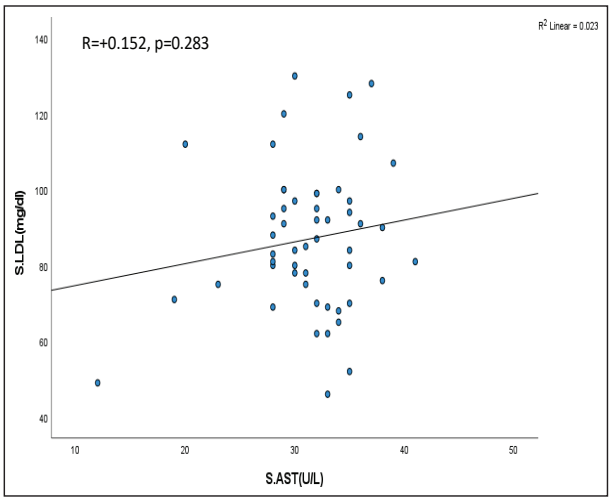

Figure 7. Correlation of S. AST with S. LDL

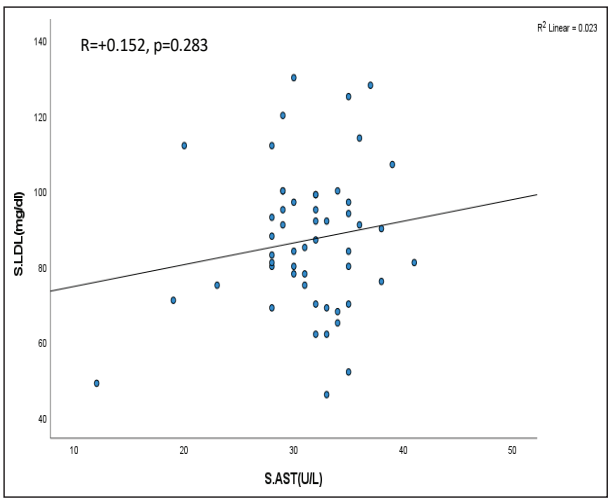

Figure 8. Correlation of S. AST with S. HDL

DISCUSSION:

The objective of this study was to evaluate the relationship between serum hepatic enzymes (ALT and AST) and lipid profiles in obese adolescents. Our findings demonstrate a significant elevation in body mass index (BMI), liver enzymes, and atherogenic lipid fractions in obese adolescents. These results align with previous studies highlighting the systemic metabolic impact of pediatric obesity.[11,12,14]

Hepatic Enzymes and Adiposity

In the present study, over 30% of adolescents had abnormally raised serum ALT (≥ 40 U/L), although elevated AST (≥ 38 U/L) was found in only 7.7% cases. This is consistent with findings by Valle-Martos et al.[16] and Putri et al.,[17] who observed that expanded adipose tissue functions as an active endocrine organ. In obesity, adipocytes release increased proinflammatory cytokines, such as TNF-α and IL-6, which trigger oxidative stress and hepatocellular injury.[9,18] Furthermore, elevated leptin levels common in obesity promote inflammatory pathways in the liver, leading to the leakage of ALT and AST into the bloodstream.[16]

While our findings regarding AST were consistent with Kelishadi et al.,[18] some researchers like Johansen et al.[19] found no significant association between AST and obesity. This discrepancy may be due to the fact that ALT is a more specific marker for hepatic steatosis than AST, which is also found in cardiac and skeletal muscle.[19]

Lipid Profile and Metabolic Alterations

Our results showed significantly higher TC, TG, and LDL levels and significantly lower HDL levels in obese adolescents. These findings are supported by Steinberger et al.[20] and Friedland et al.[12] The pathophysiological mechanism involves the delivery of excess FFAs from visceral fat to the liver, exceeding the organ's oxidative capacity.[8] This leads to the intrahepatic accumulation of triglycerides and the increased secretion of VLDL and LDL particles.[10] Furthermore, insulin resistance a hallmark of adolescent obesity impairs lipoprotein lipase activity, further elevating circulating TG and LDL while reducing HDL levels.[20]

Correlation Analysis

A key finding of this study was the significant positive correlation of ALT with TC (r = +0.365) and TG (r = +0.464) suggesting that 36% and 46% of variations in TC and TG respectively could be explained by variation in ALT. These results mirror those of Cigri et al.[15] and Pelin et al.[21] suggesting that ALT is not only a marker of liver injury but also a surrogate indicator of dyslipidemia. In our cohort, 76.9% of obese adolescents had hypertriglyceridemia, and 30.8% had elevated ALT. The strong correlation between these variables suggests that as liver enzymes rise, the lipid profile becomes increasingly atherogenic.

While AST showed positive correlations with TC, TG, and LDL, and a negative correlation with HDL, these did not reach statistical significance. This supports the clinical view that ALT is a more sensitive predictor of metabolic-associated fatty liver disease (MAFLD) and associated lipid abnormalities in the pediatric population.[17,22]

CONCLUSION

This study concludes that obese adolescents exhibit significantly higher levels of serum ALT, and AST, along with a detrimental lipid profile characterized by high TG, TC, and LDL, and low HDL. The significant positive correlation between ALT and serum lipids (TC and TG) suggests that ALT serves as a valuable early indicator of metabolic dysfunction. The most common lipid abnormality in this cohort seems to be hypertriglyceridemia followed by low HDL.

Given the high prevalence of hypertriglyceridemia (76.9%) and elevated ALT (30.8%) in our study group, routine screening of liver enzymes and lipid profiles is essential in the management of adolescent obesity. Early identification of these biochemical shifts can alert clinicians and parents to implement aggressive lifestyle and dietary interventions, potentially preventing the progression to non-alcoholic steatohepatitis (NASH) and cardiovascular disease in adulthood.

REFERENCES

1. Sahoo K, Sahoo B, Choudhury AK, Sofi NY, Kumar R, Bhadoria AS. Childhood obesity: causes & consequences. *J Family Med Prim Care* 2015;4(2):187-92.

2. Faienza MF, Chiarito M, Molina-Molina E, Shanmugam H, Lammert F, Krawczyk M, et al. Childhood obesity, cardiovascular and liver health: a growing epidemic with age. *World J Pediatr* 2020;16(5):438-45.
3. Kansra AR, Lakkunarajah S, Jay MS. Childhood and adolescent obesity: review. *Front Pediatr* 2021;8:581461.
4. Simon TG, Roelstraete B, Hartjes K, Shah U, Khalili H, Arnell H, et al. Non-alcoholic fatty liver disease in children and young adults is associated with increased long-term mortality. *J Hepatol* 2021;75(5):1034-41.
5. Mazidi M, Banach M, Kengne AP; Lipid and Blood Pressure Meta-Analysis Collaboration Group. Prevalence of childhood and adolescent overweight and obesity in Asian countries: a systematic review and meta-analysis. *Arch Med Sci* 2018;14(6):1185-203.
6. Rashid TJ, Haque M. Overweight and obesity in childhood and adolescence in Bangladesh and its consequences and challenges. *Bangladesh J Med Sci* 2022;21(4):667-75.
7. Rahman S, Islam MT, Alam DS. Obesity and overweight in Bangladeshi children and adolescents: a scoping review. *BMC Public Health* 2014;14:1-8.
8. Mazidi M, Banach M, Kengne AP; Lipid and Blood Pressure Meta-analysis Collaboration Group. Prevalence of childhood and adolescent overweight and obesity in Asian countries: a systematic review & meta-analysis. *Arch Med Sci* 2018;14(6):1185-1203. doi: 10.5114/aoms.2018.79001.
9. Fabbrini E, Sullivan S, Klein S. Obesity and nonalcoholic fatty liver disease: biochemical, metabolic, and clinical implications. *Hepatology* 2010;51(2):679-89.
10. Nobili V, Alkhouri N, Bartuli A, Manco M, Lopez R, Alisi A, et al. Severity of liver injury and atherogenic lipid profile in children with nonalcoholic fatty liver disease. *Pediatr Res* 2010;67(6):665-70.
11. Deb S, Puthanveetil P, Sakharkar P. A population-based cross-sectional study of the association between liver enzymes and lipid levels. *Int J Hepatol* 2018;2018:1-9.
12. Ponziani FR, Pecere S, Gasbarrini A, Ojetti V. Physiology and pathophysiology of liver lipid metabolism. *Expert Rev Gastroenterol Hepatol* 2015;9(8):1055-67.
13. Johansen MJ, Gade J, Stender S, Frithioff-Bøjsøe C, Lund MA, Chabanova E, et al. The effect of overweight and obesity on liver biochemical markers in children and adolescents. *J Clin Endocrinol Metab* 2020;105(2):430-42.
14. Jalili V, Poorahmadi Z, Ardekanizadeh HN, Gholamalizadeh M, Ajami M, Houshiarrad A, et al. The association between obesity with serum levels of liver enzymes, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase and gamma-glutamyl transferase in adult women. *Endocrinol Diabetes Metab* 2022;5(1):e00316.
15. Cigri E, Inan FC, Er E, Yildiz E. The relationship between lipid profile and non-alcoholic fatty liver disease in children and adolescents with obesity. *J Coll Physicians Surg Pak* 2022;32(5):591-5.
16. Valle-Martos R, Valle M, Martos R, Cañete R, Jiménez-Reina L, Cañete MD. Liver enzymes correlate with metabolic syndrome, inflammation, and endothelial dysfunction in prepubertal children with obesity. *Frontiers in Pediatrics* 2021;9:629346.
17. Putri RR, Casswall T, Hagman E. Prevalence of increased transaminases and its association with sex, age, and metabolic parameters in children and adolescents with obesity. *BMC Pediatrics* 2021;21(1):1-11.
18. Kelishadi R, Cook SR, Adibi A, Faghihimani Z, Ghatrehsamani S, Beihaghi A, et al. Association of the components of the metabolic syndrome with non-alcoholic fatty liver disease among normal-weight, overweight and obese children and adolescents. *Diabetol Metab Syndr* 2009;1:29. doi: 10.1186/1758-5996-1-29.
19. Johansen MJ, Gade J, Stender S, Frithioff-Bøjsøe C, Lund MA, Chabanova E, et al. The effect of overweight and obesity on liver biochemical markers in children and adolescents. *The Journal of Clinical Endocrinology & Metabolism* 2020;105(2):430-442.
20. Steinberger J, Moorehead C, Katch V, Rocchini AP. Relationship between insulin resistance and abnormal lipid profile in obese adolescents. *The Journal of Pediatrics* 1995;126(5):690-695.
21. Pelin AM, Balan G, Stefanescu C, Rosca S, Busila C. New criteria in defining the metabolic syndrome in children. *Progress in Nutrition* 2021;23(4):2021316.
22. Celik N, Andıran N. The relationship between serum phosphate levels with childhood obesity and insulin resistance. *Journal of Pediatric Endocrinology and Metabolism* 2011;24(1-2):81-83.