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Evaluation of Serum Chemerin and Lipid accumulation product as Predictors of Non-Alcoholic Fatty Liver Disease in Simple Obese Egyptian Children

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Abstract

Background

The increase in the prevalence of obesity worldwide has led to non-alcoholic fatty liver disease (NAFLD) becoming one of the most common causes of chronic liver disease. Chemerin is a novel adipokine which regulates adipogenesis which is also a marker of systemic and vascular inflammation. Lipid accumulation product (LAP) is associated with the presence and severity of nonalcoholic fatty liver disease (NAFLD) in adults. We aimed to evaluate the clinical usefulness of both serum chemerin and LAP as predictors of NAFLD in children with simple obesity.

Materials and Methods: This was a prospective cross-sectional study including 65 obese children with age range of 6–18 years old from pediatric obesity and endocrine outpatient clinic, Children's University Hospital, Minia University, Egypt, in addition to 30 healthy children, age and sex matched as control group. The included children were subjected to careful history taking, thorough clinical examination and laboratory investigations including liver enzymes, fasting blood glucose (FBG), fasting serum insulin, lipid profile and serum chemerin. Then LAP was calculated using waist circumference and serum triglyceride.

Results: Serum chemerin and LAP were significantly higher in obese children (p-<0.01). LAP had 95.2% sensitivity and 70.5% specificity at a cut-off point > 41; while serum chemerin at a cut-off point of > 271.7 ng/dl showed an 85.4% sensitivity and 51.4%% specificity for prediction of liver steatosis in our obese participants.

Conclusion

Based on the results, serum chemerin may be considered as an acceptable indicator of NAFLD in obese children but LAP is a more available, easy and inexpensive tool to predict NAFLD in those children.

Key Words: Children, Chemerin; LAP, Non-alcoholic fatty liver disease, Obesity.

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1- INTRODUCTION

Many studies showed that obesity has reached alarming levels in both children and adults (1). In Egypt, studies indicated that obesity has become one of the most important health problems especially in school aged children (2). Salem et al. (3) in 2002 showed that the prevalence of obesity among Egyptian children was 14% and 15% in boys and girls, respectively. This prevalence increased in 2016 to 19.6%. It was significantly higher among girls (24.7%) than boys (14%), and among urban participants (22%) than rural ones (11%) (1). Nonalcoholic fatty liver disease (NAFLD) has become one of the most common causes of chronic liver disease, in both adults and children due to the increase in the prevalence of obesity observed worldwide during the last few decades (4). NAFLD contributes to acceleration of atherosclerosis development, and there is a higher risk of developing cardiovascular disease compared to individuals in the general population (5, 6).

NAFLD is complex multi factorial disease so there is not a sufficient single marker to predict its clinical outcome or the benefits of a therapy. Despite the fact that all biomarkers and scores have their limitations, there is increased interest in the use of, these markers to predict information about progression and outcome of this disease (7). Adipose tissue is considered as a complex network of endocrine organs that secrete many active adipokines with different functions (8). This endocrine function of adipose tissues in adults is well known, but its neurohormone activity in the children is not yet clarified (9). Pharmacologically, adipokines are active, low molecular weight proteins that exert different functions through several pathways (10). Many factors are involved in the regulation of adipocytokine synthesis as body mass index (BMI), insulin, and tumor necrosis factor-α (TNF-α) (11-12). However, an accurate assessment of the role of adipokines is still deficient in the pediatric population. We should note that the studies performed in children may be more persuasive when compared to adults, due to decreased confounding factors (13). It has been thought that novel adipokines such as chemerin, omentin, vaspin might have a strong relationship with obesity, insulin resistance, atherosclerosis, metabolic syndrome and fatty liver (14). It has been shown in a few studies, comprised of adult populations, that some of these adipokines are displayed in higher concentrations in NAFLD patients compared to the control (14, 15). They may have an important role in progression from simple steatosis to inflammation and fibrosis (13). Chemerin is involved in different physiological and pathophysiological processes and it regulates adipogenesis, insulin sensitivity, and immune response, suggesting a vital role in metabolic health (16).

Studies conducted in pediatric populations confirmed that chemerin may be regarded as a marker of systemic and vascular inflammation (17, 18). Lipid accumulation product (LAP) depends only on the measurement of waist circumference (WC), and fasting triglycerides. It was first described as an alternative and powerful index for recognizing cardiovascular risk in adults (19). Several studies supported the use of this index for the screening of metabolic syndrome in healthy population and different patient groups (20-23). The LAP has also been associated with the presence and severity of nonalcoholic fatty liver disease (NAFLD) in adults (24). Cross-sectional analysis demonstrated that LAP was superior to BMI in detecting some prevalent cardio-metabolic risk factors and diabetes (19, 25). Hence, this study aimed to determine the clinical usefulness of both serum chemerin and

LAP as predictors of NAFLD in children with simple obesity.

2- MATERIALS AND METHODS

2-1. Study design and subjects

This was a prospective cross-sectional study including 65 obese children with age range of 6–18 years old from pediatric obesity and endocrine outpatient clinic, Children's University Hospital, Minia University, Egypt, in addition to 30 healthy children age and sex matched as control group. Obese children were diagnosed according to body mass index (BMI) Z-score ≥ 95th percentile according to National Center for Health Statistics (NCHS) standards (26).

Patients known to be under treatment for chronic diseases (epilepsy, bronchial asthma, etc.), and those with viral hepatitis A, B, or C infection, autoimmune hepatitis, history of alcohol intake, drug-induced fatty liver, genetic and metabolic liver disorders (Wilson, cystic fibrosis, alpha-1 antitrypsin), or any other causes of chronic liver diseases were excluded from our study. The included children were subjected to careful history taking, thorough clinical examination and laboratory investigations including liver enzymes [alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT)], fasting blood glucose (FBG), fasting serum insulin, lipid profile [total cholesterol (TC), serum triglycerides (TG) level, serum high-density lipoprotein (HDL), serum low-density lipoprotein (LDL).

All anthropometric measurements were taken using standardized equipment and following the recommendations of the International Biological program (27). Body weight, height, and waist circumference (WC), and hip circumference (HC) were measured and recorded as the mean of three measurements using standard equipment by the same clinician. Weight was measured using an electronic scale and estimated with a precision of 10 g. Height was measured using a stadiometer to the nearest 0.5 cm with the child barefoot, face looking straight forward, back against the wall. The waist circumference (WC) was measured at the end of normal expiration, midway between the lowest rib and the superior border of iliac crest, at a level parallel to the floor with a non-stretchable tape to the nearest 0.1 cm. Standard deviation (Z) scores were calculated for weight for age, height for age, weight for height, and BMI using NCHS standards (26). Blood pressure was measured with children in a sitting position, using the same mercury sphygmomanometer with cuff appropriate for body size. Hypertension was diagnosed according to Egyptian blood pressure charts for children and adolescents (28).

We employ Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) model to quantify insulin resistance using this formula: [fasting plasma glucose (mmol/l) × fasting serum insulin (mU/l)] /22.5 (29). High HOMA-IR value implies low insulin sensitivity and vice versa. HOMA-IR value of >4.0 indicates insulin resistance (30).

The LAP was calculated as [WC (cm)–58] × triglyceride (TG) concentration (mmol/L) in girls; [WC (cm)–65] × TG concentration (mmol/L) in boys (31).

2-2. Sampling and laboratory methods

Six milliliters of venous blood were withdrawn after fasting overnight collected under complete aseptic condition. Serum samples were frozen and stored at −20°C. Fasting blood glucose, ALT, AST, GGT, TC, LDL, HDL and TG levels were measured using fully automated clinical chemistry auto analyzer system Konelab 60i (Thermo Electron Incorporation, Finland). Insulin was measured using Cobas e411 (Roche

Diagnostics, Mannheim, Germany). Serum Chemerin measured by (Chemerin DuoSet ELISA Kit, R&D Systems, Inc., Minneapolis, MN, USA).

2-3. Ultrasound assessment of liver steatosis

Ultrasound was performed using NemioXG device (Toshiba Medical Systems, Tochigi, Japan) equipped with convex multi-frequency probes. The child was lain in dorsal decubitus with the right arm in the maximal abduction. Ultrasound examination was done by the same radiologist who was blinded to all clinical and biochemical characteristics of subjects. Liver steatosis was evaluated according to a series of ultrasound findings including liver echogenicity, hepatorenal echo contrast, visualization of intrahepatic vessels, and visualization of liver parenchyma and the diaphragm. Steatosis score was graded as the following: absent (score 0) steatosis was defined as normal liver echotexture; mild (score 1) steatosis as slight and diffuse increase in fine parenchymal echoes with normal visualization of diaphragm and portal vein borders; moderate (score 2) steatosis as moderate and diffuse increase in fine echoes with slightly impaired visualization of portal vein borders and diaphragm; severe (score 3) steatosis as fine echoes with poor or no visualization of portal vein borders, diaphragm, and posterior portion of the right lobe (32).

2-4. Ethics statement

The study was conducted according to the principles of Helsinki and in agreement with the Faculty of Medicine, Minia University, Ethical committee (No: 116-5-2018). Informed written consents from the patient's caregiver were obtained.

2-5. Statistical analysis

Statistical Package for Social Sciences (SPSS) software version 21.0 was used for data entry and analysis. Descriptive statistics were expressed for quantitative data by mean and standard deviation. They were presented for categorical data as number and percentage. Analyses were done for quantitative data using t-test. However, for qualitative data, Chi-square test or Fisher Exact test was done when appropriate. The degree of relationship between the variables was calculated using the Pearson correlation analysis. Correlation coefficient (r) ranges from (0-1): weak (r = 0–0.24), fair (r = 0.25–0.49), moderate (r = 0.5–0.74), strong (r = 0.75–1). Receiver operating characteristic (ROC) curve analysis was performed using SPSS to determine the optimal cut-off values and diagnostic performance of the variables. Cut-off point was determined according to ROC curve, the point with maximum specificity and sensitivity. The level of significance was taken at P-value < 0.05.

3- RESULTS

Anthropometric parameters and clinical characteristics of the studied children used in this study were summarized in **Table.1**. A total of 65 obese and 30 normal control age- and sex-matched participants were studied. We found a significant difference in weight, BMI, WC, HC, WC/HC ratio and diastolic blood pressure between the obese and control groups. **Table.2** shows that obese group also had significantly higher ALT, AST, GGT, TC, TG, LDL, FPG, fasting insulin levels, HOMA-IR, serum chemerin and LAP ($p<0.01$for each of them) while control group had a higher HDL than obese group ($p<0.01$).

Table-1: Clinical characteristics of the studied obese and control children.

Variables	Groups		P- value
	Group I, Obese children, (n=65)	Group II, Controls (n=30)	
Age (year)	9.3 ± 2.1	8.5 ± 1.3	0.15
Sex (male/female)	31/34	13/17	0.45
Weight (kg) (Z score)	2.3 ± 1	0.55 ± 0.43	<0.01*
Height (cm) (Z score)	-0.8 ± 0.99	-0.6 ± 1.1	0.42
Body mass index(Z score)	2.7± 0.6	1.5 ± 0.4	<0.01*
Waist Circumference (cm)	98.2 ± 19.3	62.5 ± 9.5	<0.01*
Hip Circumference (cm)	109.5 ± 13.7	84.9 ± 9.2	<0.01*
Waist/hip ratio	0.8 ± 0.3	0.9 ± 0.2	0.03*
Diastolic blood pressure, (mmHg)	75 ± 10	65 ± 5	<0.01*
Systolic blood pressure, (mmHg)	100 ±5	95 ± 5	0.18

* Significant.

Table-2: Biochemical profile of the studied obese and control children.

Groups	Groups		P. value (Sig.)
	Group I, Obese children, (n=65)	Group II, Controls (n=30)	
ALT (U/L)	61.3 ± 21.4	35.3 ± 4.2	<0.01*
AST (U/L)	59.8 ± 24.5	30.0 ± 4.5	<0.01*
GGT(U/L)	35±12.2	12±3.2	<0.01*
Cholsetrol (mmol/L)	6.1±0.58	4.01±0.41	<0.01*
Triclycrides (mmol/L)	1.67±0.4	0.89±0.2	<0.01*
LDL (mmol/L)	3.75±0.4	2.3±0.4	<0.01*
HDL (mmol/L)	0.85±0.3	1.6±0.4	<0.01*
FPG (mol/L)	6.3±2.2	4.3 ±0.8	<0.01*
2-hours Postprandial glucose, (mol/L)	5.4±0.5	5±0.3	0.07
Fasting insulin (µU/mL)	15.25±3.63	9.48±2.56	<0.01*
HOMA-IR	4.42±0.30	1.21±0.22	<0.01*
Ultrasound liver steatosis			<0.01*
Normal	18(27%)	30	
Grade 1	23(35%)	0	
Grade 2	21 (33%)	0	
Grade 3	3(5%)	0	
Chemerin (ng/dl)	301.3 ± 62.1	114.8 ± 23.7	<0.01*
LAP	43.3 ±40.7	22.1 ± 12.9	<0.01*

*Significant. ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, FPG: Fasting plasma glucose, GGT: Gamma-glutamyl transferase, HDL: High density lipoprotein, HOMA-IR: Homeostatic model assessment of insulin resistance, LAP: lipid accumulation product, LDL: Low density lipoprotein.

Table.3 shows the comparison between obese participants with and without liver steatosis and showed that BMI, WC, ALT, AST, GGT, TG, fasting insulin, HOMA-IR and LAP were significantly higher in obese participants with steatosis than in those without steatosis.

Table-3: Comparison of the studied clinical characteristics, and biochemical profile between children with or without liver steatosis.

Variables	Groups		P- value (Sig.)
	Group I a steatosis (n=47)	Group I b Without steatosis (n=18)	
Age (year)	9.1 ± 2.3	9.75 ± 1.2	0.6
Gender	20/27	11/7	0.08
Body mass index(Z score)	2.8± 0.7	2.5 ± 0.4	<0.01*
Waist Circumference (cm)	98.2 ± 19.3	94.5 ± 18	0.03*
ALT (U/L)	68.3 ± 25.9	45.8 ± 9.9	0.04*
AST (U/L)	62.5 ± 28.2	42.8 ± 13.5	0.02*
GGT(U/L)	55.2 ±6.2	25.8±4.5	0.05*
Cholsetrol (mmol/L)	6.2±0.4	6.1±0.55	0.78
Triclycrides (mmol/L)	1.77±0.2	1.32±0.5	0.02*
LDL (mmol/L)	3.8±0.6	3.7±0.3	0.21
HDL (mmol/L)	0.87±0.4	0.84±0.1	0.93
FPG (mol/L)	6.5±2.3	6.3±1.5	0.54
2-hours Postprandial glycemia (mg/dL)	5.45±0.4	5.4±0.3	0.09
Fasting insulin (µU/mL)	15.28±3.4	12.1±2.5	0.04*
HOMA-IR	4.44±0.41	3.41±0.35	0.03*
Serum chemerin (ng/dl)	302.4 ± 73	301.9 ± 55.2	0.9
LAP	45.3 ±35.7	32.1 ± 23.9	<0.01*

*Significant. ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, FPG: Fasting plasma glucose, GGT: Gamma-glutamyl transferase, HDL: High density lipoprotein, HOMA-IR: Homeostatic model assessment of insulin resistance, LAP: lipid accumulation product, LDL: Low density lipoprotein.

We found significant correlations between chemerin levels with AST, ALT, GGT, HDL, and insulin levels, and HOMA-IR (**Table.4**). While LAP was significantly correlated with BMI, WC, ALT, AST, GGT, TC, TG, LDL, HDL, FBS, insulin levels, HOMA-IR and, serum chemerin. ROC curve analysis of serum chemerin and LAP for prediction of hepatosteatosis showed that LAP had 95.2% sensitivity and 70.5% specificity at a cut-off point > 41 while serum chemerin at a cut-off point of > 271.7 ng/dl showed an 85.4% sensitivity, and 51.4%% specificity (**Table.5**, **Figure.1**).

Table-4: Correlations of both serum Chemerin, and LAP, with clinical and biochemical characteristics in obese children.

Variables	LAP		Serum chemerin	
	r	P-value	r	P-value
Age (year)	0.21	0.08	0.22	0.7
Body mass index(Z score)	0.55	<0.01*	0.45	0.09
Waist circumference(cm) (WC) (Z score)	0.65	<0.01*	0.52	0.07
ALT (U/L)	0.44	0.03*	0.55	0.01*
AST (U/L)	0.54	<0.01*	0.41	0.02*
GGT(U/L)	0.68	<0.01*	0.45	0.04*
Cholesterol (mg/dL)	0.56	0.02*	0.24	0.06
Triglycerides (mg/dL)	0.53	0.02*	0.14	0.88
LDL (mg/dL)	0.54	0.01*	0.32	0.7
HDL (mg/dL)	- 0.44	0.04*	- 0.25	<0.01*
FBS (mg/dl)	0.35	0.04*	0.16	0.24
2-hours Postprandial glycemia (mg/dL)	0.36	0.08	0.23	0.55
Fasting insulin (µU/mL)	0.52	0.04*	0.44	<0.01*
HOMA-IR	0.84	<0.01*	0.63	<0.01*
Serum chemerin	0.54	<0.01*	--	--

*Significant, r: Pearson correlation, Abbreviations: ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, FPG: Fasting plasma glucose, GGT: Gamma-glutamyl transferase, HDL: High density lipoprotein, HOMA-IR: Homeostatic model assessment of insulin resistance, LAP: lipid accumulation product, LDL: Low density lipoprotein.

Table-5: ROC curve analysis of serum chemerin and LAP for prediction of liver steatosis in obese children.

Parameters	AUC	Cutoff point	Sensitivity %	Specificity %	PPV	NPV
Serum chemerin (ng/dl)	0.757	> 271.7	85.4%	51.4%	68.9	70.4
LAP	0.853	> 41	95.2%	70.5%	81.0	83.3

*Significant. LAP: Lipid accumulation product, AUC: area under the curve, LAP: lipid accumulation product, NPV: Negative predictive value, PPV: positive predictive value, ROC curve: Receiver operating characteristic curve.

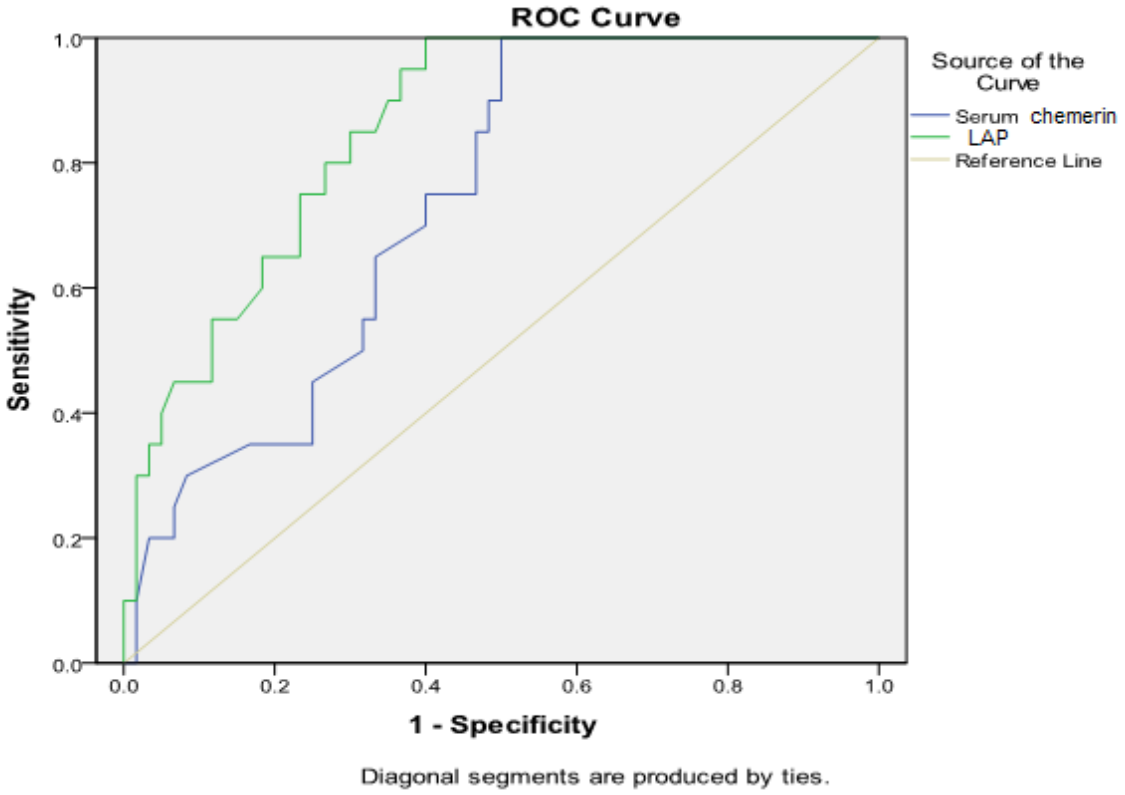

Fig.1: ROC curve analysis of serum chemerin and LAP for prediction of liver steatosis in obese children. LAP: Lipid accumulation product, ROC curve: Receiver operating characteristic curve.

4- DISCUSSION

Obesity and its complications such as NAFLD became an important public health problem with increasing prevalence in Egyptian children. We need easier and less expensive methods for screening and following-up those patients and their complications. This study was carried out to examine the diagnostic utility of serum chemerin levels and LAP as new predictors of NAFLD in childhood obesity Obesity is an important factor in the development of hypertension in children. Diastolic blood pressure (DBP) was significantly higher in obese participants than in our controls, this may be attributed to the increased levels of insulin and insulin-like growth factor I in obese patients which can increase blood pressure (33). Our results were in agreement with the results of Nageswari et al. (34) who explained high DBP by higher vasoconstrictor tone and increase in the cardiac output owing to increased circulatory load on heart due to increase in BMI. However, Divković et al. (35) showed that obese children had significantly higher systolic blood pressure compared to eutrophic children, while diastolic pressure showed no difference in both groups. Liver enzymes (AST, ALT, and GGT) showed mild to moderate elevations in our obese children; agreeing with the results of Ekstedt et al. (36) and Li Hui-ling et al. (37) who explained this by insulin resistance in those patients which increases lipolysis from the adipose tissue. Lipolysis increases Free fatty acids (FFA) taken by the liver and can cause lipid peroxidation and so, increased production of inflammatory cytokines. Our results show higher TC, LDL and TG in obese children compared with control group, while HDL was higher in healthy children than obese ones. This was in line

with the obesity-related hyperlipidemia which is primarily characterized by increased levels of plasma free fatty acids and triglycerides, decreased levels of HDL with abnormal low-density lipoprotein (LDL) composition (38). In our study, higher mean fasting blood glucose level (FBG) in obese participants was comparable to healthy ones which may indicate that obesity is a risk factor for impaired glucose tolerance. This was in agreement with the results described by Elghaffar et al. (39) who showed the positive correlation between obesity and hyperglycemia as blood sugar concentrations increased with increasing adiposity. Insulin resistance is defined as the decreased ability of tissues to respond to insulin action and one of the insulin-responsive tissues is adipose tissue.

We found significant increase in fasting insulin and HOMA-IR in obese participants than the control ones These results were consistent with Kurtoğlu et al. (40) who showed the rate of insulin resistance was found to be 37% in boys and 27.8% in girls, in prepubertal period while in the pubertal period, this rate was 61.7% in boys and 66.7% in girls. HOMA−IR cut−off values for insulin resistance in the prepubertal period were calculated to be 2.67 in boys and 2.22 in girls, and in the pubertal period, they were 5.22 in boys and 3.82 in girls.

Also, fasting insulin and HOMA-IR were higher in obese participants with liver steatosis than without steatosis which could be explained by the ability of NAFLD to reduce the inhibitory effect of insulin in the body on endogenous glucose production, and reduce the body's insulin sensitivity, making it difficult to control blood glucose (41). In our study, significantly higher serum chemerin was found in obese children compared to the control. Our results were in agreement with Ba et al. (42), and Kłusek-Oksiuta et al. (43) who showed increased chemerin level in NAFLD children and they concluded that serum chemerin can predict both intrahepatic lipid content in obese children and advanced liver steatosis in children with NAFLD. We observed higher concentrations of chemerin in children with hepatic steatosis compared to obese children without liver pathology. This observation was confirmed in the ROC analysis which showed the ability of serum chemerin to differentiate children with fatty liver from those without steatosis. This was in accordance with Kłusek-Oksiuta et al. (43) who found the ability of serum chemerin to differentiate children with fatty liver but they used Proton magnetic resonance spectroscopy (1H-MRS) for estimation of liver fat content.

Another study has confirmed a high expression of chemerin mRNA in hepatocytes (44). This can indicate the usefulness of serum chemerin as a novel marker of NAFLD in obese children. There were significant correlations between serum chemerin and liver enzymes, which may be regarded as an indicator of inflammatory processes in the liver, so it seems that children with NAFLD who demonstrate high serum chemerin may not only present simple fatty liver, but also its progression to non-alcoholic steato-hepatitis (43). Studies conducted in pediatric populations confirmed that chemerin may be regarded as a marker of systemic and vascular inflammation (17, 18).

Chemerin significantly correlates with insulin and HOMA-IR. This was the same as Sledzińska et al. (45) who showed the correlation of serum chemerin with insulin resistance indexes (HOMA-IR) and fasting insulin in children and Lee et al in young adults. These results suggested that chemerin may play a role in insulin resistance in obese peoples. Chemerin may affect the insulin receptor signaling pathway, which leads to insulin resistance

or aggravates the body's original insulin resistance (46). Lehrke et al. (47) showed that chemerin is increased in adipocytes, which may activate serine/threonine kinases, so reducing the tyrosine phosphorylation, inhibiting the translocation of glucose 4, and causing insulin resistance in adipocytes. Lipid accumulation product was first introduced by Kahn (19) as an index of excessive lipid accumulation and was suggested as a powerful tool to predict cardiovascular risk. Also, it has been considered a strong predictor of metabolic syndrome and NAFLD in adults (21, 48, 49).

The waist circumference and triglyceride level are the only 2 variables for calculating LAP (19). A fasting lipid profile is recommended for all obese children and WC value can be easily obtained (31). Our study showed higher LAP score in obese participants than the normal controls. Also, it was significantly higher in obese children with liver steatosis. With significant correlation with BMI, WC, liver enzymes, lipid profile, FBS, insulin levels, HOMA-IR and, serum chemerin. LAP showed 95.2% sensitivity and 70.5% specificity at a cut-off point > 41 for prediction of hepatosteatosis. These results were consistent with Özcabı et al. (31) who identified the cutoff value for LAP as 42.7 (sensitivity, 53.7%; specificity, 84.6%).

The LAP, systolic blood pressure, fasting insulin, ALT, uric acid and HOMA-IR values were significantly higher in their study children with NAFLD. Dai et al. (21) have evaluated the accuracy of LAP for diagnosing NAFLD in general adult population. The identified cutoff values for LAP were 30.5 in men (sensitivity, 77%; specificity, 75%), and 23.0 (sensitivity, 82%; specificity, 79%) in women, respectively. They have also marked that for both sexes the diagnostic accuracy for LAP was significantly better in younger groups.

4-1. Study Limitations

Besides the lack of morphological examination in the studied group which is the best diagnostic tool for confirming NAFLD, the relatively small sample size limits the generalizability of our findings.

5- CONCLUSION

Serum chemerin and LAP were significantly higher in obese children ($p<0.01$). LAP had 95.2% sensitivity and 70.5% specificity at a cut-off point > 41, while serum chemerin at a cut-off point of > 271.7 ng/dl showed an 85.4% sensitivity and 51.4%% specificity for prediction of liver steatosis in our obese participants. Significant correlations were observed between chemerin levels with AST, ALT, GGT, HDL, and insulin levels, and HOMA-IR, while LAP was significantly correlated with BMI, WC, ALT, AST, GGT, TC, TG, LDL, HDL, FBS, insulin levels, HOMA-IR and, serum chemerin. We concluded that serum chemerin may be considered as an acceptable indicator of NAFLD in obese children but LAP is a more available, easy and inexpensive tool to predict NAFLD in those children.

6- ABBREVIATIONS

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, FPG: Fasting plasma glucose, GGT: Gamma-glutamyl transferase, HDL: High density lipoprotein, HOMA-IR: Homeostatic model assessment of insulin resistance, LAP: lipid accumulation product, LDL: Low density lipoprotein.

7- CONFLICT OF INTEREST: None.

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