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Anthropometric Measures as Predictors of Non-Alcoholic Fatty Liver Disease in Adult Asymptomatic Egyptians

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ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease involving about 25% of the world's population. Several studies investigated the role of the different anthropometric measures in NAFLD diagnosis.

Objective: This study aimed to evaluate the diagnostic performance of the different anthropometric measures as non-invasive predictors for the presence of steatosis in a series of NAFLD patients.

Patients and methods: A cross-sectional study was conducted in a series of adult asymptomatic subjects. NAFLD was diagnosed in 100 cases by ultrasonography for whom controlled attenuation parameters (CAP) examination was done. Body mass index (BMI), waist circumference (WCir), waist to height ratio (WHtR), lipid accumulation product (LAP) were measured. Also, subcutaneous and preperitoneal fat were measured using abdominal ultrasound. Roc curve analysis was used to detect the optimal cutoff of different models that predict steatosis.

Results: BMI, WCir, WHtR, LAP, subcutaneous and preperitoneal fat had good diagnostic performance for predicting hepatic steatosis (AUROC for LAP=1 and approaching 1 in all other anthropometric measures).

Conclusion: The clinical anthropometric measures are easy applicable and non-costly promising tools for the prediction of NAFLD in Egyptian patients.

Keywords: NAFLD, Steatosis, Anthropometric, BMI.

INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) is the most common chronic liver disease worldwide affecting about 25% of the general population [(1)]. NAFLD represents a spectrum of histological findings that range from simple increase of intrahepatic lipid content (steatosis, non-alcoholic fatty liver, NAFL) to an inflammatory progressive disease known as non-alcoholic steatohepatitis (NASH). NASH could result in fibrosis, cirrhosis, and subsequently hepatocellular carcinoma (HCC) [(2)]. The increased NAFLD prevalence globally is the result of the rising rates of obesity and diabetes [(3)]. The majority of patients with NAFLD are suffering from obesity [(4)]. Owing to this strong positive association between obesity and NAFLD, it can be expected that the prevalence of NAFLD will increase with the rising rates of obesity [(5)].

Although liver biopsy is the gold standard for diagnosing fatty liver disease and assessment of its severity, it has several limitations such as the invasive nature and the risk of complications including pain, bleeding and infection [(6)]. It is also susceptible to sampling errors and assesses only a small fraction (1/50,000th) of the liver parenchyma [(7)].

A positive correlation was found between NAFLD and Body mass index (BMI) and waist circumference (WCir) which are the most commonly used indicators of obesity[(8)]. Other clinical anthropometric measures including waist-to-height ratio (WHtR) and lipid accumulation product (LAP) were found to be more sensitive and specific for discriminating visceral fat compared to the classic measure as WCir and BMI [(9)]. In this study, we aimed to determine the diagnostic performance of the different anthropometric measures as non-invasive predictors for the presence of steatosis in a series of NAFLD patients.

PATIENTS AND METHODS

This study was a hospital-based cross-sectional, nested case–control study. Participants were selected by simple random sampling from asymptomatic adults aged 18-75 years accompanying patients attending to either the Tropical Medicine and Gastroenterology Outpatient Clinic or the Inpatient Section of the Department, Sohag University Hospital. Another group of 50 subjects with no sonographic evidence for fatty liver were randomly selected as controls.

Exclusion criteria:

Participants aged < 18 years or > 75 years. Those with a diagnosis of liver diseases other than NAFLD or any end-stage liver diseases, including viral hepatitis, drug-induced liver injury, autoimmune liver disease, Wilson's disease, primary biliary cholangitis or any other CLD that might coexist with NAFLD. We also excluded participants with alcohol consumption of ≥ 30 g/day in men or ≥ 20 g/day in women. All included individuals were subjected to a thorough medical history and clinical examination.

The following anthropometric measures were calculated:

BMI was calculated using the following formula: BMI = weight (kg)/ height (m^2) [(10)]. WCir was measured as described by **WHO** [(11)] at a level midway between the lower rib margin and iliac crest with the tape all around the body. WHtR was

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calculated by WCir (cm)/height (cm). Lipid accumulation product (LAP) was calculated as: (WCir [cm] − 65) × (triglyceride concentration [mmol/L]) for men, and (WCir [cm] −58) × (triglyceride concentration [mmol/L]) for women [(12)].

The diagnosis of Metabolic syndrome (MetS) requires the presence of 3 of the following criteria: Fasting Glucose ≥100 mg/dl, WCir >102 cm in men and > 88 cm in women , TG ≥ 150 mg/dl**,** HDL-C < 40 mg/dl in men and < 50 mg/dl in women and blood pressure ≥ 130 (systolic) or ≥ 85 mm Hg (diastolic) [(13)].

Ultrasonographic examination (US):

US was used for screening because of its low cost, noninvasiveness, safety, and absence of radiation exposure. A convex-type transducer of an ultrasound device with 3.5–5-MHz frequency **(Mindray DP-2200)** was used to identify participants with fatty liver.

NAFLD was diagnosed according to the following features: (a) increased liver brightness in contrast to the kidney, (b) impaired or no visualization of portal vein wall, and (c) impaired appearance of the diaphragm [(14)].

The subcutaneous fat thickness and preperitoneal fat thickness were measured according to the procedure described by **Uchibori *et al.*[(15)]** The subcutaneous fat thickness was measured from the skin to linea alba. While the preperitoneal fat thickness was measured from linea alba to the center of the left lobe.

CAP assessment:

All patients had transient elastography examination after overnight fasting and liver stiffness measurement (LSM) and CAP score were obtained using Fibroscan 502 Touch **(Echosens, Paris, France)**. Fibroscan examination was performed by a single operator with either the M or the XL probe, according to the recommendation by the software. Adequate pressure of the probe on the skin surface over the right lobe of the liver through intercostal spaces with the patients in dorsal decubitus with the right arm in maximal abduction. LSM score was represented by the median of 10 measurements and was considered reliable only if at least 10 successful acquisitions were obtained, success rate was ≥ 60% and the IQR-to-median ratio of the 10 acquisitions was ≤ 0.3.

The median optimal cut-off value of CAP for S ≥ S1, S ≥ S2 and S ≥ S3 were 215dB/m, 252dB/m and 296 dB/m respectively [(16)]**.**

Laboratory tests:

After fasting for 8 h overnight, peripheral venous blood sample was collected under complete aseptic conditions for assays of viral hepatitis markers, liver function tests, GGT, complete blood count, lipid profile, fasting blood glucose level and renal function tests.

Ethical approval:

The study protocol was approved by Sohag Faculty of Medicine Ethical Committee. Informed written consent was obtained from all participants. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis

Data were analyzed using STATA version 16.0 (Stata Statistical Software: Release 16.0 College Station, TX: Stata Corp LP) and Med Calc program version 19.1. Quantitative data were represented as mean ± standard deviation, median and range. Data were analyzed using student t-test to compare means of two groups and ANOVA for comparison of the means of three groups or more. When data were not normally distributed Kruskal Wallis test was used for comparison of three or more groups and Mann-Whitney test was used to compare two groups. Nonparametric test for trend across ordered groups was used to compare ordered variables. Qualitative data were presented as number and percentage and compared using either Chi square test or fisher exact test. Roc curve analysis was used to detect the best cut off of different variables that predict steatosis. Sensitivity, specificity, positive predicted values and negative predictive values were also calculated. Graphs were produced by using Excel or STATA program. P value was considered statistically significant if it was ≤ 0.05.

RESULTS

Our study included 100 patients diagnosed to have NAFLD by abdominal ultrasound (41 males and 59 females) with mean age of 45.76 ± 11.01 years, for whom fibroscan was done for assessment of hepatic steatosis. Another group of 50 subjects with no sonographic evidence of fatty liver were randomly selected as controls (32 females and 18 males) their mean age was 36.64 ± 12.47 years.

The basic clinical characteristics and anthropometric measures of the studied groups are summarized in table (1). Patients with NAFLD showed statistically significant older age (P < 0.0001). The body mass index, waist circumference, LAP, waist to height ratio, both subcutaneous and preperitoneal fat showed statistically significant higher values among patients with NAFLD compared to those without NAFLD (P < 0.0001). NAFLD prevalence among patients with the different grades of obesity was 78%, which was higher than that in overweight patients (16%). While its prevalence was about (6%) among those with normal BMI**.** Patients with NAFLD had significantly higher prevalence of metabolic syndrome compared to those without NAFLD (P < 0.0001).

Laboratory data of the studied groups are summarized in table (2). Patients with NAFLD showed statistically significant higher levels of GGT (P=0.02),

serum cholesterol, triglycerides, LDL, and VLDL (P < 0.0001for each), and lower levels of HDL (P < 0.0001).

Table (3) showed comparison of the performance of each anthropometric measurement for predicting steatosis. LAP had the highest AUROC of 1 with 100% sensitivity, 100% specificity, 100% PPV and 100 % NPV at a cut off value > 33.2. WCir had the next AUROC of 0.999 with 98% sensitivity, 100% specificity, 100% PPV and 96.8% NPV at a cut off value > 86. WHtR ratio had an AUROC of 0.988 with 98% sensitivity, 100% specificity, 100% PPV and 96.2 % NPV at a cut off value > 0.52.

Subcutaneous fat had an AUROC of 0.964 with 92% sensitivity, 98% specificity, 98.9% PPV and 86 % NPV at a cut off value > 10. Preperitoneal fat had an AUROC of 0.955 with 86% sensitivity, 100% specificity, 100% PPV and 78.1 % NPV at a cut off value > 7.2. The BMI had the least AUROC (0.938) with 86% sensitivity, 100% specificity, 100% PPV and 78.1 % NPV at a cut off value > 28.1. The performance of the studied anthropometric measures was shown in figure (1).

Table (1): Baseline clinical characteristics and anthropometric measures of the studied groups

Characteristic	NAFLD N=100	Non-NAFLD N=50	P value
Age/year Mean ± SD	45.76±11.01	36.64±12.47	<**0.0001**
Gender			
Female	59 (59.00%)	32 (64.00%)	0.56
Male	41 (41.00%)	18 (36.00%)	
DM	24 (24.00%)	12 (24.00%)	1.00
Hypertension	18 (18.00%)	8 (16.00%)	0.76
BMI (kg/m^2) Mean ± SD	34.35±6.20	25.22±1.73	<**0.0001**
WCir (cm) Mean ± SD	112.18±13.16	73.46±6.85	<**0.0001**
WHtR Mean ± SD	0.69±0.13	0.44±0.03	<**0.0001**
Obesity class			
Normal	6 (6.00%)	22 (44.00%)	
Overweight	16 (16.00%)	28 (56.00%)	<**0.0001**
Obesity grade 1	36 (36.00%)	0	
Obesity grade 2	23 (23.00%)	0	
Obesity grade 3	19 (19.00%)	0	
Metabolic syndrome	57 (57.00%)	3 (6.00%)	<**0.0001**
LAP Mean ± SD	113.77±61.90	18.76±7.23	<**0.0001**
Subcutaneous fat (mm) Mean ± SD	16.67±5.46	6.86±1.85	<**0.0001**
Preperitoneal fat (mm) Mean ± SD	12.08±4.58	5.40±0.94	<**0.0001**

BMI, Body mass index; **DM**, diabetes mellitus; **LAP**, lipid accumulation product; **WCir,** Waist circumference; **WHtR**, Waist to height ratio; **SD**, standard deviation.

Table (2): Lab findings of the studied groups

Variable	NAFLD (N=100)	Non-NAFLD (N=50)	P value
ALT(IU/L) Mean ± SD	24.64±4.72	21.24±4.69	0.62
AST(IU/L) Mean ± SD	23.69±1.27	21.78±4.51	0.50
Albumin (g/dl) Mean ± SD	4.29±0.68	4.39±0.33	0.32
Bilirubin (mg/dl) Mean ± SD	0.73±0.16	0.68±0.12	0.33
GGT (IU/L) Mean ± SD	26.45±3.30	17.18±3.54	**0.02**
S. creatinine (mg/dl) Mean ± SD	0.97±0.18	0.79±0.10	0.06
WBCs (10^3/ µl) Mean ± SD	7.09±1.20	8.40±1.98	**0.0005**
Hb (gm/dL) Mean ± SD	12.37±1.82	11.98±1.50	0.20
Platelets (10^3/µl) Mean ± SD	266.11±9.28	279.52±7.64	0.25
Triglycerides (mg/dl) Mean ± SD	192.38±8.38	119.58±9.25	**<0.0001**
Cholesterol (mg/dl) Mean ± SD	205.94±45.26	169.48±13.14	**<0.0001**
HDL (mg/dl) Mean ± SD	39.17±6.60	45.16±7.95	**<0.0001**
LDL (mg/dl) Mean ± SD	120.05±9.81	101.93±13.95	**0.0001**
VLDL (mg/dl) Mean ± SD	37.18±8.69	24±3.95	**<0.0001**
FBG (mg/dl) Mean ± SD	111.52±5.91	102.4±8.90	0.61

AST, Aspartate aminotransferase; **ALT**, Alanine aminotrasferase; **Hb**, Heamoglobin; **WBCs**, white blood cells; **HDL**, High - density lipoprotein; **LDL**, Low-density lipoprotein; **VLDL**, Very low-density lipoprotein; **SD**, standard deviation; **GGT**, Gamma-glutamyl transpeptidase; **FBG**, Fasting blood glucose.

Table (3): Comparison of the performance of each anthropometric measurement of predicting steatosis

Anthropometric measurement	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
BMI	**0.938 (0.887:0.971)**	**>28.1**	**86**	**100**	**100**	**78.1**
WCir	**0.9996 (0.975:1.00)**	**>86**	**98**	**100**	**100**	**96.8**
WHtR.	**0.988 (0.954:0.999)**	**>0.52**	**98**	**100**	**100**	**96.2**
LAP	**1.00 (0.976:1.00)**	**>33.2**	**100**	**100**	**100**	**100**
Sub. Fat	**0.964 (0.920:0.987)**	**>10**	**92**	**98**	**98.9**	**86**
Per. Fat	**0.955 (0.908:0.982)**	**>7.2**	**86**	**100**	**100**	**78.1**

BMI, Body mass index; **LAP**, lipid accumulation product; **WCir,** Waist circumference; **WHtR**, Waist to height ratio; **PPV**, positive predictive value; **NPV**, negative predictive value; **AUROC**, area under the receiver-operator curve.

AUROCs are given with 95% confidence interval (95%CI).

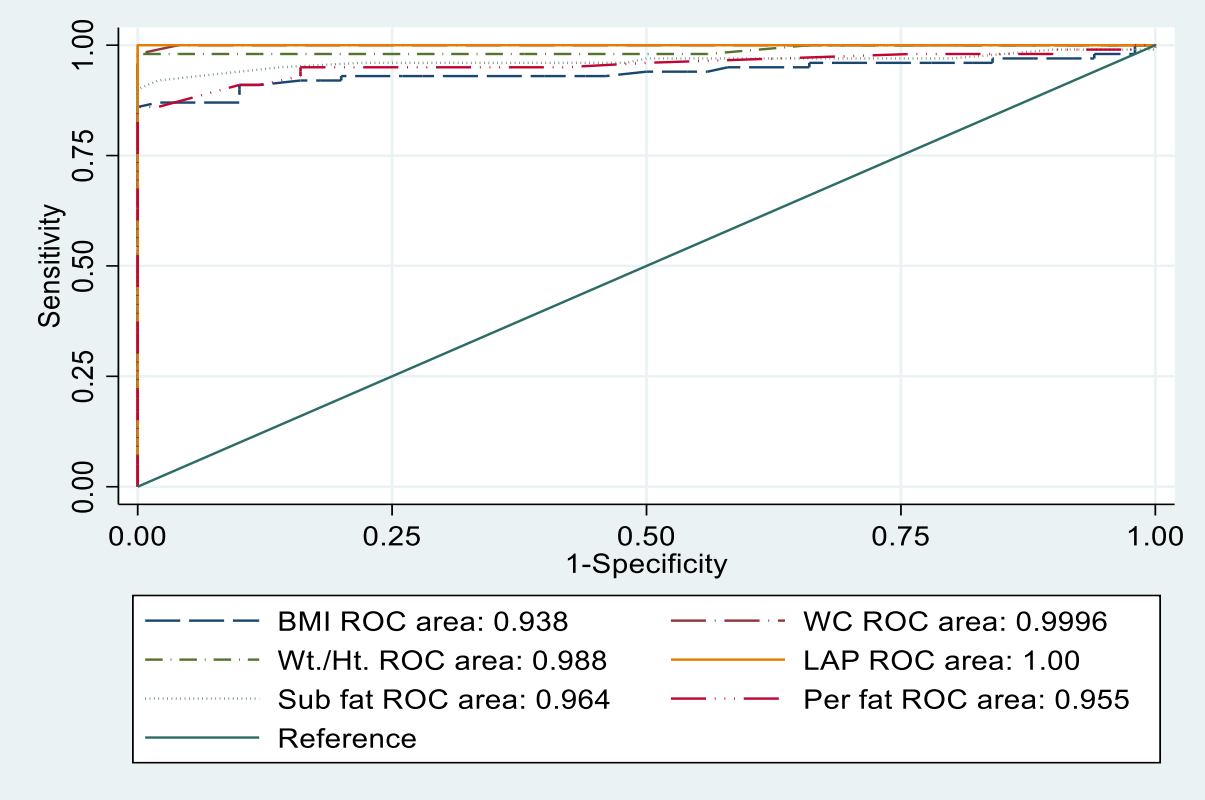

Figure (1): Comparison of the performance of each anthropometric measurement of predicting steatosis

DISCUSSION

Multiple studies confirmed that the increased BMI is associated with NAFLD. Our results showed that BMI was significantly higher in patients with NAFLD than in those without NAFLD [17-23]. In our study, BMI had an AUROC of **0.938** and at a cut off value > 28.1, it detected NAFLD with 100% specificity and a 100% positive predictive value. The BMI excluded NAFLD with **86** % sensitivity and a **78.1**% negative predictive value. According to **Zheng *et al.*** [8] BMI had an AUROC of **0.854** at a cut off value > 24.22 with 64 % specificity and 96 % sensitivity. In Egypt, **Borai *et al.*** [20] reported that BMI had an AUROC of **0.99** at a cut off value > 24.9 with 97.9 % specificity, 100 % sensitivity, 96.9 % positive predictive value and 100% negative predictive value.

Our results showed that WCir was significantly higher in patients with NAFLD than in those without NAFLD. This agrees with **Ju *et al.*** [17], **Motamed *et al.*** [19], **Borai *et al.*** [20], **Dai *et al.*** [21], **Chen *et al.*** [22] and **Cuthbertson *et al.*** [23].

In our study, WCir had an AUROC of **0.9996** and at a cut off value > **86**, it detected NAFLD with 100% specificity and 100% positive predictive value. The WCir excluded NAFLD with **98** % sensitivity and **96.8**% negative predictive value. **Borai *et al.*** [20] reported that WCir had an AUROC of **0.98** at a cut off value > 93 with 97.9 % specificity, 90.3 % sensitivity, 96.9 % positive predictive value and 93.9% negative predictive value.

We found that WHtR was significantly higher in patients with NAFLD than in those without NAFLD. This agrees with **Motamed *et al.*** [19], **Lin *et al.*** [22], **Zhang *et al.*** [24], **Özhan *et al.*** [25] and **Zhang *et al.*** [26]. In our study, WHtR had an AUROC of **0.988** and at a cut off value > **0.52**, it detected NAFLD with 100% specificity and a 100% positive predictive value. It excluded NAFLD with **98** % sensitivity and 96.2% negative predictive value. According to **Motamed *et al.*** [19] the optimal cut-off points for WHtR were 0.533 (sensitivity =82.7%, specificity = 70.8%) for men and 0.58 (sensitivity=83.3%, specificity=71.7%) for women. **Özhan *et al.*** [25], reported that the optimal cut-off level of WHtR for predicting NAFLD in children was 0.62. This value has low sensitivity (48.4 %), but high specificity (73.8%).

Zhang *et al.* [26] reported that the optimal cut off of WHtR was 0.53 (86.3% sensitivity and 55.5% specificity) in males and 0.57 (70% sensitivity and 59.1% specificity) in females. However, this study was performed on elderly patients.

In the current study, LAP was significantly higher in patients with NAFLD than those without NAFLD. Our results agree with **Dai *et al.*** [21], **Cuthbertson *et al.*** [23], **Zhang *et al.*** [26] and **Chiang and Koo** [27].

In our study, LAP had an AUROC of **1.00** and at a cut off value > **33.2**, it detected NAFLD with 100% specificity and a 100% positive predictive value. It excluded NAFLD with **100** % sensitivity and a **100**%

negative predictive value. According to **Dai *et al.* [21]**, areas under the curves (AUC) in men and women were 0.843 and 0.887 respectively with a cut-off values 30.5 (sensitivity: 77% and specificity: 75%) in men and 23.0 (sensitivity: 82% and specificity: 79%) in women. **Zhang *et al.* [26]** reported that the optimal cut off of LAP was 36.15 (79.8% sensitivity and 70.4 % specificity) in males and 49.17 (71% sensitivity and 67% specificity) in females.

Our study revealed that subcutaneous and preperitoneal fat are significantly higher in patients with NAFLD than in those without NAFLD. **Sogabe *et al.* [28]** and **Fukuda *et al.* [29]** also reported the same findings. **Eguchi *et al.* [30]** and **Parente *et al.* [31]** also reported significantly higher preperitoneal fat but not subcutaneous fat in NAFLD patients.

Our study had some limitations that should be taken into consideration. **Firstly,** the relatively small number of patients and controls. **Secondly,** it was performed in one center. **Thirdly,** although abdominal ultrasonography is a good diagnostic tool for NAFLD, it is not useful when fat accumulation is less than 30% of liver volume. Thus, it may underestimate the actual prevalence of NAFLD.

CONCLUSION

The anthropometric measures LAP, WCir, WHtR, BMI, subcutaneous fat and preperitoneal fat thickness are simple to measure and can be applied as screening tools for NAFLD in Egyptian patients. LAP is the most sensitive predictor of NAFLD at an optimal cutoff of 33.2.

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REFERENCES

1. **Younossi Z (2019):** Non-alcoholic fatty liver disease–a global public health perspective. Journal of Hepatology, 70 (3): 531-544.
2. **La Brecque D, Abbas Z, Anania F *et al.* (2014):** World Gastroenterology Organisation global guidelines: Nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Journal of Clinical Gastroenterology, 48 (6): 467-473.
3. **Welsh J, Karpen S, Vos M (2013):** Increasing prevalence of nonalcoholic fatty liver disease among United States adolescents, 1988–1994 to 2007–2010. J Pediatr., 162: 496–500.
4. **Adams L, Lymp J, St Sauver J *et al.* (2005):** The natural history of nonalcoholic fatty liver disease: a population-based cohort study. Gastroenterology, 129: 113-121.
5. **Byrne C, Targher G (2015**): NAFLD: a multisystem disease. J Hepatol., 62 (1): 47–64.
6. **Rockey D, Caldwell S, Goodman Z *et al.* (2009):** Liver biopsy. Hepatology, 49 (3): 1017-1044.
7. **Bonekamp S, Tang A, Mashhood A *et al.* (2014):** Spatial distribution of MRI-determined hepatic proton density fat fraction in adults with nonalcoholic fatty liver disease. Journal of Magnetic Resonance Imaging, 39 (6): 1525-1532.
8. **Zheng R, Chen Z, Chen J *et al.* (2012):** Role of body mass index, waist-to-height and waist-to-hip ratio in prediction of nonalcoholic fatty liver disease. Gastroenterology Research and Practice, 12: 1-6.
9. **Roriz A, Passos L, de Oliveira C *et al.* (2014):** Evaluation of the accuracy of anthropometric clinical indicators of visceral fat in adults and elderly. PLoS One, 9: 1-6.
10. **Keys A, Fidanza F, Karvonen M *et al.* (1972):** Indices of relative weight and obesity. Journal of Chronic Diseases, 25 (6-7): 329-343.
11. **World Health Organization (2000):** Obesity: preventing and managing the global epidemic. Geneva,World Health Organization. http:// www. who. int/ nutrition/ publications/ obesity/ WHO_TRS_894/en.
12. **Kahn H (2005):** The "lipid accumulation product" performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. BMC Cardiovasc Disord., 5: 1-10.
13. **National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) (2002):** Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation, 106 (25): 3143-421.
14. **Shannon A, Alkhouri N, Carter-Kent C *et al.* (2011):** Ultrasonographic quantitative estimation of hepatic steatosis in children with non-alcoholic fatty liver disease (NAFLD). Journal of Pediatric Gastroenterology and Nutrition, 53 (2): 190-195.
15. **Uchibori E, Kadoh K, Nakamura H *et al.* (2011)**: Preperitoneal fat thickness (PFT) of patients undergoing a medical examination assessed by ultrasonography method. Analysis of Biological Samples, 34: 147-150.
16. **de Lédinghen V, Vergniol J, Foucher J *et al.* (2012**): Non-invasive diagnosis of liver steatosis using controlled attenuation parameter (CAP) and transient elastography. Liver International, 32 (6): 911-918.
17. **Ju D, Choe Y, Cho Y *et al.* (2013):** The influence of waist circumference on insulin resistance and nonalcoholic fatty liver disease in apparently healthy Korean adults. Clinical and Molecular Hepatology, 19 (2): 140-145.
18. **Amirkalali B, Poustchi H, Keyvani H *et al.* (2014):** Prevalence of non-alcoholic fatty liver disease and its predictors in north of Iran. Iranian Journal of Public Health, 43 (9): 1275-79.
19. **Motamed N, Rabiee B, Hemasi G *et al.* (2016):** Body Roundness Index and Waist-to-Height Ratio are Strongly Associated with Non-Alcoholic Fatty Liver Disease: A Population-Based Study. Hepatitis Monthly, 16 (9): 575-79.
20. **Borai I, Shaker Y, Kamal M *et al.* (2017):** Evaluation of biomarkers in Egyptian patients with different grades of nonalcoholic fatty liver disease. Journal of Clinical and Translational Hepatology, 5 (2): 109-113.
21. **Dai H, Wang W, Chen R *et al.* (2017):** Lipid accumulation product is a powerful tool to predict non-alcoholic fatty liver disease in Chinese adults. Nutrition & Metabolism, 14 (1): 1-9.

22. **Chen L, Huang J, Chen Q *et al.* (2019):** Validation of fatty liver index and hepatic steatosis index for screening of non -alcoholic fatty liver disease in adults with obstructive sleep apnea hypopnea syndrome. Chin Med J., 132: 2670–2676.
23. **Cuthbertson D, Weickert M, Lythgoe D *et al.* (2014):** External validation of the fatty liver index and lipid accumulation product indices, using 1H-magnetic resonance spectroscopy, to identify hepatic steatosis in healthy controls and obese, insulin-resistant individuals. European Journal of Endocrinology, 171 (5): 561-569.
24. **Zhang X, Wan Y, Zhang S *et al.* (2015):** Nonalcoholic fatty liver disease prevalence in urban school-aged children and adolescents from the Yangtze River delta region: a cross-sectional study. Asia Pac J Clin Nutr., 24 (2): 281-288.
25. **Özhan B, Betül Ersoy B, Özkol M *et al.* (2016):** Waist to height ratio: a simple screening tool for nonalcoholic fatty liver disease in obese children. The Turkish Journal of Pediatrics, 58: 518-523.
26. **Zhang Y, Bo Li, Nan Liu *et al.* (2021):** Evaluation of different anthropometric indicators for screening for nonalcoholic fatty liver disease in elderly individuals. International Journal of Endocrinology, 21: 1-12.
27. **Chiang J, Koo M (2012):** Lipid accumulation product: a simple and accurate index for predicting metabolic syndrome in Taiwanese people aged 50 and over. BMC Cardiovascular Disorders, 12: 1-6.
28. **Sogabe M, Okahisa T, Tsujigami K *et al.* (2014):** Visceral fat predominance is associated with non-alcoholic fatty liver disease in Japanese women with metabolic syndrome. Hepatology Research, 44: 515–522.
29. **Fukuda K, Seki Y, Ichihi M *et al.* (2015):** Usefulness of ultrasonographic estimation of preperitoneal and subcutaneous fat thickness in the diagnosis of nonalcoholic fatty liver disease in diabetic patients. Journal of Medical Ultrasonics, 42: 357-363.
30. **Eguchi Y, Eguchi T, Mizuta T *et al.* (2006):** Visceral fat accumulation and insulin resistance are important factors in non-alcoholic fatty liver disease. J Gastroenterol., 41: 462–469.
31. **Parente D, Oliveira J, Emmanuel P *et al.* (2018):** Preperitoneal fat as a non-invasive marker of increased risk of severe non-alcoholic fatty liver disease in patients with type 2 diabetes. J Gastroenterol and Hepatol., 33: 511–517.