AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 4, Issue 01, 2026 ISSN (E): 2993-2149

Fatty Liver in a Group of Iraqi Children

Dr. Bashar Khaleel Ibrahim
M.B.Ch.B., F.A.B.M.S. (Pediatrics) Iraqi Ministry of Health, Al-Rusafa Health Directorate, Specialized Centre for Endocrine and Diabetes, Baghdad, Iraq

Dr. Liqaa Qahtan Ismael
M.B.Ch.B., HDip. (Family Medicine) Iraqi Ministry of Health, Diyala Health Directorate, Alkhalis PHCC, Diyala, Iraq

Dr. Khalid Jamal Ahmed
M.B.Ch.B., F.A.B.M.S. (Pediatrics) Iraqi Ministry of Health, Diyala Health Directorate, Al-Batoul Teaching Hospital, Diyala, Iraq

Abstract: 1. The fatty liver disease (FLD) among children is increasingly being accepted as a severe comorbidity, which is often linked with metabolic and endocrine pathologies. The aim of the research was to characterize the clinical prognosis of a child group of patients with FLD and, more precisely, to evaluate the correlations between the metabolic control, liver injury biomarkers, and non-invasive fibrosis index.

The study was performed as a cross-sectional study involving 39 children with FLD. The data obtained were demographic, comorbidity, primary diagnosis, and significant laboratory parameters (platelets, ALT, AST, and HbA1c). Also established was the AST to Platelet Ratio Index (APRI) as a non-invasive method for assessing hepatic fibrosis. In order to characterize data statistically, the descriptive statistics and Pearson correlation coefficients were employed to estimate the relationship among the clinical variables (age, BMI, laboratory values, and APRI scores).

The comorbidity burden was also high (46.2 with diabetes being the most common and 41.0 with hypertension being the next most common). The most common first disease diagnosis was T1DM (53.8%). Lab data showed a median of 20 U/L of ALT and 21 U/L of AST, and AST has a great outlier skewness (1011.7 U/L). Mean haemoglobin was 9.08 (±3.61), and mean APRI was 0.32 (±0.45). Correlation analysis showed that there was a close correlation between ALT and APRI ($r = 0.86$). On the contrary, both ALT and APRI had linear relationships with HbA1c.

Pediatric FLD in this group is mostly manifested in T1DM and major metabolic comorbidity. Liver injury severity and predicted risk of fibrosis occurrences, which are vigorously correlated with transaminase levels, do not seem to be closely associated with short-term glycemic regulation, assessed by HbA1c. These results indicate that FLD in children has a multifactorial etiology, and therefore, management should be more extensive than glycemic control, as it should involve overall metabolic and hepatic monitoring.

Keywords: Fatty Liver Disease In Pediatric Patients, Diabetes, Apri, And Hepatic Fibrosis.

2. Introduction

Fatty liver disease is currently recognized as a growing problem in children and adolescents [1]. It is the most common cause of liver disease in childhood, and its increase coincides with the increase in obesity [2]. It is defined by the presence of macro vesicular steatosis in more than 5% of hepatocytes, in the absence of alcohol consumption, drugs, or other pathologies that can produce fatty liver [3]. The severity of the disease varies from simple steatosis, or non-alcoholic fatty liver disease (NAFLD), which is considered to have a good prognosis, to non-alcoholic steatohepatitis (NASH), which can progress to cirrhosis [4]. It is estimated that about 5% of normal-weight or overweight children and about 38% of those with obesity have NAFLD, and a percentage of them will develop NASH. [5]

The risk factors include obesity, insulin resistance (IR), and hypertriglyceridemia. It was like a silent disease, which is often suspected of some finding during the physical examination (overweight, acanthosis nigricans, and hepatomegaly) or an alteration in the laboratory (increased alanine aminotransferase (ALT) and/or aspartate aminotransferase (AST), hypertriglyceridemia). In other instances, they provide diffuse abdominal pain that is the driving force behind the consultation. [6, 7].

NAFLD is regarded as a multifactorial disorder that has a significant genetic factor. It is assumed that the latter and environmental factors play their role in disease development and development [8]. There are also significant changes in the expression levels of many genes related to lipogenesis and inflammation, and certain polymorphisms of the regulatory casings of children and adults with NAFLD that may be implicated. [9]

Clinical, laboratory, and ultrasound findings may be used to suspect the diagnosis, but staging and classification can only be done through the use of a liver biopsy. Considering the invasiveness of it and the potential problems in such miniature patients. [10].

Diet and routine exercise combined with some lifestyle modifications are the only recognized therapies for this entity. The numerous reports suggest that this entity has become a significant problem for public health. [11]

3. Patients & Methods

The study was a cross-sectional design because it aimed at assessing clinical outcomes and the related metabolic profiles in children with fatty liver disease. The sample population included 39 pediatric patients with a diagnosis and managed in a tertiary pediatric hepatology clinic aged 8-17 years old (mean 11.86 + 2.82 years old). The inclusion criteria included (1) age of participants younger than 18 years at the time of diagnosis, (2) the confirmation of fatty liver with the help of abdominal ultrasonography, and (3) at least one year of complete clinical and laboratory history .

Electronic medical records contained the required demographic, anthropometric, and clinical data were systematically read and abstracted with a standardized data abstraction form. The main variables were age, sex, body mass index (BMI), parental ages, and elaborate profiles of comorbidities. The main diagnoses were documented, the most common was Diabetes Mellitus (T1DM) (53.8%). Laboratory parameters related to laboratory hepatic and metabolic functions were obtained at the diagnosis time of diagnosis or at the time of the initial clinical assessment. They were platelet count, alanine aminotransferase (ALT), aspartate aminotransferase (AST), glycated hemoglobin (HbA1c), and uric acid. The AST to Platelet Ratio Index (AST-to-Platelet) is a non-invasive liver fibrosis marker, which was determined by [(AST/upper limit of normal)/Platelet count, 109/L] x 100].

All variables were subjected to descriptive statistics calculation. Mean and standard deviation (SD), median (interquartile range, IQR), and range are used to provide continuous data. A correlation matrix was developed to investigate associations between the most important clinical and biochemical biomarkers. The correlation coefficients computed by Pearson were done on normally distributed variables; otherwise, the rank correlation of Spearman was applied. The

resulting heatmap and the table (Relationships section) describe the strength and direction of associations existing between variables, such as age, platelets, ALT, AST, HbA1c, BMI, APRI, and uric acid. The coefficients (r) above 0.5 were taken as strong, between 0.3 and 0.5 as moderate, and below 0.3 as weak. The statistical analysis was carried out by the SPSS program (version 24.0), and a p-value of less than 0.05 was considered to be statistically significant.

4. Results

The results emphasized the high interaction of the metabolic comorbidities, glycemic control, and liver injury markers in the population, which presents the multifactorial nature of pediatric fatty liver disease. The cohort, as indicated in **Table 1,** was comprised of pediatric patients, to which a mean age was approximately at 11.9 years with a mean BMI of 22.8, which implies that the population had a high degree of variance in adiposity. The distinguishing aspect was that a large percentage (46.2-41.0) of the participants had metabolism comorbidities with diabetes (the most prevalent) and hypertension (the second most common) as the most common being followed by hyperlipidemia (20.5) and obesity (17.9). The fact that the proportion of patients with no comorbidities (7.7) was low supported the notion that fatty liver among children is not common in metabolic isolation. The father and mother ages which were 48.0 and 42.1 years, respectively.

Table 1. Baseline and demographics outcomes of 39 patients who suffer from fatty liver disease in this study.

Variables	Frequency, [N or mean]	Percentage, [% or SD]
Age		
Age, years (Mean ± SD)	11.86	2.82
Father age (Mean ± SD)	48.0	8.36
Mother age, years (Mean ± SD)	42.05	7.08
BMI		
BMI, {kg/m2} (Mean ± SD)	22.8	6.26
Uric Acid scoring		
Uric Acid (mg/dL), (Mean ± SD)	5.2	1.7
Comorbidities		
Hypertension	16	41%
Diabetes	18	46.2%
Obesity	7	17.9%
Hyperlipidemia	8	20.5%
Hypothyroidism	2	5.1%
Arterial Insufficiency	4	10.3%
Wilson's Disease	1	2.6%
Bronchial Asthma	1	2.6%
Hyperthyroidism	1	2.6%
Insulin Resistance	1	2.6%
None	3	7.7%

According to the results of **Table 2**, the most common primary diagnosis was type 1 diabetes mellitus (T1DM) (53.8%), which indicated that there was a highly significant correlation between fatty liver development and the use of insulin therapy, glycemic fluctuation, or weight increase. Obesity per se was 10.2% and different combinations were also possible, e.g., T1DM and hypothyroidism or hyperlipidemia, which depicts the clinical heterogeneity. Other important diseases, such as Wilson's disease and Mauriac syndrome, were also identified, which suggested that fatty liver among this group might sometimes be the result of a specific etiology in need of specific treatment.

Table 2. Enroll the diagnostic features of 39 patients in this cross–sectional study.

Variables	Frequency, {n = 39}	Percentage, %
T1DM	21	53.85
Obesity	4	10.26
Hypothyroidism	3	7.69
GH Deficiency	2	5.13
T2DM Obesity	1	2.56
T1DM/Hyperlipidemia	1	2.56
Short stature/Suspected Turner	1	2.56
Severe Obesity/Mental subnormality	1	2.56
T1DM/Hypothyroidism	1	2.56
Short stature	1	2.56
Obesity/Hypothyroidism	1	2.56
Wilson disease	1	2.56
T1DM/Mauriac syndrome	1	2.56

(Table 3) showed important parameters at the laboratory level, where the mean at ALT (36.4 U/L) and AST (1011.7 U/L) are significantly different, with AST being very high with extreme values (max 36,105 U/L), which will skew the mean. The median AST (21 U/L) is much lower, and probably reflects the central tendency better, indicating that acute hepatic events or confounding conditions (e.g., myopathy, hemolysis) occurred in a subgroup. The number of platelets was normal (325.5 x10-3/ ml), which does not support the advanced cirrhosis in the majority of the patients. The overall glycemic control is poor due to the mean HbA1c of 9.08%. The mean of the APRI score (0.32) gives mild fibrosis, but the range (0.1-2.3) indicates variability.

Table 3. Identifying laboratory outcomes of liver fatty patients.

Variables	mean	std	min	50%	max
Platelets	325.4872	72.64618	159	326	543
ALT	36.36875	64.08182	5	20	310
AST	1011.705	5929.773	8	21	36105
HbA1c	9.079211	3.612006	4.5	8.9	17.9
APRI	0.321622	0.445431	0.1	0.2	2.3

Our results noticed that HbA1c and ALT were linearly correlated **(Figure 1),** suggested that HbA1c and ALT may not be directly related to each other in a simple linear manner, but rather an increase in ALT may be a sign of liver damage, as is common in fatty liver diseases. **(Figure 2)** indicated a relationship between HbA1c and APRI, meaning that the risk of fibrosis, as assessed by APRI, had no firm predictors by glycemic control in this study. Although it does not directly point to fatty liver, a larger APRI might indicate continuous liver damage, which might be related to fat buildup. This observation indicates that alternative study variables, like disease duration, lipid profiles, or genetic predispositions, could be more important in the progression of fibrosis.

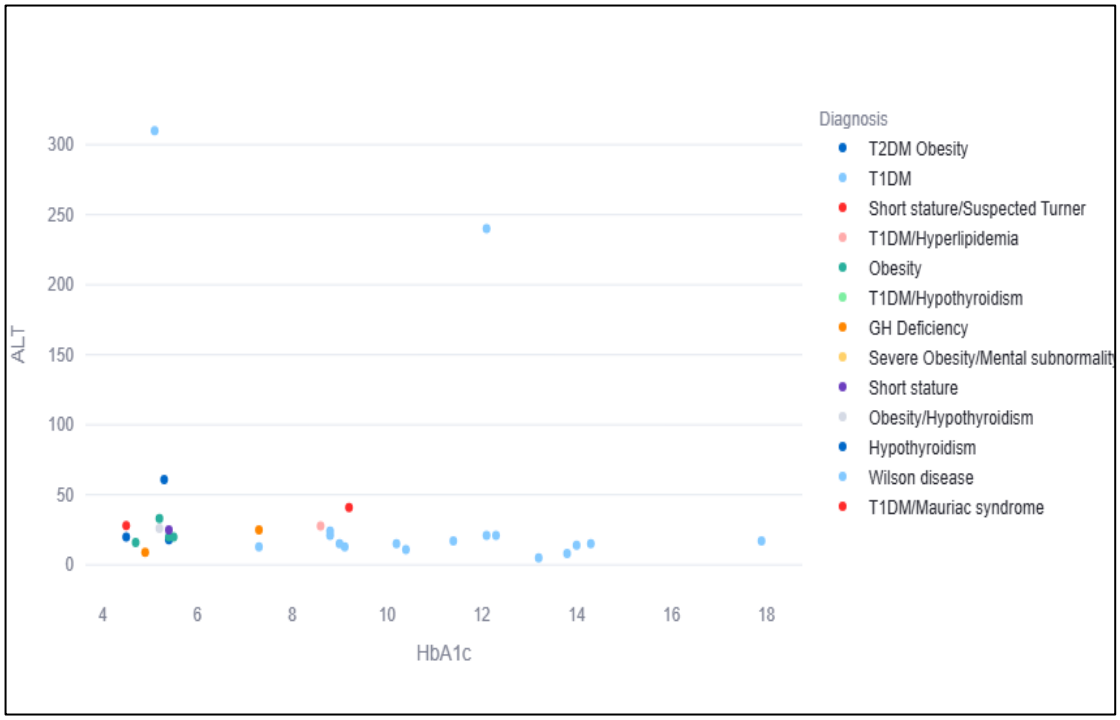

Figure 1. A positive correlation among HbA1c and ALT in patients who are diagnosed with liver fatty,

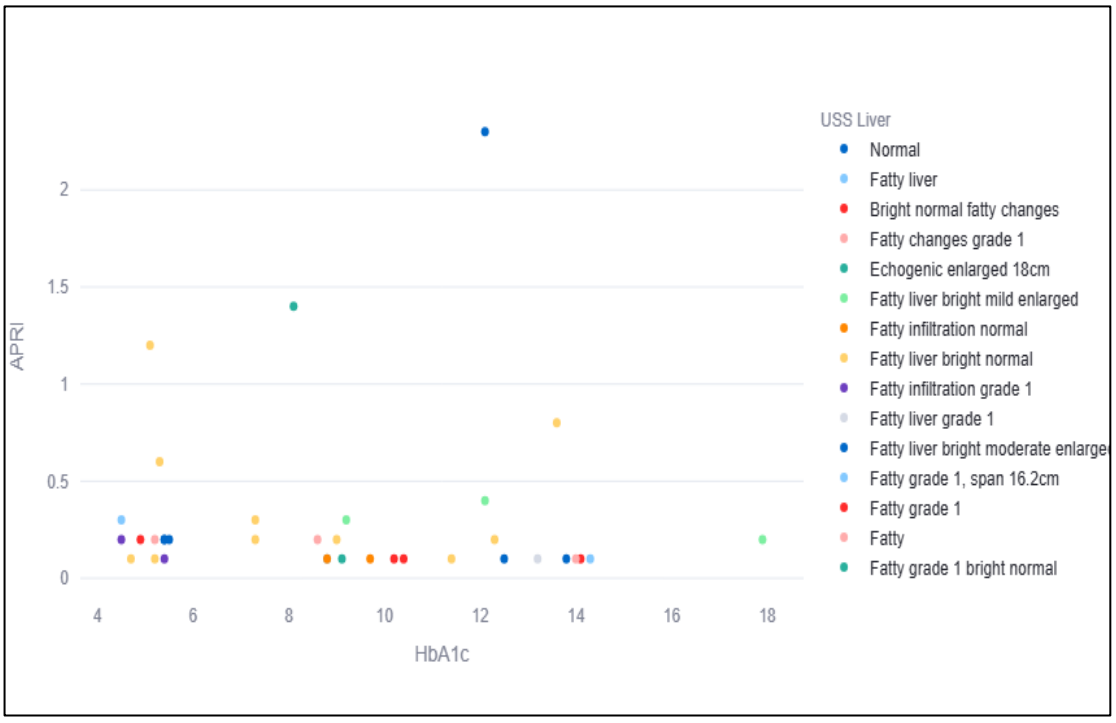

Figure 2. Determining the strong association between HbA1c and APRI in patients who are diagnosed with liver fatty,

(Figure 3) Correlation of clinical variables showed a heatmap of interactions between clinical variables. Both ALT and AST were confirmed as indices of hepatic inflammation as they have a strong positive correlation (r = 0.88). Variations in the level of fibrosis markers ALT were strongly positively correlated with APRI (r = 0.86), which supports the applicability of APRI as a non-invasive fibrosis indicator associated with the elevation of transaminase in these results. The correlation of HbA1c and BMI (r = -0.58) is quite interesting, and this could be due to the fact that lean patients with T1DM were less able to control their glycemia than obese patients with insulin resistance related to obesity. Correlations between age and all parameters in the lab

revealed weak correlations, indicating that in this small age bracket, the severity of the disease is not strongly related to age.

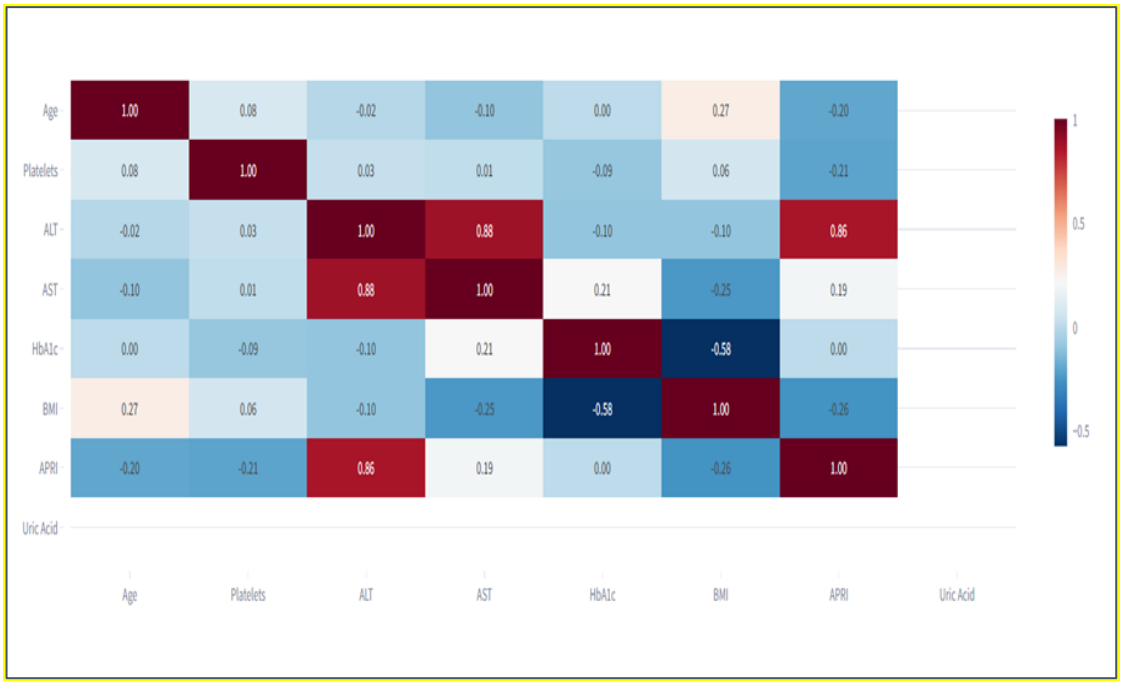

Figure 3. A performance of the correlation heat map among all clinical parameters.

5. Discussion

The cohort population characteristic showed that the average age was around 11.9 years, which falls within the average age of development of the metabolic complications related to pediatric obesity. The standard deviation of 6.3 showed a high degree of variability in adiposity, with the mean BMI measuring 22.8 and the standard deviation of 6.3 showing that there was a high variability in adiposity. Forty-one point zero and forty-six point two percent of the patients had hypertension and diabetes, respectively, and 17.9 percent were classified under obesity as a major illness. This triad reinforced the high syndromic correlation between pediatric fatty liver disease and insulin resistance and is in line with the previous paradigm of NAFLD as the hepatic expression of the metabolic syndrome. This association is further strengthened by the fact that other conditions, such as hyperlipidemia (20.5), are present.

The most common primary diagnosis was Type 1 Diabetes Mellitus (T1DM) (53.8%), which should be interpreted with special attention. Although insulin resistance is also not traditionally associated with T1DM, malglycemic regulation may facilitate hepatic steatosis by processes of overabundant glucose and lipid substrate supply to the liver [12]. Close associations of T1DM with other diseases, such as hyperlipidemia and Mauriac syndrome, in a few patients identify severe, poorly controlled diabetes as an important risk factor of fatty liver among this subgroup of individuals. Such diagnoses as Wilson disease (2.6) are a stern lesson on the significance of the secondary hepatic steatosis being a manifestation of genetic and metabolic disorders.

The average ALT value was slightly high at 36.37 U/L, but the range of the standard deviation (64.08) and the upper limit (310 U/L) indicates the presence of a subgroup with a severe effect on hepatocytes. The AST data, with a mean of 1011.7 U/L grossly distorted by extreme values (max 36,105 U/L), was more impressive because it was indicative of patients with acute hepatitis or other severe complications, as opposed to the normal progression of NAFLD. The average HbA1c of 9.08 percent justified poor glycemic management in a big percentage of the sample.

The positive relationship between ALT and AST (r=0.88) was anticipated, and it proved concomitant injury of the hepatocytes. More to the point, the correlation between ALT and the APRI score was extremely positive (r=0.86), implying that the extent of transaminase-induced

increase in this pediatric sample is strongly dependent on the risk of fibrosis, which is also in line with previous adult literature indicating that permanent increase of ALT is tightly connected with the histological development. The aspect of the negative correlation between HbA1c and BMI (r=-0.58) is interesting and possibly the result of the preponderance of T1DM patients (who are lean) in the high HbA1c stratum, which obscures the normal correlation between HbA1c and BMI in T2DM and obesity [13]. Nevertheless, the prevalence of T1DM in this group is high, which broadens the clinical image of the at-risk pediatric patients [14]. The ALT-APRI correlation provided evidence to support the use of serial ALT measurements as a crude proxy of the risk of fibrosis, which is studied in pediatrics in France. [15]

The accumulation of fat in the hepatocytes in the absence of a considerable amount of alcohol intake was the primary foundation of pediatric patients with fatty liver disease. The disease, NAFLD, arises, according to the research constructed by the USA [16], when there is an excess of liver fat (approximately 5% or more). Some studies had mentioned that fatty liver disease pathogenesis depends on obesity in children, and new studies [17, 18] indicated that in a third of overweight children, hepatic steatosis could be observed.

The presence of parameters that determined liver conditions in NAFLD children, such as ALT and AST, was crucial. Fatty liver was also often observed to be associated with high levels of ALT in children and is a pointer of hepatocellular injury. Chinese studies found out that ALT is a sensitive inflammatory liver marker [19], although not specific due to its multiparous status characteristic and comorbidities [20, 21]. It was proposed in research by Germany [22] that high ALT under obese conditions can be a useful clinical indicator of children who need to be given additional assessment of NAFLD.

6. Conclusion

The cardiometabolic risk factor clusters are extreme in the cohort; almost half of the patients have diabetes, and over 40 percent have hypertension, which directly implicates the syndrome of insulin resistance and its consequences as the main pathophysiology of the disease. The median measures of ALT and AST reflect mild injury; however, the large extreme maximum measures and significant positive correlation between ALT and the APRI fibrosis score (r=0.86) demonstrate that there is a small group of patients with active and progressive liver disease who risk developing hepatic fibrosis. The most important thing is that the correlation of HbA1c with liver damage indicators (ALT, APRI) is important, and it can be assumed that, in the aspect of this group of children, the development of liver diseases may be facilitated by other factors that are not directly related to the degree of glucose level. The risk of liver diseases, including NAFLD, can be suggested by the combination of high levels of HbA1c, ALT, and a high APRI score. All these findings point to the fact that childhood fatty liver disease is a severe comorbidity, which reflects profound underlying metabolic dysregulation and has its unique risk of perpetual hepatic consequences. The specified outcomes, in its turn, would require a paradigm shift in the pediatric practice, which would imply the active and frequent screening of liver functionality and the risk of fibrosis in all children with diabetes, overweight, or dyslipidemia without considering their symptoms to give a chance to intervene and impact the final clinical outcomes.

7. References

1. Schwimmer JB, Dunn W, Norman GJ, Pardee PE, Middleton MS, Kerkar N, et al. SAFETY study: Alanine aminotransferase cutoff values are set too high for reliable detection of pediatric chronic liver disease. Gastroenterology. 2010;138 (4):1357–64. 64 e1–2.
2. Park J, Kim S, Park M. Alanine aminotransferase and metabolic syndrome in adolescents: the Korean National Health and Nutrition Examination Survey Study. Pediatric obesity. 2014;9 (6):411–8.

3. Colantonio D, Kyriakopoulou L, Chan M, Daly C, Brinc D, Venner A, et al. Closing the gaps in pediatric laboratory reference intervals: a CALIPER database of 40 biochemical markers in a healthy and multiethnic population of children. Clin Chem. 2012;58 (5):854–68.

4. Molleston JP, Schwimmer JB, Yates KP, Murray KF, Cummings OW, Lavine JE, et al. Histological abnormalities in children with nonalcoholic fatty liver disease and normal or mildly elevated alanine aminotransferase levels. J Pediatr. 2014;164 (4):707–13 e3.

5. Awai HI, Newton KP, Sirlin CB, Behling C, Schwimmer JB. Evidence and recommendations for imaging liver fat in children, based on a systematic review. Clinical gastroenterology and hepatology: the official clinical practice journal of the American Gastroenterological Association. 2014;12 (5):765–73.

6. Shannon A, Alkhouri N, Carter-Kent C, Monti LD, Devito R, Lopez R, et al. Ultrasonographic Quantitative Estimation of Hepatic Steatosis in Children with Nonalcoholic Fatty Liver Disease (NAFLD) Journal of pediatric gastroenterology and nutrition. 2011;53 (2):190–5.

7. Sasso M, Audiere S, Kemgang A, Gaouar F, Corpechot C, Chazouilleres O, et al. Liver Steatosis Assessed by Controlled Attenuation Parameter (CAP) Measured with the XL Probe of the FibroScan: A Pilot Study Assessing Diagnostic Accuracy. Ultrasound in medicine & biology. 2016;42 (1):92–103.

8. de Ledinghen V, Wong GL, Vergniol J, Chan HL, Hiriart JB, Chan AW, et al. Controlled Attenuation Parameter (CAP) for the diagnosis of steatosis in Non-Alcoholic Fatty Liver Disease. Journal of gastroenterology and hepatology. 2015.

9. Cassinotto C, Lapuyade B, Aït-Ali A, Vergniol J, Gaye D, Foucher J, et al. Liver Fibrosis: Noninvasive Assessment with Acoustic Radiation Force Impulse Elastography—Comparison with FibroScan M and XL Probes and FibroTest in Patients with Chronic Liver Disease. Radiology. 2013;269 (1):283–92.

10. Tang A, Tan J, Sun M, Hamilton G, Bydder M, Gamst AC, et al. Nonalcoholic Fatty Liver Disease: MR Imaging of Liver Proton Density Fat Fraction to Assess Hepatic Steatosis. Radiology. 2013;267 (2):422–31.

11. Schwimmer JB, Middleton MS, Behling C, Newton KP, Awai HI, Paiz MN, et al. Magnetic resonance imaging and liver histology as biomarkers of hepatic steatosis in children with nonalcoholic fatty liver disease. Hepatology. 2014

12. Rockey DC, Caldwell SH, Goodman ZD, Nelson RC, Smith AD, American Association for the Study of Liver Diseases, Liver biopsy Hepatology. 2009;49 (3):1017–44.

13. Schwimmer JB, Newton KP, Awai HI, Choi LJ, Garcia MA, Ellis LL, et al. Paediatric gastroenterology evaluation of overweight and obese children referred from primary care for suspected non-alcoholic fatty liver disease. Alimentary pharmacology & therapeutics. 2013;38 (10):1267–77.

14. Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, et al. The diagnosis and management of non-alcoholic fatty liver disease: Practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology. 2012;55 (6):2005–23.

15. Koot B, Van der Baan-Slootweg OH, Bohte A, Nederveen AJ, Van Werven JR, Tamminga-Smeulders CL, et al. Accuracy of prediction scores and novel biomarkers for predicting nonalcoholic fatty liver disease in obese children. Obesity. 2013;21 (3):583–90.

16. Carter-Kent C, Brunt EM, Yerian L, Alkhouri N, Angulo P, Kohli R, et al. Relations of steatosis type, grade, and zonality to histological features in pediatric nonalcoholic fatty liver disease. Journal of pediatric gastroenterology and nutrition. 2011;52 (2):190–7.

17. Alkhouri N, Eng K, Lopez R, Nobili V. Non-high-density lipoprotein cholesterol (non-HDL-C) levels in children with nonalcoholic fatty liver disease (NAFLD) Springerplus. 2014;5 (3) doi: 10.1186/2193-1801-3-407.

18. Ballestri S, Lonardo A, Bonapace S, Byrne CD, Loria P, Targher G. Risk of cardiovascular, cardiac and arrhythmic complications in patients with non-alcoholic fatty liver disease. World Journal of Gastroenterology. 2014;20 (7):1724–45.

19. Xanthakos S, Jenkins T, Kleiner D, Boyce T, Mourya R, Karns R, et al. High Prevalence of Nonalcoholic Fatty Liver Disease in Adolescents Undergoing Bariatric Surgery. Gastroenterology. 2015;149 (3):623–34.

20. Sundaram SS, Sokol RJ, Capocelli KE, Pan Z, Sullivan JS, Robbins K, et al. Obstructive sleep apnea and hypoxemia are associated with advanced liver histology in pediatric nonalcoholic fatty liver disease. J Pediatr. 2014;164 (4):699–706 e1.

21. Singh G, Vitola BE, Holland M, Sekarski T, Patterson B, Magkos F, et al. Alterations in ventricular structure and function in obese adolescents with non-alcoholic fatty liver disease. J Pediatr. 2013;162 (6):1160–8.

22. Siddiqui MS, Fuchs M, Idowu MO, Luketic VA, Boyett S, Sargeant C, et al. Severity of Nonalcoholic Fatty Liver Disease and Progression to Cirrhosis are associated with Atherogenic Lipoprotein Profile. Clinical Gastroenterology and Hepatology. 2015;13 (5):1000–8.