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# The effect of obesity on FibroScan parameters, cytokeratin-18, and fibroblast growth factor-21 levels

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## ABSTRACT

**Objectives:** This study aimed to evaluate the differences in anthropometric, clinical, laboratory, and radiological parameters between obese and non-obese adolescents, focusing on the role of FibroScan parameters, Cytokeratin-18 (CK-18), and Fibroblast growth factor (FGF-21) in assessing metabolic and liver health.

**Methods:** Anthropometric data were collected, and blood pressure was measured. Laboratory parameters, including fasting glucose, insulin, HOMA-IR, liver enzymes, lipids, CK-18, and FGF-21 levels, were assessed. Liver stiffness measurement (LSM) and controlled attenuation parameter (CAP) were measured using Fibroscan. Pearson's correlation analyses were performed to identify associations between CK-18/FGF-21 levels and metabolic parameters.

**Results:** A cross-sectional study involved 193 adolescents aged 10-18, including 87 obese and 106 non-obese participants. Obese adolescents had significantly higher fasting insulin, HOMA-IR, AST, ALT, GGT, uric acid, triglycerides, and LDL-cholesterol, with lower HDL-cholesterol levels (all P<0.001). CK-18 (P=0.05) and FGF-21 levels (P=0.002) were elevated in the obese group. CAP and LSM values were also significantly higher in obese participants (P<0.001). CK-18 and FGF-21 were positively correlated with fasting insulin, HOMA-IR, GGT, and triglycerides, indicating their potential as biomarkers for metabolic dysfunction. LSM correlated significantly with CK-18 (P=0.005) and FGF-21 (P=0.007).

**Conclusions:** Obese adolescents exhibited significant metabolic and liver dysfunction. Elevated CK-18 and FGF-21 levels, along with abnormal FibroScan parameters, highlight the importance of these biomarkers in identifying early liver injury and metabolic abnormalities. These findings suggest that CK-18 and FGF-21 may be valuable non-invasive tools for assessing and managing obesity-related liver disease.

**Keywords:** Adolescent obesity, non-alcoholic fatty liver disease (NAFLD), FibroScan, cytokeratin-18 (CK-18), fibroblast growth factor-21 (FGF-21)

Adolescent obesity has emerged as a significant public health concern globally, with its prevalence rising at an alarming rate. This condition is not merely a cosmetic issue but a serious health problem associated with an increased risk of various comorbidities, including type 2 diabetes, cardiovascular diseases, and non-alcoholic fatty liver disease (NAFLD) [1] Among the complications of obesity, NAFLD stands out due to its potential to progress to more severe liver conditions such as non-alcoholic

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steatohepatitis (NASH), fibrosis, and cirrhosis [2, 3]. Early detection and assessment of liver health in obese adolescents are therefore crucial to prevent the escalation of liver damage.

FibroScan®, a non-invasive imaging technique, has gained attention for its utility in assessing liver stiffness and quantifying liver fat content, providing valuable insights into hepatic steatosis and fibrosis [4, 5] This technology offers a safer and more patient-friendly alternative to liver biopsy, making it particularly suitable for use in the pediatric population. However, while FibroScan® provides a clear picture of liver structure, biochemical markers such as CK-18 and FGF-21 offer a window into the underlying metabolic and cellular processes associated with liver health and obesity.

Cytokeratin-18 (CK-18) fragments, released during hepatocyte apoptosis, serve as a promising biomarker for detecting and monitoring liver cell injury and necrosis, particularly in the context of NASH [6]. Fibroblast Growth Factor-21 (FGF-21), a hormone involved in metabolic regulation, has been implicated in the pathophysiology of obesity and related liver diseases [7]. Elevated levels of FGF-21 have been associated with hepatic steatosis and insulin resistance, highlighting its potential role in identifying adolescents at risk of metabolic and liver complications [8, 9].

Despite the growing recognition of these tools, there remains a paucity of data on their combined application in assessing the health of obese adolescents. This study aims to bridge this gap by evaluating anthropometric, clinical, laboratory, and radiological differences between obese and non-obese adolescents, focusing on the roles of FibroScan parameters, CK-18, and FGF-21. By investigating these parameters and their interrelationships, we seek to enhance our understanding of the metabolic and hepatic alterations associated with adolescent obesity and identify potential biomarkers for early detection and management. This comprehensive analysis provides valuable insights into the complex interplay between obesity, liver health, and metabolic dysfunction in adolescents.

## METHODS

### Study Design and Participants

This cross-sectional study was conducted to evaluate the differences in anthropometric, clinical, laboratory, and radiological parameters between obese and non-obese adolescents and to explore the roles of FibroScan parameters, CK-18, and FGF-21 in this context.

### Inclusion and Exclusion Criteria

Inclusion criteria for the study were adolescents aged 10-18 years classified as either obese or of normal weight based on BMI percentiles. Exclusion criteria included any chronic disease, acute infection, or use of medications that could affect metabolic parameters.

### Anthropometric and Clinical Measurements

Anthropometric measurements, including height, weight, and waist circumference, were taken using standardized procedures. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m$^2$). Blood pressure was measured using a calibrated sphygmomanometer, and systolic and diastolic pressures were recorded.

### Laboratory Assessments

Blood samples were collected after an overnight fast. The following laboratory parameters were measured: fasting glucose, fasting insulin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), uric acid, total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, ferritin, CK-18, and FGF-21. The homeostasis model assessment for insulin resistance (HOMA-IR) was calculated using the formula: [fasting insulin (μIU/mL) × fasting glucose (mg/dL)] / 405.

### Assessment of the Liver Fibrosis

FibroScan (Echosens, Paris, France) assessed liver stiffness and hepatic steatosis. The controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) were recorded. CAP values are expressed in decibels per meter (dB/m) and LSM values in kilopascals (kPa).

### Ethical Considerations

The Institutional Review Board (IRB) of the Ethics Committee of Konya Karatay University Faculty of Medicine (2021/19) approved the study protocol. Written informed consent was obtained from all participants and their guardians before enrollment.

### Statistical Analysis

All statistical analyses were performed using SPSS version 21.0 (IBM Corp., Armonk, NY, USA). Data normality was confirmed using the Kolmogorov-Smirnov test. Continuous variables were expressed as mean ± standard deviation (SD) or median (25th–75th percentile) as appropriate. Independent samples t-test was used for normally distributed data, while the Mann-Whitney U test was employed for non-normally distributed data. Categorical variables were compared using the Chi-square test.

Correlation analyses between various parameters and CK-18/FGF-21 levels were conducted using Pearson's correlation coefficient (r). Significant correlations were considered those with P<0.05. Receiver operating characteristic (ROC) curves were generated to determine the cut-off values for CK-18 and FGF-21 in distinguishing obese from non-obese adolescents. The area under the curve (AUC), sensitivity, and specificity were calculated.

## RESULTS

The study included 193 participants aged 10-18 recruited from a pediatric outpatient clinic. Of these, 87 were classified as obese based on their body mass index (BMI) percentiles for age and sex, and 106 were classified as controls with normal BMI percentiles.

The comparison of anthropometric and clinical parameters between the obese and control groups is detailed in Table 1. There was no significant difference in age between the groups (14.34±2.64 vs. 14.32±2.49 years, P=0.85). The proportion of females was also similar between the groups (60% in control vs. 55% in obese, P=0.46). Significant differences in waist circumference and blood pressure were observed. The obese group had a greater waist circumference (100.80±16.41 cm vs. 69.23±7.25 cm, P<0.001). The systolic and diastolic blood pressures were also significantly elevated in the obese group (118.22±11.56 mmHg vs. 108.87±9.13 mmHg, P<0.001 for systolic; 75.47±9.35 mmHg vs. 68.77±7.52 mmHg, P<0.001 for diastolic).

The laboratory parameters are summarized in Table 2. Obese adolescents had significantly higher fasting insulin (P<0.001), HOMA-IR (P<0.001), liver enzymes (AST, ALT, GGT; all P<0.001), uric acid (P<0.001), and unfavorable lipid profiles, including higher triglycerides (P<0.001) and LDL-cholesterol

**Table 1. Comparison of anthropometric and clinical parameters of obese and control groups**

| | Control group (n=106) | Obese group (n=87) | P value |
|---|---|---|---|
| **Age (year)** | 14.34±2.64 | 14.32±2.49 | 0.85± |
| **Female, n (%)** | 64 (60) | 48 (55) | 0.46¶ |
| **Body weight (kg)** | 49.0±10.87 | 80.68±20.11 | **<0.001**‡ |
| **Body weight sd** | -0.38±0.82 | 2.58±1.22 | **<0.001**‡ |
| **Height (cm)** | 157.47±11.89 | 161.07±10.99 | **0.02**± |
| **Height sd** | -0.07±0.93 | 0.35±1.16 | **0.005**‡ |
| **BMI (kg/m$^2$)** | 19.45±2.32 | 30.69±5.48 | **<0.001**± |
| **BMI sd** | -0.40±0.79 | 2.43±0.81 | **<0.001**± |
| **Waist circumference (cm)** | 69.23±7.25 | 100.80±16.41 | **<0.001**‡ |
| **Systolic blood pressure (mm/Hg)** | 108.87±9.13 | 118.22±11.56 | **<0.001**± |
| **Systolic blood pressure sd** | 0.1±0.90 | 0.83±0.88 | **<0.001**± |
| **Diastolic blood pressure (mm/Hg)** | 68.77±7.52 | 75.47±9.35 | **<0.001**± |
| **Diastolic blood pressure sd** | 0.45±0.76 | 1.03±0.77 | **<0.001**± |

Data are shown as mean±standard deviation or n (%).BMI=body mass index, sd=standard deviation
Chi-square test ±Mann-Whitney U ‡Independent samples t-test

**Table 2.** Laboratory parameters of groups

| | Control group (n=106) | Obese group (n=87) | P value |
|---|---|---|---|
| **Fasting glucose (mg/dL)** | 85.82±7.86 | 87.10±7.37 | 0.24‡ |
| **Fasting insulin (µIU/mL)** | 11.81±5.73 | 31.31±19.35 | **<0.001** ± |
| **HOMA-IR** | 2.52±1.33 | 6.85±4.58 | **<0.001** ± |
| **AST (U/L)** | 17.42±4.25 | 24.83±13.78 | **<0.001** ± |
| **ALT (U/L)** | 12.862±6.85 | 38.66±36.77 | **<0.001** ± |
| **GGT (U/L)** | 10.90±3.70 | 22.30±14.78 | **<0.001** ± |
| **Uric acid (mg/dl)** | 4.03±0.96 | 5.48±1.55 | **<0.001** ± |
| **Total cholesterol (mg/dL)** | 143.97±30.27 | 162.57±29.87 | **<0.001** ± |
| **Triglycerides (mg/dL)** | 80.10±33.53 | 142.93±60.07 | **<0.001** ± |
| **HDL-cholesterol (mg/dL)** | 54.72±13.61 | 45.37±10.75 | **<0.001** ± |
| **LDL-cholesterol (mg/dL)** | 76.11±24.61 | 87.73±25.90 | **<0.001** ± |
| **Ferritin (ng/mL)** | 30.77±20.08 | 48.92±31.98 | **<0.001**± |
| **CK-18 (ng/mL)** | 0.63±0.72<br>0.46 (0.22-0.70) | 1.06±2.01<br>0.56 (0.29-1.07) | 0.05± |
| **FGF-21 (pg/mL)** | 7.29±14.81<br>3.05 (0.7-9.67) | 16.42±29.5<br>6.2 (2.2-16.4) | **0.002**± |

Data are shown as mean±standard deviation or median (25%-75%). CK-18=cytokeratin-18, FGF-21=fibroblast growth factor-21, AST=aspartate aminotransaminase, ALT=alanine aminotransaminase, GTT=gamma-glutamyl transferase, HDL=high-density lipoprotein, LDL=low-density lipoprotein, HOMA-IR=Homeostasis model assessment insulin resistance index

±Mann-Whitney U, ‡Independent samples t-test

(P<0.001), and lower HDL-cholesterol (P<0.001). CK-18 levels were elevated in the obese group (0.56 vs. 0.46 ng/ml, P=0.05), as were FGF-21 levels (6.2 vs. 3.05 pg/mL, P=0.002).

Table 3 presents the Fibroscan parameters. The controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) were significantly higher in the obese group. The CAP values were 274.40±55.41 dB/m compared to 198.08±30.60 dB/m in the control group (P<0.001). Similarly, the LSM values were elevated in the obese group (5.40±1.50 kPa vs. 4.13±0.71 kPa, P<0.001).

The correlation analyses between anthropometric parameters and FGF-21/CK 18 levels are depicted in Table 4. There was a significant positive correlation between body weight standard deviation and FGF-21

**Table 3.** Radiological parameters of groups

| | Control group (n=106) | Obese group (n=87) | P value |
|---|---|---|---|
| **CAP (dB/m)** | 198.08±30.60<br>195.5 (180.5-223) | 274.40±55.41<br>270 (237-312) | **<0.001** ‡ |
| **LSM (kPa)** | 4.13±0.71<br>4.1 (3.6-4.6) | 5.40±1.50<br>5 (4.4-6.1) | **<0.001** ± |

Data are shown as mean±standard deviation or median (25%-75%), CAP=controlled attenuation parameter. LSM=liver stiffness measurement

‡Independent samples t-test, ±Mann-Whitney U

**Table 4. Correlation analyses between anthropometric parameters and FGF-21/CK 18 levels in study groups**

| | CK 18 | | FGF-21 | |
|---|---|---|---|---|
| **Parameters** | **r** | **P value*** | **r** | **P value*** |
| Age | -0.04 | 0.55 | -0.07 | 0.35 |
| Body weight | 0.10 | 0.18 | 0.12 | 0.09 |
| Body weight sd | 0.11 | 0.14 | 0.15 | **0.03** |
| Height | 0.07 | 0.32 | 0.06 | 0.36 |
| Height sd | 0.11 | 0.13 | 0.12 | 0.11 |
| BMI | 0.08 | 0.25 | 0.13 | 0.08 |
| BMI sd | 0.09 | 0.21 | 0.15 | **0.04** |
| Waist circumference | 0.10 | 0.17 | 0.16 | **0.02** |

CK-18=cytokeratin-18, FGF-21=fibroblast growth factor-21, BMI=body mass index
*Pearson correlation

**Table 5. Correlation analyses between laboratory parameters and FGF-21/CK 18 levels in study groups**

| | CK 18 | | FGF--1 | |
|---|---|---|---|---|
| **Parameters** | **r** | **P value*** | **r** | **P value*** |
| Fasting glucose | 0.14 | 0.05 | 0.19 | **0.01** |
| Fasting insulin | 0.24 | **0.001** | 0.22 | **0.003** |
| HOMA-IR | 0.26 | **<0.001** | 0.24 | **0.001** |
| AST | 0.68 | 0.37 | 0.41 | 0.58 |
| ALT | 0.11 | 0.12 | 0.09 | 0.22 |
| GGT | 0.15 | **0.03** | 0.15 | **0.04** |
| Uric acid | 0.15 | **0.04** | 0.19 | **0.009** |
| Total cholesterol | -0.03 | 0.67 | 0.04 | 0.60 |
| Triglycerides | 0.16 | **0.03** | 0.233 | **0.002** |
| HDL-cholesterol | -0.05 | 0.48 | 0.003 | 0.97 |
| LDL-cholesterol | -0.80 | 0.29 | -0.14 | 0.06 |
| Ferritin | 0.12 | 0.09 | 0.14 | 0.05 |

CK-18=cytokeratin-18, FGF-21=fibroblast growth factor-21, AST=aspartate aminotransaminase, ALT=alanine aminotransaminase, GTT=gamma-glutamyl transferase, HDL=high-density lipoprotein, LDL=low-density lipoprotein
*Pearson correlation, in multivariate regression analysis of correlated parameters, no independent risk factor effect could be demonstrated.

**Table 6. Correlation analyses between CAP/LSM and FGF-21/CK 18 levels in study groups**

| | CK 18 | | FGF-21 | |
|---|---|---|---|---|
| **Parameters** | **r** | **P-value*** | **r** | **P-value*** |
| CAP | 0.12 | 0.10 | 0.11 | 0.13 |
| LSM | 0.21 | **0.005** | 0.20 | **0.007** |

CK-18=cytokeratin-18, FGF-21=fibroblast growth factor-21, CAP=controlled attenuation parameter, LSM=liver stiffness measurement
*Pearson correlation

**Table 7. CK18 and FGF-21 cut-off values of obese and control group**

| Variables | Cutoff | Sensitivity (95% CI) | Specificity (95% CI) | AUC (95% CI) | P value |
|---|---|---|---|---|---|
| **CK-18 (ng/mL)** | 0.53 | 52.7 (40.7-64.7) | 60.0 (49.7-69.7) | 0.588 (0.510-0.662) | **0.04** |
| **FGF-21 (pg/mL)** | 2 | 75.9 (65.3-84.6) | 46.6 (36.1-57.5) | 0.638 (0.562-0.710) | **0.001** |

CK-18=cytokeratin-18, FGF-21=fibroblast growth factor-21

levels (r=0.15, p=0.03) and between BMI standard deviation and FGF-21 levels (r=0.15, P=0.04). Additionally, waist circumference was positively correlated with FGF-21 levels (r=0.16, P=0.02).

CK-18 showed significant positive correlations with fasting insulin (r=0.24, p=0.001), HOMA-IR (r=0.26, P<0.001), GGT (r=0.15, P=0.03), uric acid (r=0.15, P=0.04), and triglycerides (r=0.16, P=0.03), indicating its association with insulin resistance and liver enzyme activity (Table 5).

Similarly, FGF-21 was positively correlated with fasting glucose (r=0.19, P=0.01), fasting insulin (r=0.22, P=0.003), HOMA-IR (r=0.24, P=0.001), GGT (r=0.15, P=0.04), uric acid (r=0.19, P=0.009), and triglycerides (r=0.23, P=0.002), highlighting its involvement in metabolic dysfunction and lipid abnormalities (Table 5).

Liver stiffness measurement (LSM) was positively correlated with both CK-18 (r=0.21, P=0.005) and FGF-21 (r=0.20, P=0.007). However, the controlled attenuation parameter (CAP) did not significantly correlate with CK-18 or FGF-21 levels (Table 6).

The cut-off values for CK-18 and FGF-21 for distinguishing between obese and control groups are pre-

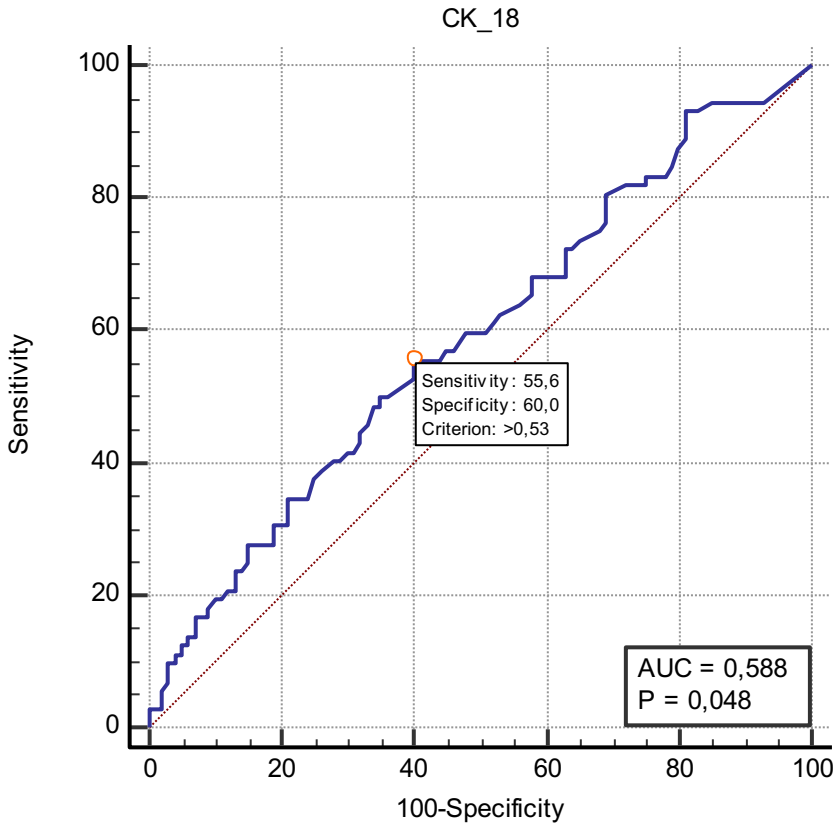


**Fig. 1. ROC curve. CK 18 cut-off value according to the study and control group.**

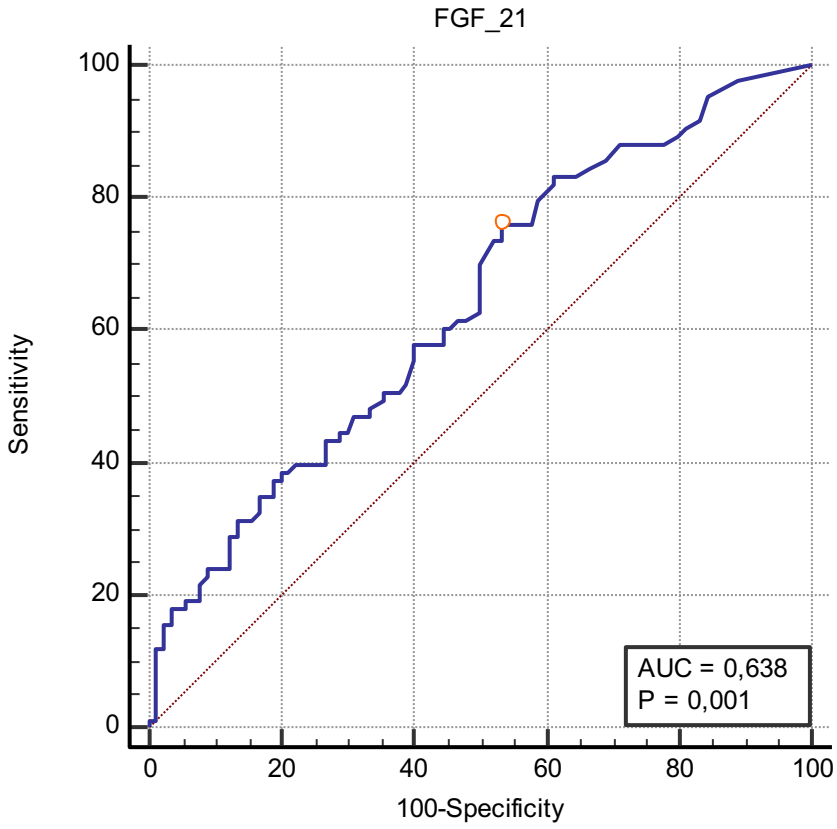


**Fig. 2. ROC curve. FGF-21 cut-off value according to the study and control group.**

sented in Table 7. The optimal cut-off value for CK-18 was 0.53 ng/mL, with a sensitivity of 52.7% and specificity of 60.0% (AUC=0.588, P=0.04) (Fig. 1). For FGF-21, the cut-off value was 2 pg/mL, with a sensitivity of 75.9% and specificity of 46.6% (AUC=0.638, P=0.001)(Fig. 2).

## DISCUSSION

This study revealed several significant differences between obese and non-obese adolescents across various parameters. Obese adolescents exhibited significantly higher waist circumference compared to their non-obese counterparts. Clinically, they also had elevated systolic and diastolic blood pressures. Laboratory results indicated that obese adolescents had higher fasting insulin levels, HOMA-IR, AST, ALT, GGT, uric acid, total cholesterol, triglycerides, and LDL-cholesterol levels, while HDL-cholesterol levels were lower. Radiologically, FibroScan assessments showed significantly higher CAP and LSM values in the obese group, indicating more significant hepatic steatosis and stiffness. Additionally, CK-18 and FGF-21 levels were significantly elevated in obese adolescents, with positive correlations between these biomarkers and various metabolic and anthropometric parameters.

Numerous studies have demonstrated the correlation between FGF-21 levels, which are involved in metabolic control, and obesity. Several investigations indicate that serum FGF-21 levels are markedly elevated in obese children relative to their lean counterparts [9, 10]. A study by Baek *et al*. [11] revealed that serum FGF-21 levels were elevated in obese children with metabolic syndrome compared to their counterparts without metabolic syndrome. In a comparable study, Li *et al*. [12] demonstrated that circulating FGF-21 levels were elevated in obese children. A recent study by El-Masry *et al*. [13] indicated that serum FGF-21 levels were significantly elevated in obese children; however, it could not serve as a reliable predictor for metabolic disorders, including waist circumference, fasting blood glucose, insulin, HOMA-IR, and lipid profile in prepubertal obese children. The

study revealed that FGF-21 levels were markedly elevated in obese adolescents relative to the control group. This result aligned with the prior investigations [9-11]. Moreover, substantial positive correlations were identified between FGF-21 levels and anthropometric metrics such as body weight standard deviation, BMI standard deviation, and waist circumference, as well as laboratory results including fasting blood glucose, GGT, insulin, HOMA-IR, uric acid, and triglyceride levels. In contrast to our study, El-Masry *et al*. [13] identified a negative association between serum FGF-21 and BMI in obese children, with no significant correlations observed with fasting blood glucose, insulin, HOMA-IR, or lipid profile. Furthermore, whereas a positive association was identified between blood FGF-21 and HDL levels in the aforementioned study, our analysis discovered no significant correlation between these two parameters. We examined the connection between serum FGF-21 levels and liver CAP and LSM values obtained by the Fibroscan equipment. Previous investigations have identified a correlation between elevated FGF-21 levels and obesity and fatty liver disease [14, 15]. Rusli *et al*. [16] demonstrated a strong correlation between serum FGF-21 levels and hepatic fat content in mice with NAFLD. Another study, including adults, revealed that the sensitivity and specificity of FGF-21 for diagnosing NAFLD were 72.6% and 85.1%, respectively, whereas for diagnosing NASH, the sensitivity, and specificity were 53.7% and 71.9%, respectively [17]. Giannini *et al*. [18] revealed a link between serum FGF-21 levels and liver fat accumulation as well as liver damage in obese young individuals. Consequently, FGF-21 has been proposed as a possible biomarker for NAFLD in adults and children [19-22]. Our investigation revealed a substantial positive association between LSM measurements obtained via Fibroscan and serum FGF-21 levels; however, no positive correlation was observed with CAP measurements. The serum FGF-21 cut-off value demonstrated moderate sensitivity but low specificity in differentiating between the non-obese control group and obese adolescents, according to our study.

CK-18 is the major intermediate filament protein in hepatocytes. There is a strong correlation between hepatocyte apoptosis and the release of caspase-coated and uncoated M30 and M65 fragments of cytokeratin-18 and their levels in serum [23]. It was demonstrated in various studies that serum CK-18 levels are elevated in patients compared with simple fatty liver disease. These findings have shown a positive correlation between high CK-18 levels and inflammation, steatosis severity, and liver fibrosis [6, 24, 25]. CK-18 fragments have recently been confirmed as a marker of NASH and even defined as the most promising non-invasive test for the diagnosis and management of NASH in recent NAFLD guidelines [26]. In a study conducted by Lebensztejn *et al*. [27] in children with NALFD, CK-18 levels were significantly higher in children with fibrosis compared to those without fibrosis. In the same study, the sensitivity and specificity in detecting fibrosis using serum CK-18 levels were found to be 79% and 60%, respectively, and it was concluded that it may be appropriate to determine serum CK-18 levels to detect fibrosis in children with NAFLD. Mandelia *et al*. [28] reported that CK-18 level was a promising new biomarker for fibrosis in children with NAFLD. In our study, CK-18 levels were found to be higher in the obese group compared to the control group. Simultaneously, a significant positive correlation was found between LSM levels, indicators of fibrosis, and CK-18 levels. However, the performance of the CK-18 level was insufficient for diagnosing NAFLD due to the low area under the receiver operating characteristics curve, according to our study. Similarly, a study by Koot *et al*. [29] found that novel biomarkers such as FGF-21 and CK-18 and non-invasive predictive scores did not have sufficient diagnostic accuracy to diagnose and exclude NAFLD in severely obese children and adolescents.

## Limitations

One of the limitations of our study is that we did not use the Steatotic Liver Disease (SLD), Metabolic Associated Steatotic Liver Disease (MASLD), and Metabolic Associated Steatohepatitis (MASH) terminology because we did not assess the full range of metabolic parameters required for these classifications. Our focus was limited to identifying fatty liver using non-invasive FibroScan parameters rather than conducting a comprehensive metabolic evaluation. Future studies incorporating detailed metabolic assessments would allow for the application of these standardized terms.

## CONCLUSION

In conclusion, obesity and NAFLD in teens rise gradually, like in all age groups. Early identification and prevention of NAFLD are crucial since certain people can develop NASH and hepatic fibrosis. Liver biopsy, an invasive diagnostic procedure, is not always possible or repeatable in children and adolescents. Thus, reliable and reproducible non-invasive imaging technologies like Fibroscan and serum indicators are essential for disease diagnosis and monitoring. This study found a correlation between serum FGF-21 and CK-18 in obese NAFLD patients and Fibroscan results. However, both serum indicators limited diagnostic sensitivity and specificity. Since hepatic apoptosis and fibrosis had not yet formed, serum CK-18 levels were ineffective in diagnosing adult NAFLD patients. Noninvasive imaging and serum biomarkers would be better for early NAFLD diagnosis and disease progression monitoring in children and adolescents.

*Ethical Statement*

The study received approval from the the Institutional Review Board (IRB) of the Ethics Committee of Konya Karatay University Faculty of Medicine (2021/19). Written informed consent was obtained from all participants and their guardians before enrollment.

*Authors' Contribution*

Study Conception: MK; Study Design: NK; Supervision: MK; Funding: MK; Materials: MK; Data Collection and/or Processing: MK; Statistical Analysis and/or Data Interpretation: NK; Literature Review: NK; Manuscript Preparation: NK; and Critical Review: MK.

*Conflict of interest*

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

*Financing*

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