Original Article

Noninvasive Assessment of Nonalcoholic Fatty Liver Disease in Children with Overweight and Obesity by Transient Elastography

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Abstract

Introduction: Childhood obesity and nonalcoholic fatty liver disease (NAFLD) are emerging as significant health concerns. While liver biopsy remains the gold standard for diagnosis, there is a pressing need for a noninvasive alternative to identify early fibrosis. **Methods:** A cross-sectional investigation was carried out from January 2020 to December 2021 involving overweight and obese children attending the pediatric outpatient department (OPD). The aim is to determine the occurrence of fibrotic and steatotic changes in the liver of overweight and obese children using transient elastography (TE) and to establish correlations between TE results, Pediatric NAFLD Fibrosis Index (PNFI), and other biochemical parameters. TE was utilized to assess both fibrotic and steatosis changes, while ultrasound (USG) was employed to detect steatosis in the liver. **Results:** Two hundred and fifty-nine eligible children participated in the study. Mean age of the study cohort was 10.8 years, with males constituting 63%. Mean Z score for BMI was 1.71 ± 0.57. Fibrosis was detected in 29.3% of children by TE, while steatosis was observed in 27.7% of children. Steatosis was identified in 23.8% of cases through USG. BMI Z score, ALT (Alanine aminotransferase), AST and PNFI score exhibited significant associations with grades of liver fibrosis and steatosis as determined by TE, as well as with grades of steatosis according to USG findings. **Conclusion:** A notable prevalence of increased liver stiffness was observed in overweight and obese children. TE proves to be a valuable tool in identifying fibrotic and steatotic changes in these children, complementing existing noninvasive modalities.

Keywords: Fibroscan, fibrosis, NAFLD, obesity, transient elastography

Introduction

Paediatric nonalcoholic fatty liver disease (NAFLD) is characterized by chronic hepatic steatosis in individuals aged 18 years or younger, not attributable to genetic, metabolic disorders, infections, steatogenic medications, ethanol consumption or malnutrition.[1] Its clinical spectrum ranges from simple steatosis (accumulation of fat in the liver) to steatohepatitis (inflammation and hepatocyte injury) to fibrosis and ultimately cirrhosis.[1,2] The prevalence of NAFLD is estimated to range from 3% to 10% in the general paediatric population, with a pooled mean prevalence of 7.6%.[3] Notably, NAFLD is more prevalent among obese children, affecting 30% to 45% of this demographic, compared to the general population. The prevalence varies across different studies globally, with a pooled mean prevalence outside India estimated at 34.5%.[3] Studies in India report NAFLD prevalence ranging from 50% to 70% among obese and overweight children.[4-7]

Most individuals with NAFLD are asymptomatic, often presenting to medical facilities with nonspecific complaints such as abdominal discomfort or obesity. Biochemical investigations typically reveal evidence of insulin resistance, elevated transaminases, dyslipidemias characterized by elevated triglycerides and total cholesterol, and reduced high-density lipoproteins (HDL).[1] While liver biopsy remains the gold standard for diagnosing NAFLD and fibrosis, its invasive nature limits routine utilization. Presently, ultrasonography (USG) is

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the most commonly employed diagnostic tool for detecting fatty liver, with an approximate sensitivity of 80%, albeit with its reliance on operator expertise and limited ability to detect early changes or fibrosis.[8] Magnetic resonance imaging-proton density fat fraction (MRI-PDFF) boasts superior sensitivity in detecting hepatic fat deposition among noninvasive methods. However, its prolonged testing duration and the requirement for sedation pose significant limitations for its widespread use as a screening tool in paediatric population.[9]

Transient elastography (TE), also known as fibroscan, is a noninvasive method based on ultrasound technology utilized to evaluate tissue stiffness by measuring shear wave velocity. Fibroscan has been employed since 2011 and has undergone validation for detecting fibrosis in adult liver diseases such as alcoholic liver disease, nonalcoholic fatty liver disease (NAFLD) and hepatitis B, among others. However, its application in paediatric populations has not been extensively investigated. The incorporation of controlled attenuation parameter (CAP) alongside fibroscan has expanded its potential in noninvasively and conveniently diagnosing liver disease.[10] The principal aim of this study was to determine the prevalence of fibrotic and steatotic changes in the livers of overweight and obese children using TE. Secondary objectives included establishing correlations between fibroscan results and various anthropometric measurements, biochemical parameters and PNFI in overweight and obese children.

Materials and Methods

A cross-sectional study was conducted in a tertiary care institute in eastern India from January 2020 to December 2022. All children attending the OPD were screened and children of age 8-14 years attending Paediatric OPD and IPD with BMI > 85th centile as per IAP charts were included in the study. Children with chronic liver disease, inborn errors of metabolism, hemolytic anaemias, storage disorders, familial dyslipidemias, psychiatric illness or secondary causes of obesity were excluded. Children on medications that increase adiposity like steroids, anti-epileptics, antipsychotics, sick children and syndromic children were also excluded.

Children were enrolled to study after obtaining consent from the parents and assent from the child wherever applicable. Details of demographic data including age, sex and other relevant history were obtained in a predesigned proforma. Anthropometric measurements were taken following WHO recommendations. Weight and height were measured to the nearest 0.1 kg and 0.5 cm, respectively, and BMI was calculated. Skinfold thickness was measured at triceps, subscapular and suprailiac regions using a Harpenden skinfold caliper (Baty International Ltd, West Sussex, UK). Fasting venous samples were taken for alanine aminotransferase (ALT), aspartate aminotransferase (AST), glucose, triglycerides (TG), total cholesterol, high-density lipoprotein cholesterol (HDL), uric acid and thyroid stimulating hormone tests.

Liver fibrosis was evaluated using a fibroscan machine (Fibroscan Expert 360, Echosens, Paris, France). Liver stiffness measurement (LSMs) was estimated using S2 probe for children of age 6-12 years and M probe for those above 12 years. A probe was kept over the right lobe of the liver in supine position after a minimum of 3 hours of fasting state. After gel application, the probe was positioned perpendicular to the skin surface in one of the intercostal spaces over the right lobe of the liver (on the midaxillary line, in the 9th to 11th intercostal space). All measurements were performed by one expert technician using the same fibroscan device. Only those examinations with at least ten valid LSMs and with an interquartile range/median ratio of less than 15% were considered reliable. Based on LSM from fibroscan, they were divided into fibrosis and no fibrosis groups, using 6.1 kilopascals as cut-off. LSM of more than 6.9 pKa was considered severe fibrosis.[7] The controlled attenuation parameter was also measured in the same method and used to classify steatosis as S0 (less than 10% steatosis) with a score of less than 248 Db/m, S2 (10 to 33% mild steatosis) score of 248 to less than 268 dB/m, S2 (33% to <66%, moderate steatosis) score of 268 to less than 280 dB/m and S3 (≥66%, severe steatosis (severe)) score of 280 dB/m or more.[11]

Liver steatosis was also assessed by using USG and based on the echotexture and liver margins, it was classified based on ultrasonographic steatosis score into No steatosis, mild, moderate and severe steatosis. Ultrasonographic steatosis score was based on the following criteria: No steatosis (score 0) is defined as normal liver echotexture, mild steatosis (score 1) as slight diffuse increase in liver fine echoes with normal visualization of diaphragm and portal vein borders, moderate steatosis (score 2) as moderate increase in fine echoes and slight impairment of visualization of portal vein and diaphragm, whereas severe steatosis (score 3) as fine echoes with poor or no visualization of portal vein or diaphragm.[8] Children diagnosed with NAFLD were treated as per the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN) guidelines 2018. Children were followed up in OPD and evaluated for any other cause of fibrosis.

The required sample size was determined to be 273, considering a 95% confidence interval and a 5% absolute precision, assuming a fibrosis prevalence of 23% in obese and overweight children based on findings from a prior Indian study. However, the intended sample size could not be attained within the specified timeframe due to the impact of the COVID-19 pandemic and the closure of outpatient department (OPD) services for several months.

Statistical analysis

Data were analysed by using Stata 11.2 software. Qualitative data was represented in the form of frequency and percentage, whereas quantitative data were presented as mean ± SD, median (IQR range). Parameters of the children with and without fibrosis were compared using the Chi-square test

or Fisher's exact test for categorical variables. Continuous variables with normal distribution were analysed by independent *t*-test whereas Kruskal–Wallis or Wilcoxon rank sum test was used for non-normal distribution. Coefficient correlation was calculated for various biochemical parameters and fibrosis.

Ethical aspect

Ethical approval was granted by the Institute Ethics Committee Vide IEC/AIIMS/BBSR/PG Thesis/2019-20/104 DATED 8 February 2020. The study was performed conforming to the Helsinki Declaration of 1975, as revised in 2000 and 2008, concerning human and animal rights, and the authors followed the policy concerning informed consent obtained in writing.

Results

A total of 259 eligible children participated in the study, with 31% categorized as overweight and the remainder as obese. The mean age of the study cohort was 10.8 years, with males constituting 63% of the participants. Hypertension was detected in 23% of overweight and obese children, as outlined in Table 1. Transaminitis was evident in 47% of these children, with a mean ALT level of 35 U/L. Dyslipidaemia was noted in 45% of the study population (refer to Table 2). Acanthosis nigricans was observed in 29.3% of children with overweight and obesity. TE revealed fibrotic changes in 29.3% of the children, with severe fibrosis observed in 7.9%. Steatosis was present in 27.7% of the children, while moderate to severe steatosis was identified in 16.1% using TE. Additionally, 19.3% of the children exhibited both steatosis and fibrosis based on TE results. Ultrasound findings indicated steatosis in 23.8% of the children, with only 4.6% showing moderate to severe steatosis (see Table 2). Children exhibiting fibrosis or steatosis on TE demonstrated significantly higher BMI SD, waist circumference, skinfold thickness, hypertension and acanthosis nigricans compared to those without. Similarly, individuals with fibrosis exhibited significantly elevated levels of ALT, AST, uric acid, total cholesterol, triglycerides, CAP value and PNFI compared to those without fibrosis. Similar patterns were observed among children with steatosis (refer to Table 3).

There existed a strong correlation between LSM and ALT (r = 0.66, $P < 0.001$) as well as AST (r = 0.66, $P < 0.001$). BMI Z score, waist circumference and PNFI score demonstrated a moderate correlation, while CAP exhibited a strong correlation with ALT (r = 0.68, $P < 0.001$) and AST (r = 0.65, $P < 0.001$). BMI Z score and PNFI exhibited moderate correlations, whereas waist circumference displayed a weak correlation (refer to Table 4). Fibrosis grades exhibited a moderate correlation with AST, ALT, BMI Z score and PNFI score. In contrast, steatosis grades identified by TE demonstrated strong correlations with AST and ALT (refer to Tables 4 and 5). However, steatosis detected via USG exhibited a moderate correlation. Multivariate regression analysis unveiled significant correlations with skinfold thickness, acanthosis nigricans and ALT.

Table 1: Demographic parameters

Parameter	Mean±SD or number (%)
Age (years)	10.81±2.04
Gender (Male/Female)	165 (63.7)/94 (36.3)
Birth weight (gram)	2665±513
Low birth weight	93 (35.9)
Exclusive breastfeeding	205 (79.2)
Family history of obesity	99 (38.2)
Family history of diabetes	59 (22.7)
Family history of liver disease	11 (4.2)
*BMI	
Overweight	82 (31.7)
Grade I obesity	128 (49.4)
Grade II obesity	45 (17.4)
Grade III obesity	4 (1.4)
BMI (Z score)	1.71±0.57
Waist circumference (centile)	81.8±13.03
Waist circumference above 90th centiles	91 (35.1)
Waist-hip ratio above 0.85 in boys and 0.95 in girls	241 (93%)
Skinfold thickness	
Triceps (mm)	12.4±3.79
Subscapular (mm)	13.09±3.85
Suprailiac (mm)	15.51±6.02
Sum of the three (mm)	41.01±11.02
**Acanthosis nigricans	
Absent	182 (70.3)
Mild	59 (22.8)
Moderate-severe	18 (6.9)
Blood pressure	
Normal	168 (64.9)
Elevated BP	31 (12.0)
Stage 1 hypertension	56 (21.6)
Stage 2 hypertension	4 (1.4)

*Overweight (BMI=85th to <95th centile), class I obesity (BMI ≥95th centile to <120% of the 95th centile), class II (BMI ≥120% to <140% of the 95th centile), and class III (BMI ≥140% of 95th centile). **mild (limited to the base of skull and visible on close inspection), and moderate-to-severe (extending to lateral margins of the neck or further anteriorly)

The mean LSM was 6.01 kPa for children with mild steatosis on USG, while it measured 6.19 kPa and 8.55 kPa for moderate and severe steatosis, respectively. CAP scores were recorded as 204 dB/m, 301 dB/m, and 363 dB/m for mild, moderate and severe steatosis, respectively. USG steatosis grades displayed a strong correlation with CAP scores (r = 0.64, $P < 0.001$), while demonstrating a weaker correlation with LSM (r = 0.37, $P < 0.001$) (refer to Table 6).

Discussion

In this study, we found that 29.3% of overweight and obese children displayed fibrosis detected by TE, while steatosis was observed in 27.7% of the same demographic. Notably, 14.3% of children exhibited both fibrosis and steatosis concurrently. Furthermore, severe fibrosis was prevalent in 7.9% of children, with 16.1% demonstrating moderate to

severe steatosis. However, USG showed steatosis in only 23.5% of the children. BMI Z score/SD positively correlated with fibrosis and steatosis by TE and USG. We observed a significantly higher AST and ALT in children with fibrosis and steatosis with a strong correlation with LSM and CAP scores on TE.

Table 2: Investigation (Biochemical and radiological parameters)

Parameter	Mean±SD or number (%)
ALT (U/L)	35.00±23.9
Transaminitis (ALT >26 U/L for boys >22 U/L for girls)	122 (47.1)
AST (U/L)	33.38±20.65
Uric acid (mg/dL)	3.97±1.60
Triglyceride (8-10 years>100 mg/dL; 10-14 years >130 mg/dL)	118 (45.6)
HDL (<40 mg/dL for boys; <45 mg/dL for girls)	34 (13.1)
Total cholesterol (mg/dL)	165.80±26.52
Total cholesterol >200 mg/dL	30 (11.6)
Elevated fasting blood glucose (>100 mg/dL)	27 (10.4)
Hypothyroidism	0 (0)
Fibrosis as per PNFI (PNFI >9)	202 (78%)
USG steatosis	
G0 (Steatosis absent)	197 (76.2)
G1 (Mild steatosis)	50 (19.2)
G2 (Moderate steatosis)	10 (3.8)
G3 (Severe steatosis)	2 (0.8)
Fibroscan	
Mean LSM (kPa)	5.44±1.21
Fibrosis as detected by fibroscan (LSM>6.1 kPa)	76 (29.3)
Severe fibrosis as detected by fibroscan (LSM>6.9 kPa)	20 (7.9)
Mean controlled attenuation parameter (CAP) (dB/m)	221.42±45.29
Fibroscan steatosis (Grading)	
S0 (<248 dB/m)	186 (71.8)
S1 (248-268 dB/m)	30 (11.6)
S2 (268-280 dB/m)	10 (3.8)
S3 (>280 dB/m)	32 (12.3)

A comparable study by Ciardullo *et al*.[12] reported a 24.1% prevalence of steatosis among obese children aged 12-18 years utilizing TE with a cut-off CAP score of 248 dB/m, akin to our findings. They also identified severe steatosis in 11.6% of obese children with a cut-off of 280 dB/m, similar to our observed prevalence of 12.3%. However, their study reported significant fibrosis in only 4.4% of children, lower than ours (7.9%), possibly due to their utilization of a higher LSM cut-off of 7.4 kPa. There is substantial variability in fibroscan-based cut-offs for defining liver fibrosis, with values ranging from 5.15 kPa to 6.5 kPa.[7,13,14] This variability can be attributed to differences in study populations and the absence of a standardized cut-off for the paediatric age group. Fitzpatrick *et al*.[14] investigated the correlation between fibrosis in histopathology and the fibroscan score, proposing a cut-off value of 6.1 kPa for children with NAFLD, with a sensitivity of 60% and specificity of 78%. A score exceeding 6.9 kPa was indicative of severe fibrosis, with a sensitivity of 72% and specificity of 85%. Pawar *et al*.[7] employed the same cut-offs and observed a 23% prevalence of fibrosis in obese and overweight Indian children, similar to our findings. We adopted the same cut-off used by Pawar *et al*.[7] and Fitzpatrick *et al*.[14] A recent study by Yang Lin *et al*.[15] from China indicated that a fibroscan LSM value >4.65kPa suggested NAFLD, while a value >5.15 kPa suggested NASH in obese children. Other studies have reported a higher prevalence but utilized a lower cut-off.[13,16] The incidence of fibrosis has been reported to be up to 50% in overweight and obese children, as demonstrated in a Korean study.[13] However, Jain *et al*.[17] and Engelmann *et al*.[18] reported a 95th percentile cut-off of 7.2 kPa and 6.8 kPa, respectively, among healthy children, higher than previous studies.

Establishment of a definitive threshold for CAP score in the detection of fatty liver using fibroscan remains uncertain. Ferraioli *et al*.[19] suggested the utilization of CAP for detecting steatohepatitis in children, where 9.3% of individuals with normal ultrasound findings were found to have steatohepatitis detected using CAP as compared to 4.2% in the present study.

Table 3: Comparison of parameters between fibrosis vs No fibrosis and steatosis vs no steatosis based on TE

Variable	Fibrosis (*n*-76) (%)	No fibrosis (*n*-183) (%)	*P*	Steatosis (*n*-72) (%)	No steatosis (*n*-187) (%)	*P*
Weight (kg)	58±12	44±11	<0.001	59±11	44±11	<0.001
Height (cm)	146±10.9	139±10.9	<0.001	146±10.3	139±11	<0.001
Low birth weight	2516±541	2727±489	0.002	2703±521	2565±480	0.05
BMI (kg/m^2)	26.8±2.97	22.4±3.0	<0.001	27.1±2.8	22.4±2.9	<0.001
BMI Z score	2.1±0.4	1.54±0.52	<0.001	2.17±0.46	1.52±0.5	<0.001
Waist circumference (mm)	84.6±8.4	76±8	<0.001	84.7±8.1	76.6±8.6	<0.001
Skinfold thickness (mm)	49.6.5±9.3	37±9.5	<0.001	49.7±8.8	37.6±9.8	<0.001
CAP (dB/m)	256±40	206±43	<0.001	277±23	199±31	<0.001
Uric acid (mg/dL)	4.8±2.1	3.6±1.1	<0.001	4.6±1.2	3.7±1.6	<0.001
AST	49 (34-70)	21 (16-30)	<0.00	53±19	25±14	<0.001
ALT	56 (35-75)	21 (17-29)	<0.001	58±22	26±17	<0.001
TG (mg/dL)	130±48	113±33	0.06	143±37	103±27	<0.001
Cholesterol (mg/dL)	171±34	165±25	0.35	181±29	159±22	<0.001
HDL (mg/dL)	43±8	49±8	0.005	51±8.1	44±7.4	<0.001
PNFI	9.6±1.1	9.3±0.9	0.19	9.8±0.24	9.1±1	<0.001

Table 4: Correlation of Anthropometric and biochemical parameters with LSM and CAP on TE

	LSM Coefficient of correlation	CAP Coefficient of correlation
BMI Z score	0.47	0.57
	<0.001	<0.001
Waist circumference	0.48	0.29
	<0.001	<0.001
Skin fold thickness (mm)	0.58	0.57
	<0.001	<0.001
CAP (dB/m)	0.56	0.56
	<0.001	<0.001
AST	0.66	0.65
	<0.001	<0.001
AST	0.66	0.68
	<0.001	<0.001
PNFI	0.52	0.48
	<0.001	<0.001

Table 5: Correlation of Anthropometric and biochemical parameters with grades of fibrosis and steatosis by TE and Steatosis by USG

	Fibrosis by TE Coefficient of correlation	Steatosis by TE Coefficient of correlation	Steatosis by USG
BMI Z score	0.44	0.52	0.44
	<0.001	<0.001	<0.001
Waist circumference	0.41	0.38	0.29
	<0.001	<0.001	<0.001
Skin fold thickness (mm)	0.52	0.50	0.41
	<0.001	<0.001	<0.001
LSM (kPa)	0.79	0.51	0.37
	<0.001	<0.001	<0.001
CAP	0.50	0.77	0.64
	<0.001	<0.001	<0.001
AST	0.57	0.62	0.54
	<0.001	<0.001	<0.001
AST	0.50	0.62	0.54
	<0.001	<0.001	<0.001
PNFI	0.46	0.45	0.32
	<0.001	<0.001	<0.001

Table 6: Values of TE and PNFI according to liver US grading

USG grade steatosis	Mean LSM (pKa)	Mean CAP (dB/m0	PNFI
G0 (No Steatosis) *n*=197	5.2±0.07	204±2.5	9.2±1
G1 (Mild steatosis) *n*=50	6.01±0.13	264±3.4	9.6±0.5
G2 (Moderate Steatosis) *n*=10	6.19±0.34	301±3.9	9.8±0.2
G3 (Severe steatosis) *n*=2	8.55±1.85	363±12	9.9±0.02

Desai *et al*.[20] proposed a CAP score of 225 dB/m as a threshold for fatty liver in children. Yang Lin *et al*.[21] suggested an optimal predictive value of CAP >258.00 dB/m for diagnosing NAFLD and CAP >276.00 dB/m for diagnosing NASH in obese children.

Increased BMI is an established risk factor for NAFLD.[22-24] Our study showed a BMI Z score/SD positively associated with an increased chance of fibrosis and steatosis both by TE and USG. A similar result has been observed by Jain *et al*.[6] who suggested that BMI SD of more than 2.2 was associated with steatosis by USG. A significant positive correlation was found between BMI-SDS and both CAP and LSM for BMI-SDS >1.28.[24]. Similarly, waist circumference had shown a significant positive correlation with fibrosis and steatosis, and previous studies reported a similar finding.[6,8]

An increased incidence of hypertension is seen in children with obesity and overweight. The study by Das *et al*.[5] showed a hypertension prevalence of 14.5% in overweight and obese children, whereas our study showed a higher prevalence of 23%. Children with increased liver stiffness were found to have a higher prevalence of hypertension than those who did not have fibrotic changes.

Both ALT and AST levels are significantly higher in children with fibrosis and steatosis and were strongly correlated with LSM and CAP scores as well as grades of fibrosis and steatosis based on TE. Median ALT in children with fibrosis was 56 (35-75) U/l, whereas AST was 49 (34-70) U/l. Jain *et al*.[6] observed a higher ALT of 49.8 (U/l) and AST of 40.6 (U/l) in children with steatosis as detected by USG similar to ours. ALT value of 33.0 U/L was more associated with NAFLD with a sensitivity of 60.8% and specificity of 66.2%. Similarly, Kwon *et al*.[13] have reported higher AST and ALT among children with obesity too. A strong correlation between ALT and AST was observed with both LSM and CAP scores, similar to those of Kwon *et al*. However, we also found a strong correlation between grades of steatosis by TE, whereas a moderate correlation was observed between fibrosis and steatosis by USG. This is contrary to Kwon *et al*. who observed a strong significant correlation was observed between fibrosis grades on CT as well as fibroscan. This may be due to older children with higher BMI in their study as compared to ours. Increased triglycerides, total cholesterol and decreased HDL, have been observed in previous studies in obese and overweight children with NAFLD similar to ours.[2,25] Increased uric acid levels in children with NAFLD, which was similar to the findings seen by Cardoso *et al*.[26] Uric acid has been associated with a higher prevalence of liver steatosis and fibrosis in a nationwide study in the USA.[27]

In our study population, the PNFI score was significantly higher in children with either fibrosis or steatosis. However, on multivariate regression analysis, we did not observe a significant correlation with LSM or CAP scores, or grades of fibrosis/steatosis. Similar findings of a poor correlation between various NAFLD scores and fibrosis or steatosis in children have been reported.[28] Nobili *et al*.[29] identified a cut-off value of 9 for PNFI, achieving a sensitivity of 41% and a specificity of 98.4%.

USG showed steatosis in only 23.5% of the children compared to TE. We could find only a weak correlation between USG steatosis and LSM, whereas a strong correlation was observed with CAP. A study from Korea by Lee *et al*.[30] showed a correlation between fibroscan and CT abdomen but not with USG findings, suggesting the low accuracy of ultrasound in detecting paediatric NASH. The limitations of this study were that it was not confirmed by histology or MRI-PDFF and that the desired sample size was not achieved.

Conclusion

Fibroscan can be an alternative noninvasive tool for detecting fibrosis and steatosis in overweight and obese children, along with currently available modalities, displaying a strong correlation with liver enzymes. Despite the ready availability of ultrasound (USG), it has its limitations. Therefore, utilizing transient elastography (TE) in conjunction with liver enzyme assessment and USG can serve as an effective screening method for children with nonalcoholic fatty liver disease (NAFLD). Importantly, it can aid in determining the necessity for a biopsy among children with NAFLD.

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Authors' contribution

AKS and SM have conceptualized the study. FM has collected data and written the manuscript. MKP and SN have helped in doing the transient elastography and USG, respectively, and also interpreted the test results. AKS has reviewed the manuscript critically and will remain as guarantor.

The manuscript has been read and approved by all the authors.

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Conflicts of interest

There are no conflicts of interest.

Data availability statement

Data will be shared on request to the corresponding author.

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