

Hepatic Steatosis in Overweight/Obese Children despite Normal Transaminase Levels: A Cross-sectional Study

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Abstract

Background: The objective is to determine the liver involvement among children who are overweight or obese in relation to serum transaminases, biochemical parameters, liver ultrasound, and shear wave elastography. **Material and Methods:** This cross-sectional study included 80 overweight and obese children aged 5–15 years attending a tertiary care hospital. Anthropometric measurements, liver function tests, fasting blood sugar, HbA1c, serum insulin, lipid profile, abdominal ultrasonography, and elastography (SWE) were performed. Statistical comparisons were performed between normal and abnormal USG groups. **Results:** The mean age was 10.4 ± 2.3 years, with 70.0% males. Forty-eight (60.0%) children were overweight and 32 (40.0%) were obese. Normal liver echogenicity was observed in 51 (63.7%), while fatty liver was detected in 29 (36.3%) children. The mean SWE was 5.41 ± 2.52 kPa and was significantly higher in children with abnormal USG than in those with normal USG (8.1 ± 1.9 vs. 3.9 ± 1.2 kPa, $p < 0.001$). LDL was also significantly higher in the abnormal-USG group (70.7 ± 20.8 vs. 61.7 ± 9.9 mg/dL, $p = 0.010$). Liver enzymes, fasting blood sugar, HbA1c, and serum insulin were statistically comparable between groups. **Conclusion:** Combining anthropometric evaluation with liver enzyme testing, ultrasonography, and elastography may enable earlier detection of hepatic abnormalities in obese children and facilitate identification of those requiring more comprehensive metabolic and hepatological assessment.

Keywords: elasticity imaging techniques, fatty liver, glycated hemoglobin, liver function tests, overweight, pediatric obesity.

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INTRODUCTION

Obesity has been increasing worldwide among children with reported prevalence rate of 6.97% in the Indian population and 8.2% in world.^[1,2] This increasing incidence has been associated with hepatic dysfunction, which may be accounted for why increase in liver enzymes or elastography technique or ultrasonography.^[3] This has been accounted for by lipid buildup in the liver and ongoing oxidative stress in the body, stimulating the inflammatory pathways and fibrosis.^[4]

The association and the prevalence of hepatic dysfunction among overweight and obese children have been less studied.^[5-7] Thus, we conducted this study to assess hepatic involvement in overweight and obese children using liver enzymes, ultrasonography, and elastography.

MATERIALS AND METHODS

The study was cross-sectional in design, including children who were obese or overweight as per the Revised Indian Academy of Pediatrics Growth Charts 2015.^[8]

Consecutively, 80 children were included who presented to the Pediatrics department of our hospital, New Delhi, after clearance from the hospital's Ethical Committee. Exclusion criteria were any child with chronic liver disease/autoimmune hepatic disorder or renal disease (such as nephrotic syndrome), usage of hepatotoxic drugs, or

prolonged > 6 months of steroid use. A formal written consent from the parents or legal guardians was obtained for the study.

For every participant, a comprehensive clinical history was taken, and a complete physical examination was carried out. Anthropometric parameters were measured for every child. Based on the Revised IAP Growth Charts 2015, obesity was defined as a BMI equivalent to an adult value of 27 kg/m^2 , and overweight as a BMI equivalent to an adult value of 23 kg/m^2 . Under aseptic conditions, roughly 7–10 mL of venous blood was drawn for routine baseline biochemical investigations. Liver ultrasonography was performed, and hepatic status was further assessed with FibroScan to measure liver stiffness (elasticity quotient) and to grade hepatic steatosis and fibrosis. All clinical, anthropometric, laboratory, and imaging data were documented in a pre-structured proforma.

Statistical analysis: Data was fed into an Excel spreadsheet and was analyzed by advanced Microsoft Excel software with

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tabulation in the form of frequency, percentage, and mean with standard deviation. Appropriate parametric or non-parametric tests such as Chi-square/Fisher's exact test, and ANOVA were applied as appropriate.

RESULTS

The mean age of participants was 10.4 ± 2.3 years. Among

them, 27 (21.6%) were aged 5–9 years, and 53 (78.4%) were aged 10–15 years. Of the 80 participants, majority [56 (70.0%)] were males. The mean weight was 53.2 ± 8.2 kg, mean height was 145.1 ± 9.2 cm, and mean BMI 25.2 ± 2.2 kg/m², mean waist circumference was 82.9 ± 6.9 cm, hip circumference was 88.1 ± 6.9 cm, waist-hip ratio was 0.94 ± 0.02 , and waist-height ratio was 0.57 ± 0.05 . Most of the participants, i.e. 48 (60.0%) were overweight, and 32 (40.0%) were obese [Table 1].

Table 1: Demographic and anthropometric data of study participants (N=80)

Age (years)	n (%)		
Mean $\pm$ SD	10.4 ± 2.3		
5-9 years	27 (21.6)		
10-15 years	53 (78.4)		
Weight (kg)	53.2 ± 8.2		
Height (cm)	145.1 ± 9.2		
BMI (kg/m ²)	25.2 ± 2.2		
Waist circumference (cm)	82.9 ± 6.9		
Hip circumference (cm)	88.1 ± 6.9		
Waist-hip ratio	0.94 ± 0.02		
Waist-height ratio	0.57 ± 0.05		
Weight Category	n (%)		
Overweight	48(60.0)	Males 34 (70.8)	Females 14 (29.2)
Obese	32(40.0)		

Mean AST was 36.8 ± 33.9 U/L, ALT 41.1 ± 78.1 U/L, ALP 275.7 ± 56.6 U/L, and GGT 18.6 ± 6.8 U/L. Mean PT was 11.4 ± 0.9 sec, INR 0.89 ± 0.10 , total protein 7.2 ± 0.4 g/dl, and serum albumin was 4.3 ± 0.7 g/dl. Thyroid parameters were T3 1.50 ± 2.34 ng/dl, T4 6.57 ± 1.89 mcg/dl, and TSH 2.45 ± 0.95 mIU/L. Mean fasting blood sugar was 88.4 ± 6.6 mg/dl, HbA1C $5.25 \pm 0.43\%$, and serum insulin 10.2 ± 4.2

μ IU/ml. Lipid profile values were total cholesterol 153.9 ± 18.6 mg/dl, HDL 42.4 ± 8.1 mg/dl, LDL 65.0 ± 15.3 mg/dl, and mean serum cortisol was 10.2 ± 1.9 μ g/dl [Table 2].

No significant differences were found in laboratory parameters between overweight and obese participants ($p > 0.05$).

Table 2: Laboratory parameters of study participants (N=80)

Parameter	Mean $\pm$ SD
Liver Function Tests	
AST (U/L)	36.8 ± 33.9
ALT (U/L)	41.1 ± 78.1
ALP (U/L)	275.7 ± 56.6
GGT (U/L)	18.6 ± 6.8
Coagulation Profile	
PT (sec)	11.4 ± 0.9
INR	0.89 ± 0.10
Protein Profile	
Total Protein (g/dl)	7.2 ± 0.4
Serum Albumin (g/dl)	4.3 ± 0.7
Thyroid Function	
T3 (ng/dl)	1.50 ± 2.34
T4 (mcg/dl)	6.57 ± 1.89
TSH (mIU/L)	2.45 ± 0.95
Metabolic Parameters	
FBS (mg/dl)	88.4 ± 6.6
HbA1C (%)	5.25 ± 0.43
Serum Insulin (microIU/ml)	10.2 ± 4.2
Lipid Profile	
Total Cholesterol (mg/dl)	153.9 ± 18.6
HDL (mg/dl)	42.4 ± 8.1
LDL (mg/dl)	65.0 ± 15.3
Other Parameters	
Serum Cortisol (mcg/dl)	10.2 ± 1.9

Normal liver echogenicity was observed in 51 (63.7%) and fatty liver in 29 (36.3%) participants (Figure 1). Among the fatty liver cases, Grade I, II, and III were present among 51 (63.7%), 20 (25.0%), and 9 (11.3%) participants,

respectively. The mean liver stiffness (SWE) was 5.41 ± 2.52 kPa.

Abnormal USG findings were most frequent in the 9–11-year age group (51.7%), but no significant association was noted

with age ($p=0.121$). Among males, 22 (75.9%) had abnormal USG findings compared to 7 (24.1%) females, with no significant association between gender and USG findings ($p=0.388$). There were no significant differences in anthropometric parameters between normal and abnormal USG groups ($p>0.05$). Similarly, no significant relationship was found between clinical findings (like acanthosis nigricans) and USG abnormalities ($p>0.05$).

Compared to those with normal USG, participants with abnormal USG had significantly higher LDL (70.7 ± 20.8 vs. 61.7 ± 9.9 mg/dl, $p=0.010$) and mean SWE (8.1 ± 1.9 vs. 3.9 ± 1.2 kPa) ($p<0.001$). LFT showed no significant association with USG findings ($P>0.05$) [Table 3].

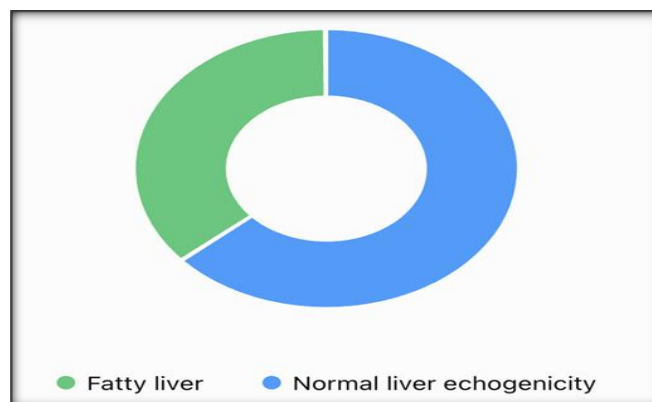


Figure 1: Distribution of ultrasonographic liver findings among study participants (N=80).

Table 3: Comparison of laboratory parameters with USG findings

Parameter	Normal USG (n=51)	Abnormal USG (n=29)	p-value
Liver Function Tests			
AST (U/L)	33.5 $\pm$ 11.1	42.8 $\pm$ 54.4	0.241
ALT (U/L)	32.8 $\pm$ 20.5	55.7 $\pm$ 126.9	0.211
ALP (U/L)	273.2 $\pm$ 59.8	280.1 $\pm$ 51.1	0.603
GGT (U/L)	18.0 $\pm$ 6.6	19.5 $\pm$ 7.1	0.363
Coagulation Profile			
PT (sec)	11.4 $\pm$ 0.9	11.2 $\pm$ 0.9	0.321
INR	0.879 $\pm$ 0.109	0.913 $\pm$ 0.093	0.160
Protein Profile			
Total Protein (g/dl)	7.19 $\pm$ 0.37	7.10 $\pm$ 0.48	0.342
Serum Albumin (g/dl)	4.24 $\pm$ 0.37	4.37 $\pm$ 1.09	0.426
Metabolic Parameters			
FBS (mg/dl)	88.2 $\pm$ 6.5	88.7 $\pm$ 6.8	0.730
HbA1C (%)	5.22 $\pm$ 0.44	5.29 $\pm$ 0.43	0.519
Serum Insulin (microIU/ml)	10.8 $\pm$ 4.7	9.1 $\pm$ 2.8	0.091
Lipid Profile			
Total Cholesterol (mg/dl)	151.8 $\pm$ 19.3	157.6 $\pm$ 17.1	0.185
HDL (mg/dl)	43.0 $\pm$ 8.2	41.5 $\pm$ 7.9	0.418
LDL (mg/dl)	61.7 $\pm$ 9.9	70.7 $\pm$ 20.8	0.010*
SWE Findings (kPa)	3.9 $\pm$ 1.2	8.1 $\pm$ 1.9	<0.001**

* $p<0.05$, ** $p<0.001$

Fatty liver was observed in 14 (43.8%) obese and 15 (31.3%) overweight participants, with mean SWE values of 5.51 ± 2.64 kPa and 5.34 ± 2.45 kPa, respectively, showing no significant difference ($p=0.769$).

DISCUSSION

The present study is one of the few studies conducted on the determination of hepatic dysfunction among overweight and obese children, wherein, among 80 children, we found that abnormal fatty liver of grade 2/3 was present in 29 (36.3%) children.

Among other studies, the rate of NAFLD in Kumar et al^[9] study was 44% and in Thiagarajan S et al,^[10] was 51.3% among overweight/obese Indian children. A systematic review and meta-analysis reported MAFLD in 41.2% of overweight/obese children.^[11]

In children with obesity, impaired liver function stems mainly from insulin resistance, which encourages the excessive buildup of fat within the liver. This overload of lipids induces oxidative stress at the cellular level and causes damage to hepatocytes, while pro-inflammatory cytokines

such as IL-6 sustain chronic systemic inflammation, thereby propelling the progression of steatohepatitis and fibrosis.^[10]

As for elastography, we found that the mean SWE was 5.41 ± 2.52 kPa, which is significantly higher among those with abnormal USG compared to normal USG (8.1 ± 1.9 vs. 3.9 ± 1.2 kPa, $p<0.001$).

This was in line with the findings of Akçiçek and Dağ,^[6] who found significantly higher liver stiffness (as measured by SWE) in obese children with USG-detected hepatic steatosis compared to those without steatosis (6.2 ± 1.7 vs. 4.9 ± 0.6 kPa, $P=0.000$). This was replicated in a case-control study whereby SWE was higher among overweight/obese children with hepatosteatosis compared to healthy children (34 vs. 8.2 kPa, P -value below 0.001).^[5] This suggests from the fact that excessive accumulation of intrahepatic fat promotes oxidative stress and persistent inflammation, leading to increased tissue viscoelasticity and collagen deposition within the extracellular matrix. These pathological changes enhance shear wave propagation through the liver, resulting in higher shear wave velocities and elevated liver stiffness measurements.^[4]

Predominantly among all the factors, it was interesting to note that serum transaminases- AST, ALT, ALP, and GGT were

statistically comparable between normal and abnormal ultrasound, indicating that transaminases may not be early markers for determining hepatic dysfunction among obese/overweight children.

In contrast, Kumar et al^[9] found that among obese children, those with NAFLD had significantly higher AST (126.8 vs. 32.7, $P<0.001$) and ALT (156.5 vs. 32.5, $p<0.001$) than those without NAFLD.

Hwang and Cho^[7] compared liver function parameters in children with USG-detected hepatic steatosis and those without steatosis, and found significantly higher AST (41 vs. 22 IU/L), ALT (64 vs. 13 IU/L) and GGT (27 vs. 12 IU/L) (all $p<0.001$).

Aminotransferase concentrations may remain normal in the early stages of hepatic steatosis, as lipid accumulation within hepatocytes can precede substantial cellular injury or inflammatory necrosis. Because serum transaminases primarily reflect active hepatocellular membrane damage rather than hepatic fat deposition itself, early and clinically silent steatosis may develop and progress without causing an initial rise in transaminase levels.^[12] However, associations may be seen if children progress to advanced stages.

Among the metabolic parameters, fasting blood sugar, HbA1c, and serum insulin were statistically comparable, which indicates that the presence of hepatic involvement in our study was not necessarily accompanied by overt abnormalities in glycemic parameters. Hwang and Cho^[7] also found comparable glucose in children with USG-detected hepatic steatosis and those without steatosis (97 vs. 97 mg/dL, $P=1$).

Specifically, the only significant finding was in terms of LDL, which was significantly higher in abnormal USG compared to normal USG (70.7 ± 20.8 vs. 61.7 ± 9.9 mg/dl, $p=0.010$), giving an indication of the use of lipid profile as an early marker along with elastography.

Kumar et al,^[9] found that among obese children, those with NAFLD had significantly higher total cholesterol (209.5 vs. 116.1), LDL (145.1 vs. 104.5) and triglyceride (183.5 vs. 113.3) ($P<0.001$ for all) than those without NAFLD. Hwang and Cho⁷ found that children with USG-detected hepatic steatosis had significantly higher total cholesterol (171.81 vs. 155.46 mg/dL, $P=0.023$), triglycerides (111 vs. 79 mg/dL, $P<0.001$) and LDL (108.6 vs. 93.79 mg/dL, $P=0.023$) than those without steatosis. Ahmed et al,^[12] reported that elevated triglycerides, LDL and HDL were present in 73.2%, 21.4%, and 21.4% of the obese children with fatty liver. The reason being that excessive hepatic fat deposition promotes increased very-low-density lipoprotein (VLDL) production and reduces efficient lipoprotein clearance, thereby contributing to elevated circulating low-density lipoprotein (LDL) levels. As these metabolic abnormalities may arise early, alongside hepatic fat accumulation and before evident hepatocellular injury, serum LDL may serve as a useful early metabolic indicator when interpreted together with quantitative liver elastography.^[7]

Limitations: This study cross-sectional design precludes establishing temporal or causal relationships between obesity and hepatic involvement. Ultrasonography is operator-dependent and has limited sensitivity for mild steatosis.

Although SWE provides a non-invasive assessment of liver stiffness, pediatric reference values and standardized cut-offs were not completely established. Liver biopsy and MRI-based evaluation were not done for histopathological confirmation or quantitative measurement of hepatic fat.

CONCLUSION

Hepatic involvement was seen in 36.3% of overweight/obese children and was detected on imaging despite largely normal transaminase levels. Abnormal ultrasonography was associated with greater liver stiffness, highlighting that biochemical tests alone may underestimate early hepatic abnormalities. SWE may therefore serve as a useful non-invasive adjunct for risk stratification. An integrated assessment using liver enzymes, ultrasonography, and elastography may improve early detection of hepatic involvement in pediatric obesity.

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Conflicts of interest

There are no conflicts of interest.

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