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Original Research Article

Clinicometabolic profile of fatty liver disease in obese children: a study from Kashmir

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ABSTRACT

Background: Childhood obesity is an emerging public health challenge and is closely associated with metabolic abnormalities and hepatic complications, particularly fatty liver disease. Objectives were to assess the clinicometabolic profile and determine the incidence of fatty liver disease among overweight and obese children from Kashmir.
Methods: This descriptive cross-sectional study was conducted over two years from August 2022 to July 2024 at a tertiary care center. A total of 112 children aged 5-18 years with body mass index (BMI) ≥85th percentile were enrolled. Anthropometry, blood pressure measurement, and biochemical evaluation including fasting glucose, lipid profile, liver enzymes, and thyroid function tests were performed. Hepatic steatosis was assessed by ultrasonography, and FibroScan was performed in children with significant ultrasonographic changes or persistent elevation of liver enzymes.
Results: The mean age was 10.29±2.71 years, with males constituting 57.1% of participants; 71.4% were obese (BMI ≥95th percentile). Metabolic abnormalities were common, including impaired fasting glucose in 19.6%, hypertriglyceridemia in 73.2%, low HDL cholesterol, and elevated blood pressure in nearly one-third of children. Elevated AST and ALT levels were observed in 52.7% and 34.8% of participants, respectively. Ultrasonography detected fatty liver in 67.9% of children, with prevalence increasing with BMI. FibroScan revealed elevated controlled attenuation parameter values consistent with hepatic steatosis, while liver stiffness values remained within the F0-F1 range.
Conclusions: Fatty liver disease is highly prevalent among obese children and is strongly associated with metabolic derangements, emphasizing the need for early screening and comprehensive metabolic evaluation

Keywords: Fatty liver, Obese children, FibroScan

INTRODUCTION

Childhood obesity has emerged as a major global health challenge, affecting both developed and developing countries alike.[1] India, in particular, has witnessed a steep rise in the prevalence of childhood overweight and obesity, resulting in an increasing burden of metabolic and non-communicable diseases.[2] Overweight and obese children are at higher risk of remaining obese into adulthood and developing early-onset type 2 diabetes mellitus, cardiovascular diseases, and metabolic syndrome. The etiology of obesity is multifactorial, involving genetic, behavioral, and environmental influences.

Obesity is defined by an excess accumulation of body fat, although specific pediatric cutoffs vary. Williams et al defined obesity as body fat ≥25% for boys and ≥30% for girls, while the centers for disease control and prevention (CDC) classifies overweight as a BMI ≥85th to <95th percentile and obesity as ≥95th percentile for age.[3-6] Some Indian studies have similarly adopted percentile-based definitions while some European studies have used values consistent with world health organization (WHO) growth references.[7-9]

Among obesity-related complications, hepatic involvement has gained increasing attention. Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of disorders ranging from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and hepatocellular carcinoma.[10-12] NAFLD is being increasingly recognized in children, with features of transaminitis ranging from 10-25% and ultrasonography showing liver brightness in 22.5-77% such patients.[13-16] Insulin resistance, oxidative stress, and proinflammatory cytokines play central roles in the pathogenesis.[17-20]Although insulin resistance is considered pivotal to the development of fatty liver, few data have been reported in the pediatric literature.[19,21,22]

Similarly, abnormal iron handling and leptin levels are also implicated in the pathogenesis of fatty liver, but data in childhood are conflicting and scarce.[19,23]

Interleukin (IL)-6 produced by fat cells induces the synthesis of C-reactive protein (CRP) by the liver; thus, obesity is associated with low-grade systemic inflammation.[24]

The diagnosis of NAFLD in children and adults is largely based on elevated liver enzymes and imaging evidence of hepatic steatosis, although liver biopsy remains the gold standard. However, its invasiveness and sampling errors limit its use, leading to an increasing reliance on ultrasonography and non-invasive elastography (FibroScan) for screening.[25-27] With rising obesity in the Kashmiri pediatric population, there is limited local data on the incidence of fatty liver disease and associated biochemical abnormalities. This study was therefore conducted to evaluate the incidence of fatty liver disease among obese children using liver enzyme profiles and ultrasonographic assessment, and to explore the degree of fibrosis in selected children using FibroScan.

METHODS

This descriptive, cross-sectional prevalence study was conducted over a period of two-years from August 2022 to July 2024 at the Department of Paediatrics and Neonatology, Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Soura. Ethical clearance was given by the institutional review board. Written informed consent was obtained from parents or guardians before enrollment.

Study population

Children and adolescents aged <18 years presenting to the pediatric OPD, emergency, or wards and meeting inclusion criteria were enrolled consecutively.

Inclusion criteria

Children aged 5-18 years with BMI between ≥85th-<95th percentile (overweight) or ≥95th percentile (obese) for age and sex, based on CDC criteria were included.[4]

Exclusion criteria

Children with chronic systemic disorders, syndromic or endocrine obesity, inborn errors of metabolism, or pre-existing hepatic, renal, or cardiac disease were excluded.

Data collection and investigations

Demographic data including age, gender, height, weight, and BMI were recorded. BMI was calculated as weight (kg)/height² (m²), and BMI z-scores were derived using WHO reference charts. A detailed family history of obesity, diabetes mellitus, hypertension, and dyslipidemia among first-degree relatives was obtained.

All participants underwent clinical examination including measurement of blood pressure and anthropometric indices. Fasting blood samples were analyzed for plasma glucose, total cholesterol, triglycerides, HDL, LDL, VLDL, liver enzymes (AST, ALT, ALP and GGT), total bilirubin, and albumin. Normal reference ranges followed institutional laboratory standards.

Ultrasonography and FibroScan

Abdominal ultrasonography was performed to detect hepatic steatosis, using standardized echogenicity criteria. Fatty liver was graded as:

Grade I (Mild)

Slight diffuse increase in fine echoes with visible intrahepatic vessels.

Grade II (Moderate)

Increased echogenicity with the obscured portal vein walls.

Grade III (Severe)

Marked echogenicity with poor visualization of diaphragm and vessels.

Children with grade II or III fatty liver or persistently elevated liver enzymes underwent FibroScan for quantification of hepatic stiffness and fibrosis.

Sample size and statistical analysis

The data collected was analyzed using SPSS version 23. Continuous variables were expressed as mean±SD and categorical data as frequencies or percentages. A $p<0.05$ was considered significant.

RESULTS

A total of 112 overweight and obese children were included in the study. The mean age of the participants was 10.29±2.71 years, ranging from 5-15 years, and males constituted 57.1% of cohort. Most children were markedly obese, with 71.4% falling above the 95th BMI percentile and mean BMI of 29.21±3.64 kg/m^2. BP assessment revealed that nearly one-third of cohort had elevated values, as 25% met criteria for stage 1 and 7.1% for stage 2 hypertension, indicating a substantial burden of cardiovascular risk among these children (Table 1).

Biochemical evaluation demonstrated multiple metabolic abnormalities. Impaired fasting glucose levels were present in 19.6% of participants. Dyslipidemia was highly prevalent, with triglyceride elevation being the most frequent abnormality; 73.2% of children exhibited triglyceride values greater than 130 mg/dL, and many also had depressed HDL levels, elevated total cholesterol, or high LDL concentrations. Liver function testing showed considerable hepatocellular enzyme derangements. More than half of the participants (52.7%) had AST values exceeding 35 U/L, and 34.8% had ALT levels above 45 U/L. ALP elevations were common, observed in over 80% of the children, while GGT abnormalities occurred in 34.8%. Thyroid function analysis demonstrated that more than one-third of the cohort (35.7%) had elevated TSH values, suggesting a pattern of subclinical hypothyroid tendencies among obese participants (Table 2).

Ultrasonographic evaluation of the liver revealed that fatty liver changes were widespread. Although 32.1% of children had a normal echopattern, majority demonstrated hepatic steatosis, with grade 1 changes observed in half of all participants and grade 2/3 changes in an additional 17.9%. The increasing severity of ultrasonographic abnormalities was closely aligned with higher BMI percentiles, showing a clear trend toward worsening steatosis with increasing adiposity (Table 3). FibroScan assessment, performed for participants with significant ultrasound changes or biochemical abnormalities, further characterized hepatic involvement. The mean CAP score was 285.74±20.58 dB/m, consistent with substantial hepatic fat infiltration, while the mean liver stiffness value was 5.02±1.01 kPa. All children fell within the F0-F1 fibrosis range, indicating the absence of clinically significant fibrosis across the cohort despite the high prevalence of steatosis (Table 3).

Associations between anthropometric, biochemical, and imaging parameters demonstrated a coherent metabolic-hepatic pattern. Higher BMI values were consistently accompanied by more severe steatosis, both on ultrasound and FibroScan, reflected by higher CAP scores and increased liver stiffness measurements. Children with elevated ALT levels were more likely to have grade 2-3 steatosis, and these same children exhibited higher CAP scores, suggesting concordance between biochemical markers of hepatocellular injury and radiologic indicators of hepatic fat accumulation. Triglyceride elevations showed tendency to cluster with higher BP values, indicating overlapping metabolic and cardiovascular risks. Subgroup of children with elevated TSH also demonstrated trends toward higher BMI and ALT levels, supporting possible interplay between thyroid dysfunction and metabolic liver disease, although this association requires formal statistical testing for confirmation.

Overall, results demonstrate a high burden of metabolic dysregulation, hepatic steatosis, and early indicators of cardiometabolic strain among overweight and obese children, with strong interrelationships between adiposity, lipid derangements, liver enzyme abnormalities, and imaging findings. Importantly, despite widespread steatosis, no cases of advanced hepatic fibrosis detected.

Table 1: Baseline demographic and anthropometric characteristics, (n=112).

Variables	Mean±SD/N (%)	Range/IQR
Age (in years)	10.29±2.71	5-15
Male sex	64 (57.1)	-
Female sex	48 (42.9)	-
BMI (kg/m^2)	29.21±3.64	23.17-39.02
BMI >95th percentile	80 (71.4)	-
Weight (kg)	57.57±13.35	32-88
Height (cm)	139.78±14.79	112-182
Systolic BP (mmHg)	110.16±8.62	96-128
Diastolic BP (mmHg)	69.46±6.17	56-83
Any HTN	36 (32.1)	-

Table 2: Biochemical abnormalities in study population.

Parameters	Mean±SD	Abnormal (%)
Fasting glucose (mg/dl)	87.54±13.62	19.6% ≥100
Triglycerides (mg/dl)	164.39±62.66	73.2% >130
HDL (mg/dl)	39.46±7.94	21.4% <35
LDL (mg/dl)	100.04±26.42	8.0% >130
AST (U/l)	38.60±17.04	52.7% >35
ALT (U/l)	43.96±27.47	34.8% >45
GGT (U/l)	28.55±8.07	34.8% >30
ALP (U/l)	198.65±86.47	80.4% >120
TSH (µIU/ml)	-	35.7% >4.12

Table 3: Imaging findings: ultrasound and FibroScan.

Imaging parameters	Value/ N (%)
Ultrasound: Normal	36 (32.1)
Ultrasound: grade 1	56 (50.0)
Ultrasound: grade 2	18 (16.1)
Ultrasound: grade 3	2 (1.8)
CAP score (dB/m), mean±SD	285.74±20.58
Liver stiffness (kPa), mean±SD	5.02±1.01
Fibrosis stage F0-F1	100%
Fibrosis ≥F2	0%

DISCUSSION

The present study provides an important evaluation of the burden of fatty liver disease among obese children and highlights how obesity, metabolic abnormalities, and liver involvement are strongly interconnected. Childhood obesity has been well established as a global phenomenon, with its prevalence rising not only in developed countries but also in transitional and low-income nations as reported by Barry and Colleen. Their assessment of obesity trends worldwide illustrates that many regions have reached obesity levels comparable to Western nations, supporting the global relevance of the current study.[1]

In the present cohort, higher BMI was consistently associated with increased severity of hepatic steatosis on ultrasonography and FibroScan. This aligns with the findings of Moran et al who first identified steatohepatitis as a cause of chronic liver dysfunction in obese children, and Kinugasa et al who described fibrotic changes in livers even in simple pediatric obesity.[28,29] Their work emphasizes that obesity in childhood is not a benign condition but can initiate structural liver injury at an early age.

Similarly, Franzese et al reported liver involvement-including fatty infiltration and inflammation-in obese children, paralleling the observations of the current study where increased BMI and waist-related indices correlated with more severe steatosis. In a Japanese population, Tominaga et al showed that fatty liver could be detected as early as 6 years of age, with a strong positive relationship between the degree of obesity and steatosis, reaffirming the association found in our cohort.[30]

Biochemical parameters further supported these imaging findings. The present study identified significantly elevated ALT levels in children with advanced fatty liver disease. Similar patterns were observed by Strauss et al who reported a high prevalence of abnormal ALT among overweight and obese adolescents. Likewise, Tazawa et al demonstrated that ALT activity is strongly linked with hepatic steatosis in obese children.[14,15] These biochemical abnormalities reflect underlying hepatic inflammation and help reinforce ALT as an accessible marker for early detection, despite its limitations.

Several studies have emphasized that ALT alone is not always sufficient for accurate diagnosis. Clark et al and Brunt highlighted that aminotransferase levels may remain within the normal range in early steatosis and may vary significantly across populations.[10,31] Nonetheless, when combined with imaging modalities such as ultrasonography or FibroScan, ALT significantly improves diagnostic accuracy. The current study supports this view: ultrasonography and FibroScan CAP measurements helped differentiate degrees of hepatic steatosis and correlated well with ALT values. The utility of FibroScan as a non-invasive tool is reinforced by Al-Ghamdi who noted its reliability in fibrosis assessment.[27]

Metabolic dysfunction was another central component of disease severity. The study found a strong association between elevated triglycerides and increasing fatty liver grade. This mirrors the findings of Kawasaki et al who showed that hyperinsulinemia and lipid abnormalities correlate directly with hepatic fat accumulation in obese children.[21] Further, Schwimmer et al reported that insulin resistance and other metabolic abnormalities-including dyslipidemia-are key clinicopathological correlates of pediatric NAFLD.[22] These studies reinforce the metabolic underpinnings observed in the present cohort.

The coexistence of elevated blood pressure with higher fatty liver grades in our population highlights a high-risk metabolic phenotype. Blood pressure elevation is known to accompany increased adiposity in children, as demonstrated by Williams et al.[3] Additionally, Visser et al described how chronic low-grade inflammation in overweight children contributes to metabolic dysfunction and cardiovascular risk.[24] The relationship between hepatic steatosis and systemic metabolic dysregulation, as seen in this study, supports the conceptualization of fatty liver disease as a multi-organ metabolic disorder.

Recent shifts in terminology from NAFLD to MAFLD emphasize the central role of obesity and metabolic derangements in disease progression. Eslam et al proposed this new definition to better capture the metabolic drivers of hepatic steatosis, requiring evidence of metabolic dysfunction in addition to hepatic fat deposition.[32] The strong clustering of metabolic disturbances-high BMI, hypertension, elevated ALT, and dyslipidemia-in our cohort aligns well with this redefined framework and supports its applicability in pediatric populations.

Pathophysiologically, findings of present study can be interpreted in context of mechanisms described by Day et al, Tilg et al and Chitturi et al. These authors highlighted insulin resistance, oxidative stress, cytokine imbalance, and lipotoxicity as key drivers of progression from simple steatosis to non-alcoholic steatohepatitis.[17,19,20] Elevated ALT levels, metabolic abnormalities, and imaging evidence of fatty infiltration in this cohort indicate that obese children may already have early manifestations of these pathogenic mechanisms.

Considering the high prevalence of fatty liver disease in the current study and the strong associations with BMI and metabolic markers, early screening becomes essential, however the timing and method of screening differs between various international guidelines.[33] These contrasts underline the need for region-specific data-such as that generated by the present study-to guide local screening and management strategies.

Overall, the study reinforces that pediatric obesity is not merely an anthropometric concern but a systemic metabolic disorder with early hepatic and cardiovascular manifestations. By demonstrating that fatty liver disease in children correlates strongly with BMI, lipid abnormalities, liver enzymes, and blood pressure, the present findings contribute valuable evidence supporting early diagnosis, comprehensive metabolic assessment, and prompt intervention. These steps are essential to prevent progression to NASH, fibrosis, and long-term cardiometabolic complications in this vulnerable population.

Limitations

This study's cross-sectional design limits causal inference and precludes assessment of disease progression over time. The modest sample size and incomplete control of potential confounders, including dietary and physical activity factors, may restrict the generalizability of the findings. Additionally, reliance on non-invasive liver assessments and adult ALT cut-off values may have underestimated the severity of paediatric liver involvement.

CONCLUSION

Elevated BMI in children is significantly associated with adverse liver, metabolic, and cardiovascular parameters, highlighting the interconnected burden of pediatric obesity. These findings emphasize the importance of early detection, routine monitoring, and comprehensive management to reduce the risk of long-term complications such as NAFLD and cardiovascular disease.

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