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Clinical, Anthropometric and Metabolic Profile of Non-Alcoholic Fatty Liver Disease in Obese Adolescents: A Hospital-Based Cross-Sectional Observational Study

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ABS TRAC T

***Background*:** Non-alcoholic fatty liver disease, now designated metabolic dysfunction-associated steatotic liver disease, is the commonest chronic liver disorder of childhood and tracks closely with the rising prevalence of adolescent obesity in India. Because the condition is largely asymptomatic and hepatic transaminases are frequently normal, the clinical and metabolic phenotype that identifies affected adolescents remains incompletely characterised in Indian hospital populations. This study was undertaken to determine the prevalence of the disorder among obese adolescents and to define its clinical, anthropometric, biochemical and sonographic profile. ***Methods*:** A hospital-based cross-sectional observational study was conducted over six months in the Department of Paediatrics of a tertiary care teaching hospital. One hundred and twenty adolescents aged 10 to 19 years with a body mass index at or above the 95th centile for age and sex on the revised Indian Academy of Paediatrics growth charts were enrolled by consecutive sampling. Participants with significant alcohol intake, viral or autoimmune hepatitis, Wilson disease, hepatotoxic drug exposure or secondary obesity were excluded. All underwent anthropometry, blood pressure measurement, fasting biochemistry including transaminases, lipids, glucose and insulin, and abdominal ultrasonography graded by a single blinded radiologist. ***Results*:** Fatty liver was detected in 76 of 120 participants, a prevalence of 63.3% (95% confidence interval 54.6 to 72.0). Affected adolescents had a higher body mass index z-score (2.48±0.44 versus 2.10±0.46; p<0.001), waist-to-height ratio (0.63±0.05 versus 0.58±0.05; p<0.001), alanine aminotransferase (48.6±22.4 versus 26.2±10.8 U/L; p<0.001) and HOMA-IR (5.28±2.46 versus 3.06±1.42; p<0.001), and lower high-density lipoprotein cholesterol (p<0.001). Grade 1, 2 and 3 steatosis occurred in 53.9%, 35.5% and 10.5% respectively, and transaminases, HOMA-IR and triglycerides rose progressively across grades. On multivariable analysis, HOMA-IR, body mass index z-score, waist-to-height ratio, acanthosis nigricans and triglycerides were independently associated with the disorder. ***Conclusion*:** Nearly two-thirds of obese adolescents attending a tertiary centre had sonographic fatty liver, and insulin resistance and central adiposity rather than absolute weight identified those affected. Routine anthropometric and metabolic screening of obese adolescents is warranted.

Keywords: *Non-Alcoholic Fatty Liver Disease, Pediatric Obesity, Adolescent, Insulin Resistance, Metabolic Syndrome, Ultrasonography.*

INTRODUCTION

Non-alcoholic fatty liver disease is defined by the accumulation of fat in more than 5% of hepatocytes in the absence of significant alcohol consumption or any other identifiable cause of hepatic steatosis. In 2023 a multisociety Delphi process replaced the term with metabolic dysfunction-associated steatotic liver disease, an affirmative designation that requires at least one cardiometabolic risk factor for diagnosis and removes the stigmatising and imprecise elements of the older label [1]. A companion paediatric statement subsequently endorsed the new nomenclature while emphasising that steatotic liver disease in children carries features distinct from the adult disorder, including a different histological pattern, a broader differential diagnosis and a substantially longer horizon

of cumulative exposure [2]. The present study uses the older term for continuity with the body of literature from which its comparators are drawn, while recognising that essentially the entire study population would also satisfy the newer criteria.

The epidemiology of the condition in the young has changed markedly within a single generation. Modelling work synthesising global and regional data for individuals aged 5 to 24 years documents a steadily rising burden closely tied to the trajectory of paediatric obesity [3]. Systematic review of population-based studies places the pooled prevalence at approximately 7% in unselected children and adolescents, but this figure rises steeply with adiposity, reaching roughly one-third to one-half in those who are overweight or obese [4]. The seminal autopsy series that first quantified the problem found fatty liver in 9.6% of children aged 2 to 19 years and in 38% of obese children, establishing obesity as the dominant determinant [5]. An international expert consensus on paediatric metabolic dysfunction-associated fatty liver disease later formalised diagnostic criteria specific to this age group, reflecting a recognition that adult definitions map imperfectly onto growing children [6].

India occupies a particular position within this global picture. Systematic review of Indian data documents a rising prevalence of childhood overweight and obesity, most steeply in urban and affluent populations, driven by dietary transition, reduced physical activity and increasing screen exposure [7]. Indian adolescents also display a metabolically adverse body composition at any given body mass index, with a greater proportion of visceral and hepatic fat than their Western counterparts, so that the metabolic consequences of a given degree of overweight are correspondingly greater. Recent reviews of paediatric disease note that prevalence figures vary widely between regions and diagnostic methods, and that data from South Asian hospital cohorts remain comparatively sparse [8].

The clinical importance of identifying affected adolescents lies in the natural history of the disorder. What begins as simple steatosis may progress through steatohepatitis to bridging fibrosis and cirrhosis, and cases requiring transplantation in early adulthood are now well documented. Practice guidance for children emphasises that the disease is frequently silent, that hepatomegaly and right upper quadrant discomfort are inconsistent findings, and that a normal alanine aminotransferase concentration does not exclude significant steatosis [9]. Beyond the liver, the condition is strongly associated with insulin resistance, dyslipidaemia, hypertension, prediabetes and type 2 diabetes, so that its recognition serves as a marker of systemic cardiometabolic risk rather than as an isolated hepatic diagnosis.

Screening practice remains contested. Existing paediatric guidance recommends screening obese children and overweight children with additional risk factors using alanine aminotransferase from the age of nine to eleven years, while acknowledging the limited sensitivity and specificity of that test [10]. Ultrasonography is widely used in practice because it is inexpensive, free of ionising radiation and readily available, although it is insensitive to steatosis involving less than approximately one-third of hepatocytes and is subject to operator variability. Magnetic resonance proton density fat fraction and liver biopsy provide superior accuracy but are impractical for routine use in most Indian institutions on grounds of cost, availability and, for biopsy, acceptability to families of asymptomatic adolescents.

Against this background, several questions of direct practical relevance remain unanswered for the Indian hospital setting. The prevalence of sonographic fatty liver among obese adolescents attending tertiary care in this country is imprecisely characterised, with published estimates spanning a wide range. It is unclear which components of the clinical assessment, whether simple anthropometric indices such as the waist-to-height ratio, readily observed physical signs such as acanthosis nigricans, or laboratory measures of insulin resistance, best discriminate between obese adolescents with and without hepatic steatosis. It is likewise unclear how closely the severity of sonographic steatosis tracks the underlying metabolic derangement, a relationship that would determine whether ultrasonographic grading carries prognostic as well as diagnostic information.

The present study was therefore designed to determine the prevalence of non-alcoholic fatty liver disease among obese adolescents attending a tertiary care hospital, and to delineate the clinical, anthropometric, biochemical and sonographic characteristics that distinguish those affected. By examining the relationship between severity of steatosis and the accompanying metabolic profile, and by testing the discriminatory performance of simple bedside and laboratory measures, the study aimed to provide practical guidance for the identification of adolescents who warrant hepatic evaluation in settings where advanced imaging is not routinely accessible.

Aims and Objectives

The aim of the present study was to determine the prevalence of non-alcoholic fatty liver disease among obese adolescents attending a tertiary care teaching hospital and to characterise the clinical, anthropometric, biochemical and sonographic profile of those affected, so that adolescents warranting hepatic assessment could be identified using measures available in routine practice.

The primary objective was to estimate the prevalence of ultrasonographically detected non-

alcoholic fatty liver disease among adolescents aged 10 to 19 years with a body mass index at or above the 95th centile for age and sex. The secondary objectives were to compare demographic, clinical and anthropometric characteristics between obese adolescents with and without hepatic steatosis, with particular attention to the body mass index z-score, waist circumference, waist-to-height ratio, blood pressure and the presence of acanthosis nigricans; to compare the biochemical profile between the two groups, including hepatic transaminases, gamma-glutamyl transferase, fasting plasma glucose, fasting insulin, the homeostasis model assessment of insulin resistance, glycated haemoglobin, the fasting lipid profile and serum uric acid; to determine the distribution of ultrasonographic grades of steatosis and to examine whether anthropometric and metabolic derangement increased in a graded fashion across those grades; to determine the frequency of the metabolic syndrome and of its individual components in each group; and to identify independent predictors of hepatic steatosis using multivariable logistic regression. An additional objective was to assess the discriminatory performance of alanine aminotransferase, the homeostasis model assessment of insulin resistance and the waist-to-height ratio for the detection of hepatic steatosis, and to derive optimal cut-off values for each.

Materials and Methods

Study Design and Setting

This was a hospital-based, cross-sectional, observational study conducted in the Department of Paediatrics at PGIMER, Chandigarh, a tertiary care teaching hospital in Chandigarh, India. Participants were recruited from the paediatric and adolescent outpatient services and from the obesity clinic over a period of 6 months, from September 2025 to February 2026. The protocol was approved by the Institutional Ethics Committee PGIMER/IEC/2025/121, dated July 2025 and the study was conducted in accordance with the Declaration of Helsinki and the National Ethical Guidelines for Biomedical and Health Research Involving Human Participants issued by the Indian Council of Medical Research. Written informed consent was obtained from a parent or legal guardian and written assent was obtained from every participant aged 12 years or older before any study procedure was performed. The report conforms to the STROBE statement for cross-sectional studies.

Participants and Eligibility Criteria

Adolescents of either sex aged 10 to 19 years were screened for eligibility. Participants were included if the body mass index, calculated as weight in kilograms divided by the square of height in metres, was at or above the 95th centile for age and sex on the revised Indian Academy of Paediatrics growth charts for 5 to 18 year old Indian children, and if the parent or guardian and the adolescent consented and assented respectively to participation.

Participants were excluded if there was a history of alcohol consumption exceeding the thresholds specified in the prevailing diagnostic criteria, or any alcohol intake in those below the legal age; serological evidence of hepatitis B or hepatitis C infection; features suggesting autoimmune hepatitis, Wilson disease or alpha-1 antitrypsin deficiency on clinical, biochemical or serological assessment; exposure within the preceding six months to drugs known to cause hepatic steatosis, including corticosteroids, valproate, methotrexate, amiodarone and antiretroviral agents; a known chronic liver disease of any other aetiology; secondary or syndromic obesity, including hypothyroidism, Cushing syndrome, growth hormone deficiency and recognised genetic obesity syndromes; known type 1 diabetes mellitus; pregnancy; and any acute febrile or systemic illness at the time of assessment. Screening thyroid function was performed in all participants and those with untreated hypothyroidism were excluded.

Sample Size Determination

The sample size was calculated for the primary objective of estimating prevalence using the single-proportion formula, $n = Z^2p(1-p)/d^2$. Assuming an anticipated prevalence of 62.5%, an absolute precision of 9%, and a two-sided alpha of 0.05, the minimum required sample size was 112. Considering feasibility and the expected proportion of incomplete assessments, a final recruitment target of 120 participants was adopted.

Clinical Assessment and Anthropometry

A structured history was obtained from the adolescent and the accompanying parent using a predesigned and pretested proforma, covering the duration of obesity, dietary pattern, frequency of consumption of sweetened beverages and fried foods, duration of moderate to vigorous physical activity, daily screen time, symptoms referable to the liver, symptoms of obstructive sleep apnoea, menstrual history in girls, birth weight, and family history of obesity, type 2 diabetes mellitus, hypertension and liver disease. A complete general and systemic examination was performed, with particular attention to acanthosis nigricans, striae, hepatomegaly and features of syndromic obesity.

Weight was measured to the nearest 0.1 kg on a calibrated digital scale with the participant in light clothing and without footwear, and height to the nearest 0.1 cm using a wall-mounted stadiometer with the head in the Frankfort horizontal plane. The body mass index was calculated and converted to a z-score for age and sex. Waist circumference was measured to the nearest 0.1 cm at the midpoint between the lowest rib and the iliac crest at the end of normal expiration, and the waist-to-height ratio was derived. Blood pressure was recorded as the mean of three readings taken in the right arm after five minutes of seated rest using an appropriately sized cuff, and was interpreted against

age, sex and height centile-specific reference values. Pubertal staging was recorded using the Tanner criteria.

Laboratory Investigations

Venous blood was drawn after an overnight fast of at least ten hours. Alanine aminotransferase, aspartate aminotransferase, gamma-glutamyl transferase, alkaline phosphatase, total and direct bilirubin, total protein and albumin were measured on an automated analyser using standard enzymatic and colorimetric methods. Fasting plasma glucose was measured by the glucose oxidase-peroxidase method and glycated haemoglobin by high-performance liquid chromatography. Fasting serum insulin was measured by chemiluminescent immunoassay, and insulin resistance was quantified using the homeostasis model assessment, calculated as fasting insulin in microunits per millilitre multiplied by fasting glucose in milligrams per decilitre divided by 405. The fasting lipid profile comprised total cholesterol, triglycerides and high-density lipoprotein cholesterol measured enzymatically, with low-density lipoprotein cholesterol derived by the Friedewald equation where triglycerides were below 400 mg/dL. Serum uric acid, creatinine, thyroid stimulating hormone, hepatitis B surface antigen and anti-hepatitis C virus antibody were also assayed. All samples were processed in the institutional central laboratory, which participates in an external quality assurance programme.

Ultrasonographic Assessment

Abdominal ultrasonography was performed using a GE LOGIQ P9 ultrasound system (GE Healthcare, USA) equipped with a 3–5 MHz curvilinear transducer. All examinations were performed by a radiologist who was blinded to the participants' clinical and biochemical data. Hepatic steatosis was diagnosed based on increased hepatic echogenicity relative to the renal cortex, attenuation of the ultrasound beam in the deeper hepatic parenchyma, and reduced visualization of the diaphragm and intrahepatic vessel walls. Steatosis severity was graded as grade 1 (mild) when there was a diffuse, minimal increase in hepatic echogenicity with preserved visualization of the diaphragm and intrahepatic vessel borders; grade 2 (moderate) when there was a moderate increase in echogenicity with impaired visualization of the intrahepatic vessels and diaphragm; and grade 3 (severe) when there was marked hepatic echogenicity with poor or absent visualization of the intrahepatic vessels, diaphragm, and posterior segment of the right hepatic lobe. Liver span was measured along the midclavicular line. A randomly selected 15% of ultrasound examinations were independently reassessed by a second radiologist to estimate inter-observer agreement, which was quantified using the weighted kappa (κ) statistic.

Operational Definitions

Obesity was defined as a body mass index at or above the 95th centile for age and sex on the revised Indian Academy of Paediatrics charts. Non-alcoholic fatty liver disease was defined as the presence of ultrasonographic hepatic steatosis of any grade in the absence of the exclusion criteria specified above. Insulin resistance was defined as a homeostasis model assessment value at or above 3.16 for the age group studied. The metabolic syndrome was defined according to the International Diabetes Federation consensus criteria for children and adolescents, requiring central obesity together with at least two of raised triglycerides, reduced high-density lipoprotein cholesterol, raised blood pressure and raised fasting plasma glucose. Prediabetes was defined as a fasting plasma glucose of 100 to 125 mg/dL or a glycated haemoglobin of 5.7% to 6.4%. Elevated alanine aminotransferase was defined as a value exceeding 40 U/L, and elevated blood pressure as a systolic or diastolic value at or above the 95th centile for age, sex and height.

Statistical Analysis

Data were entered into a Microsoft Excel spreadsheet and analysed using SPSS version 26.0 IBM Corporation, Armonk, New York, United States. The distribution of continuous variables was assessed using the Shapiro-Wilk test and by inspection of histograms and normal quantile-quantile plots. Normally distributed continuous variables were expressed as mean with standard deviation and compared between the two groups using the independent samples t test; skewed variables were expressed as median with interquartile range and compared using the Mann-Whitney U test. Categorical variables were expressed as frequencies with percentages and compared using the chi-square test, with the Fisher exact test applied where any expected cell frequency was below five. The prevalence of hepatic steatosis was reported with a 95% confidence interval calculated by the Wilson score method.

Comparisons across the three ultrasonographic grades of steatosis were performed using one-way analysis of variance for normally distributed variables, with the Tukey honestly significant difference test for post hoc pairwise comparison, and using the Kruskal-Wallis test with Dunn post hoc testing for skewed variables. Linear trend across ordered grades was assessed using the Jonckheere-Terpstra test. Correlation between continuous variables was examined using the Pearson or Spearman coefficient as appropriate. Variables associated with hepatic steatosis at a p value below 0.10 on univariate analysis, together with variables of established biological relevance, were entered into a multivariable binary logistic regression model using the enter method; results were expressed as adjusted odds ratios with 95% confidence intervals, model calibration was assessed by the Hosmer-Lemeshow goodness-of-fit test and explanatory power by the Nagelkerke R^2. Multicollinearity was examined

using the variance inflation factor. Receiver operating characteristic curve analysis was used to assess the discriminatory performance of alanine aminotransferase, the homeostasis model assessment of insulin resistance and the waist-to-height ratio, with the area under the curve reported with its 95% confidence interval and optimal cut-off values derived by the Youden index. A two-sided p value of less than 0.05 was considered statistically significant throughout.

RESULTS

Of 150 obese adolescents screened during the study period, 120 satisfied the eligibility criteria and completed all clinical, biochemical and sonographic assessments and were included in the final analysis. Hepatic steatosis was detected on ultrasonography in 76 participants, giving a prevalence of non-alcoholic fatty liver disease of 63.3% (95% confidence interval 54.6 to 72.0). Inter-observer agreement for the sonographic grading of steatosis in the 18 scans reassessed by a second radiologist was substantial (weighted kappa 0.82). The prevalence was 69.1% among boys and 55.8% among girls, a difference that did not reach statistical significance (p=0.13), and rose progressively across categories of adiposity, from 52.9% in those with a body mass index z-score between 2.0 and 2.5, to 68.3% between 2.5 and 3.0, and 84.2% above 3.0 (test for trend p=0.006). These distributions are displayed in Figure 1.

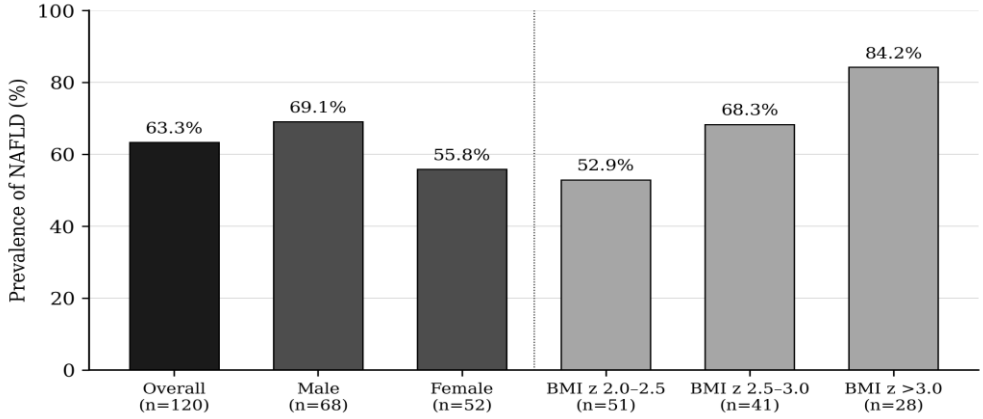

Figure 1: Prevalence of non-alcoholic fatty liver disease in the study population, overall and stratified by sex and by category of body mass index z-score. The test for trend across categories of adiposity was significant (p=0.006)

The demographic and anthropometric characteristics of the whole cohort are summarised in Table 1. The mean age was 14.6±2.4 years and 68 participants (56.7%) were boys. The mean body mass index was 29.8±3.6 kg/m² with a mean z-score of 2.34±0.48, and the mean waist circumference was 94.2±9.8 cm. Acanthosis nigricans was present in 74 participants (61.7%). A sedentary pattern, defined as screen exposure exceeding two hours daily, was reported by 89 participants (74.2%), and 96 participants (80.0%) reported less than sixty minutes of moderate to vigorous physical activity per day. A family history of type 2 diabetes mellitus was present in 58 participants (48.3%).

Table 1: Demographic, anthropometric and lifestyle characteristics of the study population (n=120)

Characteristic	Frequency or mean ± SD	Percentage	Range
Age, years	14.6 ± 2.4	—	10–19
Male sex	68	56.7	—
Female sex	52	43.3	—
Age group 10–13 years	44	36.7	—
Age group 14–16 years	48	40.0	—
Age group 17–19 years	28	23.3	—
Body mass index, kg/m²	29.8 ± 3.6	—	24.2–41.6
Body mass index z-score	2.34 ± 0.48	—	2.01–3.86
Waist circumference, cm	94.2 ± 9.8	—	76–124
Waist-to-height ratio	0.61 ± 0.06	—	0.51–0.78
Acanthosis nigricans present	74	61.7	—
Clinical hepatomegaly	22	18.3	—
Screen time >2 hours/day	89	74.2	—
Physical activity <60 minutes/day	96	80.0	—
Sweetened beverage intake ≥1/day	71	59.2	—
Family history of type 2 diabetes	58	48.3	—

Family history of obesity	62	51.7	—
Tanner stage IV–V	74	61.7	—

SD, standard deviation.

Comparison of clinical and anthropometric variables between adolescents with and without hepatic steatosis is presented in Table 2. Participants with steatosis were older (15.1±2.2 versus 13.8±2.5 years; p=0.004) and had a significantly higher body mass index (30.9±3.4 versus 27.9±3.2 kg/m²; p<0.001), body mass index z-score (2.48±0.44 versus 2.10±0.46; p<0.001), waist circumference (97.6±9.1 versus 88.4±8.2 cm; p<0.001) and waist-to-height ratio (0.63±0.05 versus 0.58±0.05; p<0.001). Acanthosis nigricans was substantially more frequent in the group with steatosis, occurring in 56 participants (73.7%) compared with 18 participants (40.9%) without steatosis (p<0.001). Mean systolic blood pressure was higher in the group with steatosis (118.4±10.2 versus 112.6±9.4 mmHg; p=0.002), as was mean diastolic blood pressure (76.2±7.4 versus 72.8±7.0 mmHg; p=0.014), and elevated blood pressure at or above the 95th centile was present in 18 participants (23.7%) compared with 4 participants (9.1%) respectively (p=0.045). Male sex was more common among those with steatosis but the difference did not reach significance (61.8% versus 47.7%; p=0.13). Symptoms attributable to the liver were uncommon and did not differ significantly between the groups, with right upper quadrant discomfort reported by 11 participants (14.5%) with steatosis and 4 participants (9.1%) without (p=0.39).

Table 2: Clinical and anthropometric characteristics by presence of non-alcoholic fatty liver disease

Variable	NAFLD present (n=76)	NAFLD absent (n=44)	p value
Age, years	15.1 ± 2.2	13.8 ± 2.5	0.004
Male sex, n (%)	47 (61.8)	21 (47.7)	0.13
Body mass index, kg/m²	30.9 ± 3.4	27.9 ± 3.2	<0.001
Body mass index z-score	2.48 ± 0.44	2.10 ± 0.46	<0.001
Waist circumference, cm	97.6 ± 9.1	88.4 ± 8.2	<0.001
Waist-to-height ratio	0.63 ± 0.05	0.58 ± 0.05	<0.001
Acanthosis nigricans, n (%)	56 (73.7)	18 (40.9)	<0.001
Clinical hepatomegaly, n (%)	19 (25.0)	3 (6.8)	0.014
Systolic blood pressure, mmHg	118.4 ± 10.2	112.6 ± 9.4	0.002
Diastolic blood pressure, mmHg	76.2 ± 7.4	72.8 ± 7.0	0.014
Blood pressure ≥95th centile, n (%)	18 (23.7)	4 (9.1)	0.045
Right upper quadrant discomfort, n (%)	11 (14.5)	4 (9.1)	0.39
Easy fatigability, n (%)	26 (34.2)	11 (25.0)	0.29
Screen time >2 hours/day, n (%)	60 (78.9)	29 (65.9)	0.11
Physical activity <60 minutes/day, n (%)	65 (85.5)	31 (70.5)	0.045
Family history of type 2 diabetes, n (%)	44 (57.9)	14 (31.8)	0.006
Tanner stage IV–V, n (%)	52 (68.4)	22 (50.0)	0.045

NAFLD, non-alcoholic fatty liver disease. Values are mean ± standard deviation unless otherwise stated. Continuous variables compared by the independent samples t test; categorical variables by the chi-square test.

The biochemical profile of the two groups is set out in Table 3. Mean alanine aminotransferase was almost twice as high among participants with steatosis (48.6±22.4 versus 26.2±10.8 U/L; p<0.001), and aspartate aminotransferase followed the same pattern (36.4±15.2 versus 24.8±8.6 U/L; p<0.001). The aspartate to alanine aminotransferase ratio was correspondingly lower in the group with steatosis (0.78±0.18 versus 0.98±0.22; p<0.001). Notably, alanine aminotransferase exceeded the conventional threshold of 40 U/L in only 42 of the 76 participants with steatosis (55.3%), so that 34 affected adolescents (44.7%) would have been missed by transaminase-based screening alone. Gamma-glutamyl transferase was also higher in the group with steatosis (32.6±14.8 versus 21.4±8.2 U/L; p<0.001).

Indices of glucose metabolism showed the same direction of difference. Fasting plasma glucose was 94.8±10.6 mg/dL in the group with steatosis and 89.6±8.4 mg/dL in the group without (p=0.005), fasting insulin was 22.4±9.6 versus 13.8±6.2 µIU/mL (p<0.001), and the homeostasis model assessment of insulin resistance was 5.28±2.46 versus 3.06±1.42 (p<0.001). Glycated haemoglobin was modestly but significantly higher in the group with steatosis (5.6±0.5% versus 5.4±0.4%; p=0.021). The lipid profile was more atherogenic in the presence of steatosis, with higher triglycerides (148.2±52.6 versus 112.4±38.2 mg/dL; p<0.001), higher total cholesterol (178.6±32.4 versus 164.2±28.6 mg/dL; p=0.016), higher low-density lipoprotein cholesterol (108.6±28.2 versus 98.4±25.4 mg/dL; p=0.048) and lower high-density lipoprotein cholesterol (38.4±7.2 versus 44.6±8.1 mg/dL; p<0.001). Serum uric acid was also significantly higher in the group with steatosis (6.2±1.4 versus 5.2±1.2 mg/dL; p<0.001).

Table 3: Biochemical and metabolic profile by presence of non-alcoholic fatty liver disease

Parameter	NAFLD present (n=76)	NAFLD absent (n=44)	p value
Alanine aminotransferase, U/L	48.6 ± 22.4	26.2 ± 10.8	<0.001
Aspartate aminotransferase, U/L	36.4 ± 15.2	24.8 ± 8.6	<0.001
AST/ALT ratio	0.78 ± 0.18	0.98 ± 0.22	<0.001
ALT >40 U/L, n (%)	42 (55.3)	5 (11.4)	<0.001
Gamma-glutamyl transferase, U/L	32.6 ± 14.8	21.4 ± 8.2	<0.001
Alkaline phosphatase, U/L	196.4 ± 62.8	188.2 ± 58.4	0.48
Serum albumin, g/dL	4.28 ± 0.32	4.34 ± 0.30	0.31
Fasting plasma glucose, mg/dL	94.8 ± 10.6	89.6 ± 8.4	0.005
Fasting insulin, µIU/mL	22.4 ± 9.6	13.8 ± 6.2	<0.001
HOMA-IR	5.28 ± 2.46	3.06 ± 1.42	<0.001
Glycated haemoglobin, %	5.6 ± 0.5	5.4 ± 0.4	0.021
Total cholesterol, mg/dL	178.6 ± 32.4	164.2 ± 28.6	0.016
Triglycerides, mg/dL	148.2 ± 52.6	112.4 ± 38.2	<0.001
HDL cholesterol, mg/dL	38.4 ± 7.2	44.6 ± 8.1	<0.001
LDL cholesterol, mg/dL	108.6 ± 28.2	98.4 ± 25.4	0.048
Serum uric acid, mg/dL	6.2 ± 1.4	5.2 ± 1.2	<0.001
Insulin resistance (HOMA-IR ≥3.16), n (%)	62 (81.6)	18 (40.9)	<0.001

NAFLD, non-alcoholic fatty liver disease; ALT, alanine aminotransferase; AST, aspartate aminotransferase; HOMA-IR, homeostasis model assessment of insulin resistance; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Among the 76 participants with hepatic steatosis, grade 1 or mild steatosis was present in 41 (53.9%), grade 2 or moderate steatosis in 27 (35.5%) and grade 3 or severe steatosis in 8 (10.5%). This distribution is shown in Figure 2. Anthropometric and metabolic derangement increased in a graded fashion across the three categories, as detailed in Table 4. The body mass index z-score rose from 2.36±0.40 in grade 1 to 2.56±0.42 in grade 2 and 2.74±0.46 in grade 3 (p=0.021), and waist circumference rose from 94.6±8.2 cm to 99.8±8.6 cm and 106.2±9.4 cm respectively (p<0.001). Alanine aminotransferase rose from 39.8±16.2 U/L in grade 1 to 54.2±20.4 U/L in grade 2 and 72.6±26.8 U/L in grade 3 (p<0.001), and the homeostasis model assessment of insulin resistance rose from 4.42±1.86 to 5.86±2.32 and 7.64±3.04 respectively (p<0.001). Post hoc testing confirmed significant differences between grade 1 and grade 3 for both parameters (p<0.001 for each), and the Jonckheere-Terpstra test confirmed a significant ordered trend across grades (p<0.001). These gradients are illustrated in Figure 3.

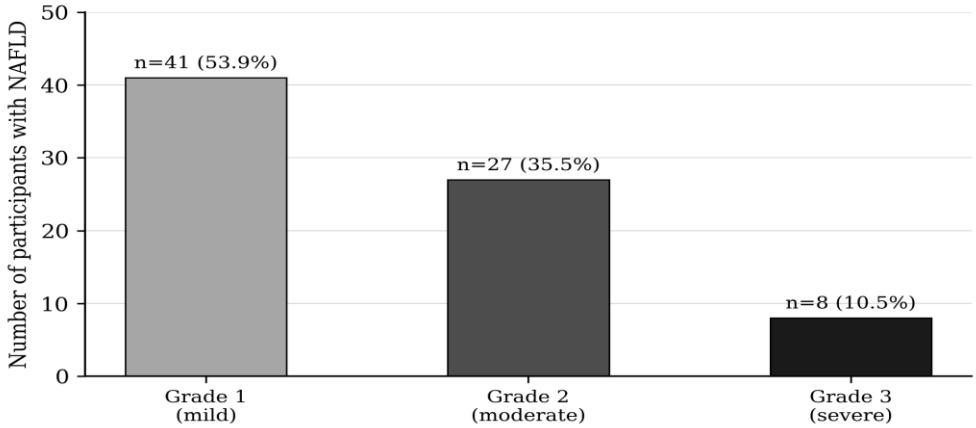

Figure 2: Distribution of ultrasonographic grade of hepatic steatosis among the 76 participants with non-alcoholic fatty liver disease

Table 4: Anthropometric and metabolic parameters across ultrasonographic grades of hepatic steatosis (n=76)

Parameter	Grade 1 (n=41)	Grade 2 (n=27)	Grade 3 (n=8)	p value
Age, years	14.6 ± 2.2	15.4 ± 2.1	16.2 ± 1.8	0.084
Body mass index, kg/m²	29.8 ± 3.0	31.6 ± 3.2	33.4 ± 3.6	0.004
Body mass index z-score	2.36 ± 0.40	2.56 ± 0.42	2.74 ± 0.46	0.021
Waist circumference, cm	94.6 ± 8.2	99.8 ± 8.6	106.2 ± 9.4	<0.001
Waist-to-height ratio	0.61 ± 0.04	0.64 ± 0.05	0.68 ± 0.05	<0.001

Alanine aminotransferase, U/L	39.8 ± 16.2	54.2 ± 20.4	72.6 ± 26.8	<0.001
Aspartate aminotransferase, U/L	31.2 ± 11.4	39.6 ± 14.8	52.4 ± 19.6	<0.001
Fasting insulin, µIU/mL	19.2 ± 7.8	24.6 ± 9.4	31.8 ± 11.6	<0.001
HOMA-IR	4.42 ± 1.86	5.86 ± 2.32	7.64 ± 3.04	<0.001
Triglycerides, mg/dL	132.4 ± 42.6	158.6 ± 52.4	192.8 ± 64.2	0.002
HDL cholesterol, mg/dL	40.2 ± 6.8	37.1 ± 6.9	33.6 ± 6.2	0.014
Serum uric acid, mg/dL	5.8 ± 1.2	6.4 ± 1.4	7.2 ± 1.6	0.008
Liver span, cm	13.8 ± 1.2	14.6 ± 1.4	15.8 ± 1.6	<0.001
Metabolic syndrome, n (%)	14 (34.1)	14 (51.9)	6 (75.0)	0.048

HOMA-IR, homeostasis model assessment of insulin resistance; HDL, high-density lipoprotein. Comparison across the three grades by one-way analysis of variance with Tukey post hoc testing for continuous variables and by the chi-square test for proportions.

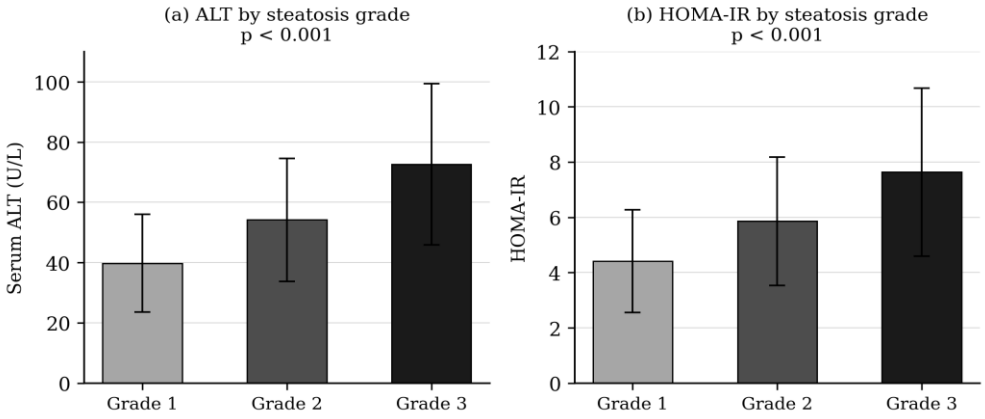

Figure 3: Mean serum alanine aminotransferase (panel a) and homeostasis model assessment of insulin resistance (panel b) across ultrasonographic grades of hepatic steatosis. Error bars denote one standard deviation. Both gradients were significant on one-way analysis of variance and on the Jonckheere-Terpstra test for ordered trend

The frequency of metabolic comorbidity by steatosis status is presented in Table 5. The metabolic syndrome, defined by the International Diabetes Federation consensus criteria for this age group, was present in 34 participants (44.7%) with steatosis and in 6 participants (13.6%) without (p<0.001). Hypertriglyceridaemia was recorded in 33 participants (43.4%) versus 8 participants (18.2%) respectively (p=0.005) and a reduced high-density lipoprotein cholesterol in 46 participants (60.5%) versus 14 participants (31.8%) (p=0.002). Impaired fasting glucose was present in 16 participants (21.1%) versus 3 participants (6.8%) (p=0.038), and prediabetes defined by glycated haemoglobin in 21 participants (27.6%) versus 5 participants (11.4%) (p=0.037). Four participants (5.3%), all in the group with steatosis, met criteria for type 2 diabetes mellitus, although this difference did not reach statistical significance (p=0.29). Hyperuricaemia occurred in 29 participants (38.2%) with steatosis and 6 participants (13.6%) without (p=0.004). Among the 52 female participants, polycystic ovary syndrome was diagnosed in 11 of 29 girls (37.9%) with steatosis and in 4 of 23 girls (17.4%) without, a difference that did not reach statistical significance (p=0.10).

Table 5: Frequency of metabolic comorbidity by presence of non-alcoholic fatty liver disease

Comorbidity	NAFLD present (n=76)	NAFLD absent (n=44)	p value
Metabolic syndrome, n (%)	34 (44.7)	6 (13.6)	<0.001
Hypertriglyceridaemia (≥150 mg/dL), n (%)	33 (43.4)	8 (18.2)	0.005
Low HDL cholesterol, n (%)	46 (60.5)	14 (31.8)	0.002
Elevated LDL cholesterol, n (%)	24 (31.6)	9 (20.5)	0.19
Any dyslipidaemia, n (%)	58 (76.3)	20 (45.5)	<0.001
Elevated blood pressure, n (%)	18 (23.7)	4 (9.1)	0.045
Impaired fasting glucose, n (%)	16 (21.1)	3 (6.8)	0.038
Prediabetes (HbA1c 5.7–6.4%), n (%)	21 (27.6)	5 (11.4)	0.037
Type 2 diabetes mellitus, n (%)	4 (5.3)	0 (0.0)	0.29
Insulin resistance (HOMA-IR ≥3.16), n (%)	62 (81.6)	18 (40.9)	<0.001
Hyperuricaemia, n (%)	29 (38.2)	6 (13.6)	0.004
Symptoms of obstructive sleep apnoea, n (%)	14 (18.4)	3 (6.8)	0.078

Polycystic ovary syndrome (females only), n (%)	11/29 (37.9)	4/23 (17.4)	0.10

NAFLD, non-alcoholic fatty liver disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; HbA1c, glycated haemoglobin; HOMA-IR, homeostasis model assessment of insulin resistance. Metabolic syndrome defined by International Diabetes Federation consensus criteria for children and adolescents.

Multivariable binary logistic regression, presented in Table 6, identified five variables independently associated with the presence of hepatic steatosis. The homeostasis model assessment of insulin resistance carried the most consistent association, with each unit increase raising the adjusted odds by 62% (adjusted odds ratio 1.62; 95% confidence interval 1.24 to 2.12; p<0.001). Each standard deviation increase in body mass index z-score approximately trebled the odds (adjusted odds ratio 2.86; 95% confidence interval 1.42 to 5.76; p=0.003), and each 0.05 increment in the waist-to-height ratio approximately doubled them (adjusted odds ratio 2.14; 95% confidence interval 1.26 to 3.64; p=0.005). Acanthosis nigricans (adjusted odds ratio 2.48; 95% confidence interval 1.02 to 6.03; p=0.045) and serum triglycerides per 10 mg/dL increment (adjusted odds ratio 1.14; 95% confidence interval 1.03 to 1.26; p=0.011) retained independent significance. Male sex, age and family history of type 2 diabetes were not independently associated once these variables were accounted for. The model showed satisfactory calibration on the Hosmer-Lemeshow test (χ^2=6.24; p=0.62), explained approximately half of the variance in outcome (Nagelkerke R^2=0.48) and correctly classified 79.2% of participants. No variance inflation factor exceeded 2.4.

Table 6: Multivariable logistic regression analysis for predictors of non-alcoholic fatty liver disease (n=120)

Predictor variable	Adjusted OR	95% confidence interval	p value
HOMA-IR (per 1 unit)	1.62	1.24 to 2.12	<0.001
Body mass index z-score (per 1 SD)	2.86	1.42 to 5.76	0.003
Waist-to-height ratio (per 0.05)	2.14	1.26 to 3.64	0.005
Acanthosis nigricans (present)	2.48	1.02 to 6.03	0.045
Triglycerides (per 10 mg/dL)	1.14	1.03 to 1.26	0.011
Male sex	1.56	0.64 to 3.80	0.33
Age (per year)	1.18	0.96 to 1.45	0.11
Family history of type 2 diabetes	1.84	0.76 to 4.46	0.18
Physical activity <60 minutes/day	1.72	0.66 to 4.48	0.27

OR, odds ratio; HOMA-IR, homeostasis model assessment of insulin resistance; SD, standard deviation. Model summary: Hosmer-Lemeshow $\chi^2 = 6.24$, $p = 0.62$; Nagelkerke $R^2 = 0.48$; overall correct classification 79.2%.

Receiver operating characteristic curve analysis, displayed in Figure 4, was performed to assess the discriminatory performance of three readily available measures. Alanine aminotransferase yielded an area under the curve of 0.812 (95% confidence interval 0.734 to 0.890; p<0.001), with an optimal cut-off of 32.5 U/L providing a sensitivity of 76.3% and a specificity of 77.3%. The homeostasis model assessment of insulin resistance yielded an area under the curve of 0.784 (95% confidence interval 0.698 to 0.870; p<0.001), with an optimal cut-off of 4.15 providing a sensitivity of 71.1% and a specificity of 75.0%. The waist-to-height ratio yielded an area under the curve of 0.746 (95% confidence interval 0.654 to 0.838; p<0.001), with an optimal cut-off of 0.60 providing a sensitivity of 68.4% and a specificity of 72.7%. The optimal alanine aminotransferase cut-off derived here was appreciably lower than the conventional laboratory threshold of 40 U/L, consistent with the observation that almost half of the affected adolescents had transaminase values within the reported reference range. Alanine aminotransferase correlated moderately with the homeostasis model assessment of insulin resistance (r=0.52; p<0.001) and with waist circumference (r=0.46; p<0.001).

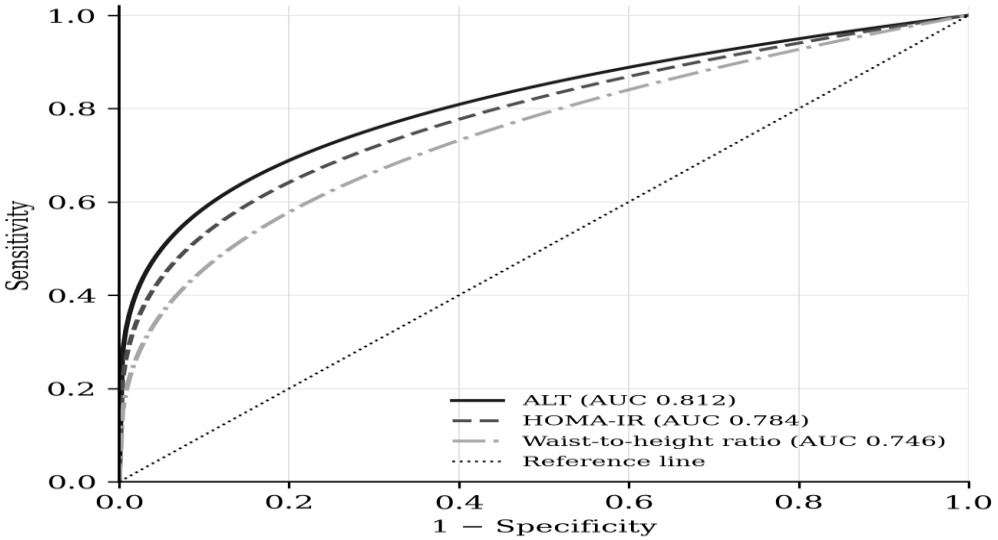

Figure 4: Receiver operating characteristic curves for alanine aminotransferase, the homeostasis model assessment of insulin resistance and the waist-to-height ratio in the detection of ultrasonographic hepatic steatosis among obese adolescents

DISCUSSION

This cross-sectional study of 120 obese adolescents attending a tertiary care hospital found sonographic evidence of hepatic steatosis in 63.3%. Those affected were distinguished not by weight alone but by a coherent constellation of central adiposity, insulin resistance, atherogenic dyslipidaemia and hyperuricaemia, and the severity of steatosis tracked the degree of that metabolic derangement. Almost half of the affected adolescents had an alanine aminotransferase within the conventional reference range, and the optimal discriminatory threshold derived on receiver operating characteristic analysis was 32.5 U/L, well below the usual laboratory cut-off.

The prevalence observed here sits at the upper end of the published range for clinically obese young people but is not exceptional. A systematic review and meta-analysis of paediatric studies reported a pooled prevalence of approximately 7% in the general population rising to about 34% in obese children, with wide variation attributable to differences in diagnostic modality, age range and obesity definition [4]. A more recent meta-analysis restricted to children and adolescents with overweight or obesity reported a pooled prevalence of 39.2% in the combined group and 52.5% among those with obesity, with boys more frequently affected than girls [11]. Analysis of United States national survey data using transient elastography found that approximately 70% of obese adolescents met criteria for steatotic liver disease [12]. The figure of 63.3% reported here falls between these estimates and is consistent with the composition of the present sample, which was drawn from a tertiary obesity clinic and therefore enriched for severe adiposity, as reflected in the mean body mass index z-score of 2.34.

The graded relationship between severity of steatosis and metabolic derangement is one of the more clinically useful findings. Body mass index z-score, waist circumference, transaminases, insulin resistance and triglycerides all rose monotonically from grade 1 to grade 3, and the frequency of the metabolic syndrome rose from 34.1% to 75.0% across the same gradient. This supports the interpretation that ultrasonographic grading, despite its acknowledged limitations, carries information beyond the binary presence or absence of steatosis. Histological studies of paediatric disease have similarly demonstrated that increasing steatosis grade accompanies increasing inflammatory activity and fibrosis stage, although the paediatric pattern of periportal rather than centrilobular injury differs from that seen in adults.[13] The present finding cannot be equated with histological progression, but it does suggest that an adolescent reported to have grade 3 steatosis warrants closer metabolic evaluation than one with grade 1.

The observation that 44.7% of adolescents with sonographic steatosis had an alanine aminotransferase of 40 U/L or below has direct implications for screening practice. Current paediatric guidance recommends alanine aminotransferase as the primary screening test in obese children from the age of nine to eleven years, while acknowledging its imperfect performance [10]. Referral-based data have shown that a substantial proportion of overweight and obese children referred for suspected fatty liver have normal or only marginally elevated transaminases, and that the biochemical threshold used materially alters the apparent yield of screening [15]. The present analysis, which derived an optimal cut-off of 32.5 U/L, is consistent with the argument that sex-specific upper limits of normal considerably lower than 40 U/L should be applied in adolescents. Applying the conventional threshold in this cohort would have failed to identify 34

of 76 affected adolescents.

Insulin resistance emerged as the single most robust independent correlate of steatosis, with each unit increase in the homeostasis model assessment raising the adjusted odds by 62%, and 81.6% of affected adolescents met the threshold for insulin resistance compared with 40.9% of those without steatosis. This is mechanistically coherent, since hepatic and peripheral insulin resistance drives increased free fatty acid flux to the liver and de novo lipogenesis, and it accords with the established association between paediatric fatty liver and disorders of glucose metabolism. A large multicentre paediatric study documented prediabetes in approximately one-quarter and type 2 diabetes in a small but appreciable minority of children with biopsy-confirmed disease [14]. The corresponding figures in the present cohort, prediabetes in 27.6% and type 2 diabetes in 5.3% of those with steatosis, are closely comparable and reinforce the argument that fatty liver in an obese adolescent should trigger formal glycaemic assessment rather than reassurance.

The finding that the waist-to-height ratio discriminated almost as well as laboratory measures deserves emphasis in the Indian context. A ratio of 0.60 provided a sensitivity of 68.4% and a specificity of 72.7% in this cohort, using nothing more than a tape measure. South Asian populations accumulate visceral and hepatic fat disproportionately at a given body mass index, and simple indices of central adiposity may therefore outperform weight-based measures in this setting. Indian guidance on the nomenclature, diagnosis and treatment of fatty liver disease has similarly emphasised the importance of central adiposity and of applying ethnicity-appropriate anthropometric thresholds [16]. Acanthosis nigricans, present in 73.7% of affected adolescents and independently associated with steatosis on multivariable analysis, offers a second no-cost bedside marker.

Contrasting evidence exists and should be acknowledged. Studies using transient elastography or magnetic resonance based methods report prevalence estimates and severity distributions that differ appreciably from ultrasound-based series, because ultrasonography is insensitive to steatosis affecting less than approximately 30% of hepatocytes and cannot quantify fat fraction [17]. Recent guidance on imaging-based non-invasive assessment accordingly favours controlled attenuation parameter or magnetic resonance proton density fat fraction where available [18]. The prevalence reported here is therefore likely to be an underestimate of true steatosis, and the grade 1 category in particular is likely to encompass a heterogeneous group. Conversely, some cross-sectional series in less severely obese or community-based adolescent populations have reported considerably lower prevalence, which underscores that the present figure applies to a tertiary referral population and should not be extrapolated to obese adolescents in the community. It is also relevant that the newer diagnostic construct requires at least one cardiometabolic criterion, and analyses comparing the older and newer definitions have shown substantial but incomplete overlap, so that a small number of adolescents classified here would not satisfy the current criteria [20].

Several limitations qualify these findings. The cross-sectional design permits no inference about causation or about the temporal sequence linking insulin resistance and hepatic fat accumulation. Ultrasonography rather than histology or magnetic resonance was used to define the outcome, which limits both sensitivity and quantitative precision, although the single blinded reader and the substantial inter-observer agreement mitigate measurement variability. Hepatic fibrosis was not assessed, so the proportion of participants with steatohepatitis or advanced disease is unknown. Dietary intake was assessed by recall rather than by validated food frequency questionnaire, and physical activity was self-reported, both of which are subject to reporting bias in adolescents with obesity. Genetic determinants, notably the PNPLA3 I148M polymorphism, which is common in South Asian populations and materially modifies risk, were not examined. The single-centre tertiary care setting limits generalisability, and the sample size, while adequate for prevalence estimation, provided limited power for subgroup analyses such as the comparison of polycystic ovary syndrome frequency among female participants. Finally, no follow-up was undertaken, so the clinical trajectory of the cohort remains undefined. Prospective studies with quantitative imaging, fibrosis assessment and longitudinal follow-up are required to establish which obese adolescents progress and over what interval [21].

CONCLUSION

Non-alcoholic fatty liver disease was present in 63.3% of obese adolescents attending a tertiary care hospital, indicating that hepatic steatosis is the rule rather than the exception in this population. Affected adolescents were distinguished by a coherent cardiometabolic phenotype comprising greater central adiposity, higher insulin resistance, atherogenic dyslipidaemia, hyperuricaemia and a higher frequency of the metabolic syndrome, and the severity of sonographic steatosis tracked the degree of that derangement in a graded fashion.

Three findings carry direct practical implications. First, almost half of the affected adolescents had an alanine aminotransferase within the conventional reference range, and the optimal discriminatory threshold in this cohort was 32.5 U/L, so transaminase-based screening using a cut-off of 40 U/L will miss a substantial proportion of cases. Second, the waist-to-height ratio and acanthosis nigricans, both obtainable at no cost during a routine consultation,

discriminated meaningfully between adolescents with and without steatosis and can be used to prioritise those who warrant imaging where resources are constrained. Third, more than a quarter of affected adolescents had prediabetes and one in twenty had established type 2 diabetes, so the detection of fatty liver in an obese adolescent should prompt formal metabolic evaluation rather than reassurance.

Routine anthropometric and metabolic screening of obese adolescents, coupled with a low threshold for hepatic ultrasonography, is therefore warranted in Indian tertiary care practice. Prospective studies incorporating quantitative imaging, assessment of fibrosis and longitudinal follow-up are needed to define which of these adolescents progress and to establish the point at which intervention alters that trajectory.

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