



ANTHROPOMETRY AND METABOLIC ABNORMALITIES IN OBESE CHILDREN WITH NON ALCOHOLIC FATTY LIVER DISEASE

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Abstract:

Background: Obesity has emerged as a global epidemic in children with a spectrum of psycho-social and medical consequences manifesting across the lifespan. Obesity-related morbidities that emerge early in childhood are an alteration in glucose metabolism and fatty infiltration of the liver. Non-alcoholic fatty liver disease (NAFLD), chronic liver disease is a metabolic complication of obesity.

Objective: To identify the prevalence of NAFLD among obese children and anthropometric and metabolic abnormalities among these obese children with NAFLD.

Methodology: This was a cross sectional study included 50 obese children and adolescents, attending Paediatric Endocrine OPD, BIRDEM from June 2010 to December 2010. BMI with 95th centile for age and sex was used as an anthropometric marker to diagnose obesity. Obesity with dismorphism, endocrine, chromosomal abnormalities, chronic liver diseases due to infectious or metabolic causes were excluded from the study. Fasting blood samples were collected for measurement of blood glucose, lipid profile, FT4, TSH and SGPT. Fatty liver was diagnosed by USG which includes increased echogenicity of liver, blurring of vascular margins and increased acoustic attenuation.

Results: The prevalence of fatty liver disease among obese children was 36% with male predominance (M72.2%, F 27.8%). Mild NAFLD was 72% followed by moderate 28%. High Cholesterol, high LDL and high SGPT were more prevalent in obese children with NAFLD which was statistically significant ($P < 0.004$, < 0.05 and < 0.04 respectively).

Conclusion: Obesity with liver disease can get overlooked among the plethora of adverse outcomes related to childhood obesity. Our study was attempted to find out the obese children with NAFLD and related metabolic abnormalities, so that deleberate strategies could be taken to prevent this irreversible liver damage among obese children.

Keywords:

Obesity, NAFLD, Anthropometry, Hyperlipidemia, SGPT.

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Introduction:

Obesity is currently approaching epidemic worldwide. It is escalating both in industrialized and developing countries. It is estimated that about 20% children in developed countries are obese.¹ Childhood obesity is associated with significant co-morbidities. A less well recognized association with obesity is a chronic liver disease, known as non alcoholic fatty liver disease

(NAFLD). Paralleling the increasing prevalence of obesity in the pediatric population, NAFLD is expected to become one of the most common causes of end stage liver disease in both children and young adult.

The theoretical definition of obesity is a degree of somatic overweight that carries detrimental health consequences.² Body mass index (BMI) is an anthropometric marker to diagnose obesity in children.

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In childhood, comparison of BMI to normal curves for age allows the categorization of BMI above the eighty fifth percentile as overweight and above the ninety fifth percentile as obese.¹

Adiposity affects all organs of the body but some systems like cardiovascular and endocrine are affected to a greater extent than others. Obesity plays a central role in the insulin resistance syndrome, which includes hyperinsulinemia, hypertension, hyperlipidemia, type2 diabetes mellitus and an increased risk of atherosclerotic cardiovascular disease.³ It has been proposed that individual component of Metabolic Syndromes (Mets) contribute to a higher risk of NAFLD with insulin resistance being pivotal to the pathogenesis of NAFLD. On this basis, NAFLD has been included as part of the metabolic syndrome or syndrome X.⁴

The prevalence of NAFLD in obese children has been reported to range from 20% to 77%.⁵ Most cases of NAFLD occur in preadolescent and adolescent age group with male predominance. Certain ethnic groups such as Hispanic and Asian may be more susceptible.⁶ Rapidly progressive and high grade obesity in short duration has been emerged as a potentially new risk factor of liver involvement in the pediatric obese population. Hyperglyceridemia rather than hypercholesterolemia may increase the risk of NAFLD. Hypertriglyceridemia and mixed hyperlipidemia have also been independently linked to NAFLD.⁷

NAFLD represents fatty infiltration of the liver in the absence of alcohol consumption. According to the American Association for the Study of Liver Diseases (AASLD), NAFLD is defined as fat accumulation in the liver exceeding 5% to 10% by weight, determined by the percentage of fat-laden hepatocytes on light microscopy.⁸ The clinicopathologic spectrum of NAFLD ranges from isolated fatty infiltration-simple steatosis to nonalcoholic steatohepatitis (NASH) which may progress to fibrosis, cirrhosis and hepatocellular carcinoma.⁹ Hepatic steatosis is characterized by abnormal excessive accumulation of lipids mainly triglyceride in liver.¹⁰ When accumulation of lipids becomes chronic, it induces pressure effect on the architecture of liver; produces inflammation, fibrosis and cirrhosis, leading to a chronic condition termed as non alcoholic steatohepatitis.

Pathologically, NAFLD is a result of complex hepatocellular metabolic dysfunctions in which insulin action is deranged, leading to the deranged metabolism of fat and free fatty acids and subsequent oxidant-mediated damage to the hepatocytes. Progression of pure fatty change to the necro-inflammatory fibrotic stage is a 'two-hit process' proposed by Day and James in 1998.¹⁰ Fat accumulation in the liver is the first hit; a consequence of the imbalance between triglyceride accumulation on one hand and lipid beta-oxidation and export on the other. Fat in the liver makes it vulnerable to the second hit, resulting in hepatocyte injury, inflammation and fibrosis. Insulin resistance is widely implicated in the initiation of NAFLD/NASH. Factors involved in delivering the second hit are thought to include oxidative stress and subsequent lipid per oxidation, proinflammatory cytokines and adipocytokines.¹⁰

Patients with only steatosis had best prognosis whereas patients with advanced steatohepatitis and fibrosis had the worst outcome. According to recent review, steatosis may develop into NASH in one third of cases. Patients with NASH have a significantly increased risk of cirrhosis. Up to 50% of NASH patients can develop fibrosis and 15% to 19% develop cirrhosis. HCC has been observed in 2.4-13% of NASH patients.¹¹ Mortality rate from liver failure in NASH is estimated to be 2-3%¹²

Liver disease may be overlooked among the plethora of adverse outcomes related to childhood obesity. The condition is potentially reversible and preventable. If unchecked, it is likely to progress to steatohepatitis and cirrhosis. Thus, deliberate strategies for helping children to avoid these serious liver problems are warranted in Bangladesh.

Methods:

It was a cross-sectional study conducted in BIRDEM from June 2010 to December 2010. Total 50 Children and adolescents, 8 to 18 years of age, attending paediatric endocrine outpatient department with complaints of excessive weight gain were included in our study. Body mass index was used as an anthropometric marker to diagnose obesity. According to official centers for disease control (CDC), children with BMI $\geq 85^{\text{th}}$ centile were classified as overweight and BMI $\geq 95^{\text{th}}$ centile were classified as obese. Obese children having any dismorphism, endocrine or chromosomal abnormalities or diagnosed case of chronic hepatitis due to metabolic, infectious or

autoimmune cause were excluded from study. A predesigned data collection sheet was used for each subject and information regarding history, clinical examination and investigations were recorded.

Weight was measured by spring scale (Bathroom scale) in kilogram to the nearest 100 gram; standing height was measured by stadiometer and measurement was done to nearest 0.1 cm. The Body mass index (BMI) was calculated as weight in kilogram (KG) divided by square of the height in meter. Waist circumference was measured at the level midway between the lower rib margin and the iliac crest, at the level of umbilicus (in centimeter) with the person breathing out gently and hip circumference was measured at the maximum width over the buttocks at the level of the greater trochanter (in centimeter) by measuring tape. Pubertal staging was done according to Tanner staging. Blood pressure was measured using appropriate size of cuff encircling at least 2/3 rd of upper arm.

Fasting blood samples were collected for the measurement of fasting blood glucose, SGPT, lipid profile, FT4 & TSH. In 2-15 years of age group triglyceride (TG) level ≥ 100 mg/dl was considered high while in 15-19 years of age group TG ≥ 125 mg/dl was considered high. Hypercholesterolemia was defined as serum cholesterol ≥ 170 mg/dl. Low density lipoprotein (LDL) ≥ 110 mg/dl indicated high LDL and High density lipoprotein (HDL) ≤ 35 mg/dl indicated low HDL level. SGPT ≥ 35 U/L was defined as high SGPT.¹³

Real time USG examination of the liver was performed by 3.5 MHz curvilinear transducer using SONOACE 8000 and Sonoline Antares ultrasound machine following 6-hour fast to rule out fatty liver. In USG, a hyperechogenic (bright liver) indicated steatosis.

Informed consent was taken from the parents. SPSS, version 12.0 for Windows software was used for data recording and analysis. Chi-square test and Students t-test were used for comparing group ratios and group averages, respectively. A P value less than 0.05 was considered significant.

Results:

A total of 50 children were enrolled in the study. This was comprised of 30 boys and 20 girls of 8 to 18 years of age. 33 children (66%) belonged to 8-12 years age group while 17 children (34%) belonged to 13-18 years age group. The mean age of the children was

11.24 years. The prevalence of obesity was higher among male (60%) compared to female (40%) with male to female ratio 1.5:1.

In total, 18 obese children had ultrasonographic evidence of nonalcoholic fatty liver among 50 obese children. The prevalence of NAFLD was 36 % which is shown in figure-1.

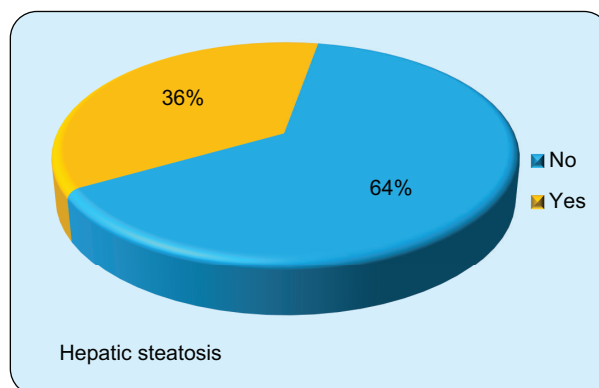


Figure 1: Distribution of the patients according to the presence of NAFLD.

13 children (72%) were found to have mild NAFLD while five (28%) had moderate NAFLD. There was no evidence of severe degree of NAFLD which is shown in figure-2

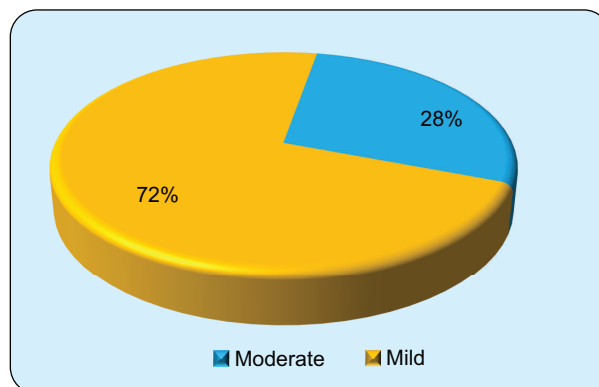


Figure 2: Distribution of the patients according to grading of NAFLD.

Out of twenty girls, five had NAFLD as did thirteen boys. Prevalence of NAFLD was high among male (72.2%) compared to female (27.8%). The mean age of children having NAFLD was 11.67 years.

The mean anthropometrical measurements of obese children with NAFLD is shown in table I.

Table-I*Anthropometric measurements of obese children with NAFLD*

Anthropometric Measurements (Mean) of obese children with NAFLD	
Height	148.32 (± 8.78)
Weight	61.78 (± 16.78)
Waist circumference	72.98 (± 33.79)
Hip circumference	57.40 (± 42.73)
Waist – Hip ratio	0.93 (± 0.05)
BMI	31.08 (± 42.73)

The mean biochemical parameters of obese children with NAFLD is shown in table II.

Table-II*Mean biochemical parameters of obese children with NAFLD*

Biochemical parameters	Mean±SD
SGPT	49.07 (± 23.81)
TG	186.39 (± 42.24)
Cholesterol	191.56(± 28.57)
LDL	119.33 (± 33.25)
HDL	34.77 (± 5.78)
FBG	4.93 (± 0.51)

Table-III: showed comparison of metabolic abnormalities in between two groups. High SGPT, high cholesterol and LDL were significantly common in obese children with NAFLD than children without NAFLD (P < 0.004, <0.05 and < 0.04 respectively).

Table-III*Metabolic abnormalities in obese children and adolescents with and without NAFLD*

Biochemical parameters	NAFLD		Odd ratio	P value
	Yes	No		
High SGPT	11(61.1%)	7(21.9%)	5.61(1.58-19.87)	0.004
High Cholesterol	15(83.9%)	19(59.4%)	3.42(0.82-14.24)	0.05
High TG	16(88.9%)	23(71.9%)	3.13(0.59-16.45)	0.09
High LDL	12(66.7%)	13(40.6%)	2.92(0.87-9.78)	0.04
Low HDL	7(38.9%)	11(34.4%)	1.21(0.37-4.02)	0.38
DM	2(11.1%)	1(3.1%)	3.88(0.33-46.05)	0.17
IGT	2(11.1%)	4(12.5%)	0.87(0.14-5.32)	0.45
Hypertension	5(27.8%)	3(9.4%)	3.72(0.77-17.94)	0.06

Discussion:

NAFLD is an increasingly recognized common, silent and underdiagnosed syndrome. In children NAFLD is mainly associated with obesity and metabolic syndrome and is therefore considered as a metabolic complication of obesity. Paralleling the increasing prevalence of obesity in the pediatric population, NAFLD is expected to become one of the most common causes of End Stage Liver Disease in both children and young adults. This study was done to identify the prevalence of NAFLD among obese children attending Pediatric Endocrine OPD at BIRDEM and anthropometric and metabolic abnormalities among these children.

In the present study the prevalence of obesity was highest in the 8-12 years age group. 66% of obese children and adolescents were in this age group. Male preponderance has been reported in most of the studies in Bangladesh and in neighboring countries.^{14,15} In the present study, male: female ratio was found 1.5:1 which was similar to the previous study done in BIRDEM, showing male: female ratio 1.3:1^{14,15}

The exact prevalence of NAFLD is not well established. Information on its prevalence among children is scanty. Our findings can be compared with the similar findings in Northern Japan, where fatty liver was diagnosed by ultrasound was 10-35% among obese children and

adolescents.¹⁶ However the prevalence of NAFLD in obese children and adolescents is reported to be between 20-77% in various studies performed in different countries.^{5,17} In present study 72% had mild, 28% had moderate NAFLD, while no patient was identified to have severe NAFLD. This finding is comparable to study done to evaluate fatty liver in children-therapeutics and clinical risk management.¹⁸

There had been male predominance (M 72.2%, F 27.8%) found in our study with mean age of 11.67±2.25 years. In Japan, they conducted a study for prevention of fatty liver in Japanese children and relationship to obesity: an epidemiological ultrasonographic survey¹⁶ where there was no difference between fatty liver prevalences between boys and girls. However, male predominance and younger age at presentation were postulated in different previous studies.^{5,18}

In our study, anthropometric measurements were compared among obese children and adolescents with or without NAFLD which was not statistically significant. In china, a study was conducted for Hepatic steatosis in obese children⁵ showed, NAFLD was positively correlated with BMI, waist and hip circumference, triglyceride, and insulin resistance, indicating that higher adiposity may lead to a greater degree of fatty acid accumulation in the hepatocytes. Several studies have shown that the presence of truncal obesity strongly predicts increased risk for insulin resistance and metabolic syndrome in obese children. Waist circumference is an easy and practicable tool to identify central obesity in children thus to assist in decision making regarding metabolic risk. In a study¹⁹ found that waist circumference more than or equal to 75th percentile could be the optimum threshold to predict metabolic syndrome in children.

Hyperlipidemia appeared to be an important biochemical parameter of paediatric NAFLD. Prevalence of hyperlipidemia varies between 20 to 92% among NAFLD.⁷ In our study, high cholesterol, high LDL and high SGPT, were more prevalent in obese children with NAFLD in comparison to children without NAFLD but there was no significant difference in TG, HDL and blood sugar level between these two groups. In a study among Iranian obese children and adolescents, total cholesterol and SGPT were significantly higher among NAFLD group which is consistent with our findings.¹⁸ The best enzyme marker for NAFLD appears to be SGPT with a high

sensitivity and specificity. Another study²⁰ used SGPT as the predictor of fatty liver and found the correlation with of obesity to be the third best as compared to immunoreactive insulin and serum triglyceride.

Conclusion:

In the present study, it was observed that hyperlipidemia i.e. high cholesterol and high LDL were more prevalent in obese children with NAFLD compared to obese children without NAFLD. The best enzyme marker for NAFLD, SGPT was also significantly high in obese children with NAFLD. Our study concluded that hyperlipidemia with raised SGPT are important signs of liver dysfunction in obese children with NAFLD.

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