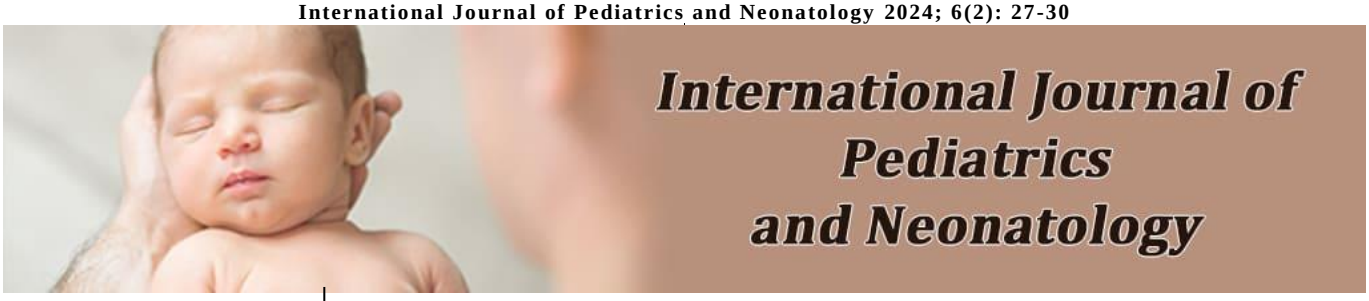

ISSN Print: 2664-8350
ISSN Online: 2664-8369
Impact Factor: RJIF 5.26
IJPN 2024; 6(2): 27-30
www.pediatricsjournal.net
Received: 16-05-2024
Accepted: 21-06-2024

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Lipid profile in obese children

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DOI: https://doi.org/10.33545/26648350.2024.v6.i2a.78

Abstract
Background: Obesity and overweight are the most prevalent nutritional disorders among children and adolescents in the United States and worldwide. The prevalence of childhood obesity has increased significantly, making it a major global health concern. The aim of the present study to show the association between obesity and hyperlipidemia in children and adolescents.
Method: A case-control study assessed obesity in 50 children and adolescents (aged below five years, five to ten years, and above ten years) at two hospitals in Babylon from March to December 2012. Obesity was defined as BMI > 95th percentile for age and gender, excluding genetic syndromes and chronic diseases. Lipid profiles were measured using enzymatic assays, with LDL calculated by the Friedewald formula.
Results: A case-control study on childhood obesity (n=100) in Babylon hospitals found predominant obesity among 5-10-year-olds (48% male, 48% female). Serum lipid profiles showed non-significant total cholesterol differences but significant increases in triglycerides in obese females aged <5 and 5-10 years ($p< 0.05$). HDL levels were lower in obese groups without significance. LDL levels were significantly higher in obese males across all age groups ($p< 0.05$).
Conclusion: Obese children had greater triglycerides in girls and LDL in boys, but cholesterol is similar.

Keywords: Lipid, profile, obese, children

Introduction
Obesity and overweight are the most prevalent nutritional disorders among children and adolescents in the United States and worldwide [1]. The prevalence of childhood obesity has increased significantly, making it a major global health concern [1]. The National Health and Nutrition Examination Survey (NHANES) indicates a rise in obesity across all pediatric age groups, sexes, and various ethnic and racial groups [2]. Childhood obesity is influenced by multiple factors, including genetics, environment, socioeconomic status, family size, parental obesity, sedentary behaviors, lifestyle, and eating habits. Over 90% of obesity cases are idiopathic, with less than 10% linked to hormonal or genetic causes [1]. There is no universally accepted definition of childhood obesity. The Centers for Disease Control and Prevention (CDC) defines obesity in children and adolescents aged 2 to 20 years as a Body Mass Index (BMI) at or above the 95th percentile of sex-specific BMI-for-age values from the 2000 CDC growth charts [3]. "At risk of overweight" is defined as a BMI between the 85th and 95th percentiles or exceeding 30 kg/m² at any age [1]. Various methods exist to measure body fatness, including underwater weighing, dual-energy X-ray absorptiometry (DXA), and computed tomography, but these are often limited to research due to their complexity and cost [4, 5]. Public health evaluations and clinical screenings commonly use anthropometric measurements such as skinfold thickness and BMI, which is calculated as weight in kilograms divided by height in meters squared [4-6]. The rapid increase in childhood obesity prevalence has alarmed public health agencies, healthcare clinicians, researchers, and the general public [4]. In the United States, obesity prevalence among children has increased from 5% in 1963 to 17% in 2003-2004, with an estimated 17% of children and adolescents aged 2 to 19 years being overweight according to the 2003-2004 NHANES [7]. Internationally, childhood obesity rates vary, with at least 10% of children being overweight in 77% of the countries analyzed [8]. Racial and ethnic disparities in obesity rates are evident, with higher prevalence among Puerto Rican, Cuban American, and Native

American preschoolers, and among black, Native American, Puerto Rican, Mexican, and native Hawaiian school-aged children [7, 8]. Obesity is also more common during specific life periods, such as ages 5-7 years and adolescence, and is predictive of adult obesity, with 80% of obese teenagers continuing to be obese as adults [8]. Obesity results from an imbalance between energy intake and expenditure. Adipose tissue development begins in the mid- to late third trimester of pregnancy, with critical periods of adipocyte differentiation occurring during infant feeding, puberty, and other life stages [9, 10]. Hormones such as leptin and adiponectin play crucial roles in regulating energy balance and metabolism. Leptin deficiency can lead to massive obesity, while adiponectin increases insulin sensitivity and acts as an anti-inflammatory mediator [11, 12]. The etiology of childhood overweight is multifactorial, involving genetic, biological, social, and environmental pathways. Factors such as maternal weight during pregnancy, infant feeding methods, and birth weight are associated with childhood overweight [13]. Dietary habits, including the affordability and accessibility of food, along with decreased physical activity, contribute to rising obesity rates. Sedentary behaviors, such as television viewing and playing computer games, are linked to increased obesity prevalence [14, 15]. Lipid profile abnormalities, including elevated cholesterol and triglycerides, are commonly associated with obesity. Lipoprotein metabolism involves various enzymes and lipoproteins, which play roles in energy storage, cell structure, and hormone production [16, 17]. Hyperlipidemia, a disorder characterized by high levels of lipids in the blood, is often seen in obese individuals and can increase the risk of cardiovascular disease and other health issues [18, 19]. The aim of the present study to show the association between obesity and hyperlipidemia in children and adolescents.

Method

A case-control study was conducted at the outpatient clinics of the Pediatric Endocrinology in Childs Center Teaching Hospital and the Pediatric and Obstetric Hospital of Babylon from March 1, 2012, to December 15, 2012. The study aimed to assess obesity in male and female children and adolescents. The sample included 100 patients, divided into three groups based on age: group 1 (Below five years), group 2 (Between five and ten years), and group 3 (above ten years). Each group contained both obese and control patients matched by sex and age. Obesity was defined as a BMI above the 95th percentile for age and gender. Exclusion criteria included the presence of genetic syndromes associated with obesity or chronic diseases such as asthma, diabetes mellitus, hypothyroidism, and nephrotic syndrome on steroid therapy. Of the initial 63 patients, 13 were excluded for various reasons: five were younger than two years, four had a chronic disease detected or reported during consultation, two had Prader-Willi syndrome, and two did not attend the scheduled exams or tests. Thus, the final sample consisted of 50 patients. The control group also included 50 children and adolescents who visited the outpatient clinic for minor illnesses and were matched for age and gender (With a variation of up to six months). The anthropometric assessment used BMI, as recommended by the World Health Organization, which defines obesity as values above the 95th percentile [1]. The lipid profile was assessed by measuring fasting total cholesterol, HDL-cholesterol, and triglycerides (TG) using the enzymatic colorimetric assay with LABTEST reagents, following Kwiterovich's reference values. LDL-cholesterol values were calculated using the Friedewald formula (LDL = Total Cholesterol - HDL - Triglyceride/5) [20]. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) version 19. Categorical data were presented as count and percentage. Associations were tested using cross-tabulation of variables and Pearson Chi-square tests. Numerical data were presented as mean and standard error of mean, and paired sample t-tests were used for comparisons between each two groups. The level of significance was set at $p \leq 0.05$.

Results

The predominant age group were between five and ten years in obese group (48% in male, 48%in female) as shown in table 1.

Table 1: Distribution of patients and control groups according to age and gender.

		Study groups					
		Control			Obese		
Age groups		Female	Male	Total	Female	Male	Total
<5 years	No.	7	5	12	6	6	12
	% within age group	58.33%	41.66%	24.00%	50.00%	50.00%	24.00%
	% within gender type	28.00%	20.00%	100.00%	24.00%	24.00%	100.00%
5-10 years	No.	11	13	24	12	12	24
	% within age group	44.00%	52.00%	48.00%	48.00%	48.00%	48.00%
	% within gender type	45.83%	54.17%	100.00%	50.00%	50.00%	100.00%
>10 years	No.	7	7	14	7	7	14
	% within age group	28.00%	28.00%	28.00%	28.00%	28.00%	28.00%
	% within gender type	50.00%	50.00%	100.00%	50.00%	50.00%	100.00%
Total	No.	25	25	50	25	25	50
	% within age group	100.00%	100.00%	100.00%	100.00%	100.00%	100.00%
	% within gender type	50.00%	50.00%	100.00%	50.00%	50.00%	100.00%
Mean ±S.D		7.29±2.85	7.82±2.37	7.56±2.60	7.34±2.30	8.46±2.24	7.90±2.32
Minimum		2	3.5	2	2.5	4	2.5
Maximum		12.5	13.5	13.5	12	13	13

Serum total cholesterol show an increase in obese children at all age groups in comparison to control but of no significant value, as shown in table (2).

Table 2: Total cholesterol level destireputon in studied groups.

Age groups		Cholesterol level		P value
		Control	Obese	
< 5 years	Female	4.30±0.75	4.80±0.44	>0.05 NS
	Male	3.10±0.94	4.90±0.22	>0.05 NS
5-10 years	Female	4.39±1.03	4.73±0.33	>0.05 NS
	Male	4.74±0.30	5.11±0.96	>0.05 NS
> 10 years	Female	4.27±0.21	5.05±1.63	>0.05 NS
	Male	3.81±0.70	4.70±1.16	>0.05 NS

ns=no significant * = significant

Serum triglyceride show a significant increase in groups (1&2) of obese female (p value < 0.05), as shown in table 3

Table 3: Triglyceride level distribution in studied groups.

Age group		Triglyceride level		P value
	Sex	Control	Obese	
< 5 years	Female	0.95±0.56	2.09±0.37	<0.05
	Male	1.01±0.67	1.80±0.20	>0.05 NS
5-10 years	male	0.93±0.51	1.73±0.22	>0.05 NS
	female	0.93±0.34	1.89±0.29	<0.05
> 10 years	Female	0.96±0.55	1.34±0.28	>0.05 NS
	Male	0.98±0.42	1.14±0.30	>0.05 NS

NS = No significant * = Significant

Serum HDL Show decrease in obese children in both gender but without significant value, as shown in table 4.

Table 4: HDL level in studied groups

Age groups		HDL level		P value
	Sex	Control	Obese	
< 5 years	Female	1.28±0.32	1.05±0.32	>0.05NS
	Male	1.60±0.25	1.27±0.35	>0.05 NS
5-10 years	Female	1.35±0.50	1.18±0.15	>0.05 NS
	Male	1.24±0.20	1.36±0.31	>0.05 NS
> 10 years	Female	1.67±0.91	1.42±0.40	>0.05 NS
	Male	1.15±0.15	1.44±0.08	>0.05 NS

NS= no significant

Serum LDL Show significant increase in obese male at all age groups (1, 2&3) (P value < 0.05), as shown in table 5.

Table 5: LDL level in studied groups

Age groups		LDL level		P value
	Sex	Control	Obese	
< 5 years	Female	2.73±0.78	3.24±0.21	>0.05 NS
	Male	1.30±0.52	3.05±0.33	<0.05*
5-10 years	Female	2.13±0.13	2.53±1.14	>0.05 NS
	Male	1.20±0.46	3.44±1.04	<0.05 *
> 10 years	Female	3.00±0.57	3.60±0.61	>0.05 NS
	Male	2.04±1.19	3.37±0.71	<0.05*

NS = No significant * = Significant

Discussion

The predominant age of obesity in this study was between five and ten years, which is similar to the findings of Lima *et al.* [21]. The male to female ratio in our study was 1:1, whereas Lima *et al.* reported a ratio of 1.4:1, indicating a slightly higher prevalence of obesity among males in their study. Both studies, however, have small sample sizes, which may limit the generalizability of the findings. Total cholesterol levels in the obese groups in our study were comparable to those observed in the American population, as reported by Webber *et al.* [22]. In their study, cholesterol levels were 4.2 mmol/l in males and 4.4 mmol/l in females. Our results showed higher cholesterol levels, with females having an average of 4.84 ± 0.78 mmol/l and males having 4.88±0.48 mmol/l, indicating a slightly higher cholesterol level in our sample. These findings are consistent with those of Lima *et al.* [21]. LDL-cholesterol levels in our study were higher than those reported by Lima *et al.* [21], which could be attributed to a higher intake of dietary fat and a lower intake of dietary fiber among the participants. This difference highlights the potential impact of dietary habits on lipid profiles in obese children and adolescents. HDL-cholesterol, which is known to be a protective factor against heart diseases, showed lower borderline values in the studied groups, but the differences were not statistically significant. This result is similar to the findings of Hamdi *et al.* [23], who also reported lower HDL levels in obese individuals. It is possible that HDL levels may take years of obesity history to develop significantly, and the degree of obesity could affect HDL levels later in life, as observed by Lis *et al.* [24]. Serum triglyceride levels showed a significant increase in the female obese groups (Groups 1 and 2), but no statistically significant increase was observed in group 3. This could be explained by the hormonal changes occurring during this stage of development, which may act as a protective factor against changes in the lipid profile. After menarche, changes in lipid profile are sensitive to the influence of sex hormones, particularly estrogen, which has a favorable effect on lipoproteins by increasing HDL-c and reducing LDL-c levels and triglycerides. This hormonal advantage is observed in females during adolescence and adulthood, as noted by Moura *et al.* [25]. In the male groups, there were no significant changes in serum triglyceride levels between the obese and control groups, which is consistent with the findings of Lima *et al.* [21]. This lack of significant difference may suggest that triglyceride levels in males are less influenced by obesity during childhood and adolescence compared to females. Overall, our study highlights the complex interplay between obesity, lipid profiles, and hormonal changes during childhood and adolescence. The findings underscore the importance of considering gender-specific factors and dietary habits when addressing obesity and its associated metabolic disorders. The higher levels of total and LDL cholesterol observed in our study compared to some previous studies suggest a need for targeted interventions to improve dietary habits and reduce the risk of cardiovascular diseases in obese children and adolescents. The protective role of HDL cholesterol and the influence of sex hormones on lipid profiles further emphasize the need for a comprehensive approach to managing obesity in pediatric populations.

Conclusion

Lipid profile in obese children are different from the normal children the triglyceride is significantly higher increased in female and the LDL has significantly higher increased in obese male but cholesterol has no significant difference from control.

Conflict of Interest: Not available

Financial Support: Not available

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How to Cite This Article
Khudair AM, Hashim BI, Abood MAA. Lipid profile in obese childrean. International Journal of Pediatrics and Neonatology 2024; 6(2): 27-30.