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Rouba Farajallah Manachi
Prime Hospital Al Garhood Airport Road, Dubai, Saudi Arabia

Corresponding Author:
Rouba Farajallah Manachi
Prime Hospital Al Garhood Airport Road, Dubai, Saudi Arabia

Adipose tissue content, distribution and lipid profile as a risk factor in children with bronchial asthma

Rouba Farajallah Manachi

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Abstract
Background: Obesity induces some physiological changes which are conducive to either development of asthma or cause of poorly controlled asthma state. The aim of this work was to study the relationships between obesity and bronchial asthma in children and whether adipose tissue content and body fat percentage may affect the degree of asthma severity and level of asthma control, also to assess the role of lipid profile in asthmatic children.
Methods: This study carried out on 90 children with varying degree of severity and control with age range 2-16 years. All the studied cases were subjected to detailed History (frequency of symptoms, number of exacerbations, medications use, degree of asthma severity and level of asthma control), anthropometric data (Height, Weight, BMI, Skin fold thickness, Body fat percentage) and Investigations.
Results: Asthma control had highly significant relations with body height, BMI and body fat percent with P value < 0.001. Body fat percentage of the studied group is significantly related to height, weight, BMI, abdominal skin fold thickness, sub scapular skin fold thickness, triceps skin fold thickness, the sum of three skin folds and with upper arm circumference. body fat percent is significantly affected by serum cholesterol level and serum LDL level with P value <0.001.
Conclusions: Obesity increases the risk of subsequent asthma, although the evidence does not support the hypothesis that asthma leads to increased obesity. Adiposity is associated with poorer asthma. High serum levels of (cholesterol, TG, HDL, LDL) worse asthma control.

Keywords: Adipose tissue content, lipid profile, bronchial asthma

Introduction
Bronchial asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation [1].
The prevalence of childhood asthma is increasing world-wide and varies from 1% to more than 30% in different populations [2]. In Egypt, the prevalence among primary school children was reported to be 9.4% in Cairo city [3].
The obesity prevalence has alarmingly increased by more than 3 fold in the last three decades [4]. Obesity induces some physiological changes which are conducive to either development of asthma or cause of poorly controlled asthma state [5]. Since 2015, the Centers for Disease Control and Prevention (CDC) has listed obesity as a major risk factor for asthma in children [6]. The mechanisms that lead to these disorders may start at a pediatric age and include changes in lung mechanics, co morbidities, dietary intake and low physical activity, alterations in insulin and/or glucose metabolism, and systemic inflammation. Moreover, among children with asthma, overweight is associated with a decreased response to drugs and poorer quality of life [7].
Excess of adipose tissue and body fat percentage seem to play an important role in the complicated dependencies between obesity and the risk of developing asthma [8].
An alternate day calorie restriction diet has been reported to improve asthma symptoms by decreasing levels of serum cholesterol and triglycerides, reducing markers of oxidative stress and increasing levels of the antioxidant uric acid. Therefore, to investigate the lipid profile in asthmatic children may be important in asthma control treatment [9].

Obesity appears to be associated with distinct asthma characteristics, and therefore optimal management of the obese asthmatic child requires a thoughtful, tailored approach and to receive attention in many studies [10].

The aim of this work was to study the relationships between obesity and bronchial asthma in children and whether adipose tissue content and body fat percentage may affect the degree of asthma severity and level of asthma control, also to assess the role of lipid profile in asthmatic children.

Patients and methods

This study carried out on 90 children with varying degree of severity and control with age range 2-16 years. This study was carried out at the Pediatric Allergy and Pulmonology Clinic of the Specialized Children Hospital Cairo University Hospital (Abo El-Rich) during the period from October 2017 to July 2018.

Ethics committee approval was obtained before conducting the study. Informed written consent was obtained from each patient.

Inclusion criteria were both males and females, age from 2-16 years, children with persistently (mild, moderate and severe) asthma and controlled, partially controlled and uncontrolled asthmatic children.

Exclusion criteria were any patient with chronic diseases (Causes of wheezy chest: bronchiolitis oblitrance, cystic fibrosis, autoimmune disorders, immunocompromized patients.

Gastro-esophageal reflux disease) and patients on any long-term medications that affect adipose content and distribution as: long term systemic steroids.

Methods

All the studied cases were subjected to: Detailed History (frequency of symptoms, number of exacerbations, medications use, degree of asthma severity and level of asthma control), anthropometric data (Height, Weight, BMI, Skin fold thickness, Body fat percentage) and Investigations.

Skin fold thickness

Measured using skin fold caliper

Triceps skin fold thickness: A vertical fold was measured on the posterior surface of the arm above the triceps brachii muscle, half the distance between the (acromion) and the elbow joint (olecranon). The arm is relaxed, hanging by the side. The measurement was repeated twice.

Abdominal skin fold thickness: A horizontal fold was lifted one third the distance between the umbilicus and the anterior superior iliac spine, i.e. closer to the umbilicus.

Sub scapular skin fold thickness: A slightly oblique fold along the rib line; was measured directly below the bottom angle of the shoulder blade.

Upper arm circumference: Measured at the mid-point between the tip of the shoulder (olecranon process) and the tip of the elbow (the acromium) using non retractable plastic tape.

Body fat percentage

Investigations

Serum lipid profile: 3ml venous blood were withdrawn from all subjects and were collected on plain tubes, left for 10 minutes to clot and then centrifugated at 3000 rpm for 15 minutes, the separated serum was used for lipid profile assay (Cholesterol, triglycerides, HDL, LDL).

Total cholesterol: Tests were done on cobas 6000 automated analyzer by enzyme colorimetric method

Cholesterol esters+H_2O --- ^{esterase} --> cholesterol+RCOOH

Cholesrerol+O_2----- ^{oxidase} -------->cholesterol-3-one+H_2O_2

$2H_2O_2$+ 4-aminophenazone+phenol---- ^{peroxidase} ---> 4phenazone+$4H_2O$.

The produced color was measured on wavelength 500nm.

Triglycerides: Triglycerides are measured after enzymatic splitting with lipoprotein lipase. Indicator is quinoneimine which is generated from 4-aminoantipyrine and 4-chlorophenol by hydrogen peroxide under the catalytic action of peroxidase.

Triglycerides + 3H2 O -------LPL-----> glycerol + fatty acids

Glycerol + ATP ----------GK---------> glycerol-3-phosphate + ADP

Glycerol-3-phosphate + O_2---GPO---> dihydroxyacetone phosphate + H_2O_2

$2H_2O_2$ + 4-aminophenazone + 4-chlorophenol -----POD-----> Quinoneimine + $4H_2O$ + HCl.

High density lipoprotein (HDL) cholesterol: HDL is measured directly in serum. The basic principle of the method is as follows:

The apoB containing lipoproteins in the specimen are reacted with a blocking reagent that renders them non-reactive with the enzymatic cholesterol reagent under conditions of the assay. The apoB containing lipoproteins are thus effectively excluded from the assay and only HDL-cholesterol is detected under the assay conditions. The reagents are purchased from Roche/Boehringer-Mannheim Diagnostics. The method uses sulfated alpha-cyclodextrin in the presence of Mg^{+2}, which forms complexes with apoB containing lipoproteins, and polyethylene glycol-coupled cholesteryl esterase and cholesterol oxidase for the HDL-cholesterol measurement. The reactions are as follows:

ApoB containing lipoproteins + α-cyclodextrin + Mg^{+2} dextran SO_4 ---> soluble non-reactive complexes with apoB-containing lipoproteins.

HDL-cholesterol esters ----------> HDL-unesterified cholesterol + fatty acid.

Unesterified cholesterol + O_2 ---PEG----- > cholestenone + H_2O_2.

H_2O_2 + 5-aminophenazone + N-ethyl-N-(3-methylphenyl)-N'_succinyl ethylene diamine + H2O + H^+ ---peroxidase---> qunoneimine dye + H_2O [11].

Low density lipoprotein (LDL) cholesterol: Most of the circulating cholesterol is found in three major lipoprotein fractions: very low-density lipoproteins (VLDL), LDL and HDL:

[Total cholesterol] = [VLDL-cholesterol] + [LDL-cholesterol] + [HDL-cholesterol].

LDL-cholesterol is calculated from measured values of total cholesterol, triglycerides and HDL cholesterol according to the relationship:

[LDL-cholesterol] = [total cholesterol] - [HDL-cholesterol] - [TG]/5.

Where [TG]/5 is an estimate of VLDL-cholesterol and all values are expressed in mg/dL [11].

The cutpoints for high and borderline high represent approximately the 95th and 75th percentiles, respectively. Low cutpoints for HDL-C represent approximately the 10th percentile [12].

Statistical analysis

Data were coded and entered using the statistical package SPSS (Statistical Package for the Social Sciences) version 25. Data was summarized using mean, standard deviation, median, minimum and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. Comparisons between quantitative variables were done using the non-parametric Kruskal-Wallis and Mann-Whitney tests. For comparing categorical data, Chi square ($\chi 2$) test was performed. Exact test was used instead when the expected frequency is less than 5. Correlations between quantitative variables were done using Spearman correlation coefficient. P-values less than 0.05 were considered as statistically significant.

Results

Table 1 shows Demographic, Clinical characteristics, Skin fold thicknesses and Other anthropometric measures of the studied asthmatic children at Specialized Children Hospital of Cairo University.

Table 1: Demographic, clinical characteristics, skin fold thicknesses and other anthropometric measures of the studied asthmatic children at Specialized Children Hospital of Cairo University

Variable		Count (%)
Sex:	Male	49 (54.4%)
	Female	41 (45.6%)
Residence:	Cairo	33 (36.7%)
	Giza	56 (62.2%)
	Others	1 (1.1%)
Age:		6.54 ± 2.7
Clinical characteristics		
Family history of asthma		90 (100%)
Atopic manifestations		68 (75.6%)
Detailed atopic manifestations		
Allergic rhinitis		17 (18.9%)
Conjunctivitis		27 (30%)
Dermatitis		24 (26.7%)
Seasonal variations for asthma		89 (98.9%)
Asthma control	Controlled	69 (76.7%)
	Partially controlled	12 (13.3%)
	Uncontrolled	9 (10%)
Asthma severity	Mild intermittent	57 (63.3%)
	Mild persistent	23 (25.6%)
	Moderate persistent	10 (11.1%)
	Sever persistent	0 (0%)
Inhaled steroids		70 (77.8%)
Skin fold thicknesses		
Abdominal skin fold thickness (cm)		11.33 ± 4.68
Sub scapular skin fold thickness (cm)		10.53 ± 4.51
Triceps skin fold thickness (cm)		12.25 ± 3.89
The sum of 3 skin fold thicknesses		34.14 ± 11.74
Upper arm circumference (cm)		18.18 ± 4.57
Height (cm)	Normal	66 (73.3%)
	Short	23 (25.6%)
	Tall	1 (1.1%)
Weight (kg)	Normal	70 (77.8%)
	Under weight	14 (15.6%)
	Over weight	6 (6.7%)
BMI (kg/m²)	Normal	49 (54.4%)
	Under weight	11 (12.2%)
	Over weight	10 (11.1%)
	Obese	20 (22.2%)
Fat%	Healthy	32 (46.7%)
	Under fat	36 (40%)
	Over fat	7 (7.8%)
	Obese	5 (5.6%)
Fat %	No high fat%	78 (86.7%)
	High fat %	12 (13.3%)
Cholesterol (mg/dl)	Acceptable	74 (82.2%)
	High	16 (17.8%)

TG (mg/dl)	Acceptable	79 (87.8%)
	High	11 (12.2%)
HDL (mg/dl)	Acceptable	64 (71.1%)
	Abnormal (low)	26 (28.9%)
LDL (mg/dl)	Acceptable	80 (88.9%)
	High	10 (11.1%)

Data is expressed as the mean ±SD and frequency (%)

There is no significant statistical difference between sex and body weight and between sex and body height in the studied group. There is significant statistical difference between sex and body fat percentage (P value < 0.001). Table 2

Table 2: Sex correlation with BMI, Relation between sex and body weight, body height, body fat percent, serum levels of cholesterol, TG, HDL, LDL

BMI (kg/m²)		Male	Female	P value
Normal		30 (61.2%)	19 (46.4%)	0.561
Under weight		5 (10.2%)	6 (14.6%)	
Over weight		5 (10.2%)	5 (12.2%)	
Obese		9 (18.4%)	11 (26.8%)	
Weight (kg)	Normal	39 (79.6%)	31 (75.6%)	0.928
	Under weight	7 (14.3%)	7 (17.1%)	
	Over weight	3 (6.1%)	3 (7.3%)	
Height (cm)	Normal	37 (75.5%)	29 (70.7%)	0.711
	Short	11 (22.4%)	12 (29.3%)	
	Tall	1 (2%)	0 (0%)	
Fat %	Healthy	13 (26.5%)	29 (70.7%)	< 0.001*
	Under fat	28 (57.2%)	8 (19.5%)	
	Over fat	5 (10.2%)	2 (4.9%)	
	Obese	3 (6.1%)	2 (4.9%)	
Cholesterol (mg/dl)	Acceptable	44 (89.8%)	30 (73.2%)	0.04*
	High	5 (10.2%)	11 (26.8%)	
TG (mg/dl)	Acceptable	46 (93.9%)	33 (80.5%)	0.05
	High	3 (6.1%)	8 (19.5%)	
HDL(mg/dl)	Acceptable	36 (73.5%)	28 (68.3%)	0.589
	Abnormal	13 (26.5%)	13 (31.7%)	
LDL (mg/dl)	Acceptable	45 (91.8%)	35 (85.4%)	0.503
	High	4 (8.2%)	6 (14.6%)	

Data in this table is expressed by number and %. TG: Triglycerides, HDL: High density lipoprotein, LDL: low density lipoprotein. *: P value < 0.05 is considered significant.

There is significant statistical difference between atopic manifestations and BMI, fat% with P value 0.002 & 0.047 respectively, while no relation between atopic manifestations and weight, height. there is no significant statistical difference between atopic manifestations and lipid profile in the studied group. The degree of asthma severity was significantly associated with high body weight, BMI, fat percentage and fat% interpretation in the studied group with P value 0.001, 0.033, 0.02, 0.001 respectively with no significant difference with body height. asthma severity is significantly related to serum cholesterol level with P value 0.006; hypercholesterolemia increases the level of asthma severity (Table 3).

Table 3: Relation between atopic manifestations and obesity parameters, lipid profile and between asthma severity and anthropometric measures, with lipid profile

Variable		Atopic manifestations		P value
		Present	Not present	
Height (cm)	Normal	49 (72.1%)	17 (77.3%)	0.838
	Short	18 (26.5%)	5 (22.7%)	
	Tall	1 (1.5%)	0 (0%)	
Weight (kg)	Normal	49 (72.1%)	21 (95.5%)	0.081
	Underweight	13 (19.1%)	1 (4.5%)	
	Overweight	6 (8.8%)	0 (0%)	
BMI (kg/m²)	Normal	31 (45.6%)	18 (81.8%)	0.002*
	Underweight	10 (14.7%)	1 (4.5%)	
	Overweight	7 (10.3%)	3 (13.6%)	
	Obese	20 (29.4%)	0 (0%)	

Fat%	Healthy	34 (50%)	8 (36.4%)	0.047*
	Under fat	22 (32.4%)	14 (63.6%)	
	Over fat	7 (10.3%)	0 (0%)	
	Obese	5 (7.4%)	0 (0%)	
Cholesterol (mg/dl)	Acceptable	54 (79.4%)	20 (90.9%)	0.339
	High	14 (20.6%)	2 (9.1%)	
TG (mg/dl)	Acceptable	58 (85.3%)	21 (95.5%)	0.282
	High	10 (14.7%)	1 (4.5%)	
HDL (mg/dl)	Acceptable	48 (70.6%)	16 (72.7%)	0.847
	Abnormal	20 (29.4%)	6 (27.3%)	
LDL(mg/dl)	Acceptable	60 (88.2%)	20 (90.9%)	1
	High	8 (11.8%)	2 (9.1%)	
Asthma severity				
		Mild intermittent	**Mild persistent**	**Moderate persistent**	
Height (cm)	Normal	41 (71.9%)	17 (73.9%)	8 (80%)	1
	Short	15 (26.3%)	6 (26.1%)	2 (20%)	
	Tall	1 (1.8%)	0 (0%)	0 (0%)	
Weight (kg)	Normal	43 (75.4%)	21 (91.3%)	6 (60%)	0.001*
	Underweight	13 (22.8%)	1 (4.3%)	0 (0%)	
	Overweight	1 (1.8%)	1 (4.3%)	4 (40%)	
BMI (kg/m²)	Normal	34 (59.6%)	12 (52.2%)	3 (30.0%)	0.033*
	Underweight	10 (17.5%)	1 (4.3%)	0 (0%)	
	Overweight	5 (8.8%)	4 (17.4%)		
	Obese	8 (14.0%)	6 (26.1%)	6 (60.0%)	
Fat%	Healthy	25 (43.9%)	14 (60.9%)	3 (30.0%)	0.002*
	Under fat	29 (50.9%)	5 (21.7%)	2 (20.0%)	
	Over fat	1 (1.8%)	3 (13.0%)	3 (30.0%)	
	Obese	2 (3.5%)	1 (4.3%)	2 (20.0%)	
Fat% interpretation	No high fat%	54 (94.7%)	19 (82.6%)	5 (50.0%)	0.001*
	High fat %	3 (5.3%)	4 (17.4%)	5 (50.0%)	
Cholesterol (mg/dl)	Acceptable	52 (91.2%)	16 (69.6%)	6 (60%)	0.006*
	High	5(8.8%)	7 (30.4%)	4 (40%)	
TG (mg/dl)	Acceptable	53 (93.0%)	19 (82.6%)	7 (70%)	0.068
	High	4 (7.0%)	4 (17.4%)	3 (30%)	
HDL (mg/dl)	Acceptable	44 (77.2%)	15 (65.2%)	5 (50%)	0.155
	High	13 (22.8%)	8 (34.8%)	5 (50%)	
LDL (mg/dl)	Acceptable	54 (94.7%)	18 (78.3%)	8 (80%)	0.055
	High	3 (5.3%)	5 (21.7%)	2 (20%)	

Data in this table is expressed by number and %. TG: Triglycerides, HDL: High density lipoprotein, LDL: low density lipoprotein. *: P value < 0.05 is considered significant

Asthma control had highly significant relations with body height, BMI and body fat percent with P value < 0.001 while body weight was not significantly related. Age is significantly related to fat percentage in the studied group with P value < 0.001. Asthmatic females had more fat percentage than males (Table 4).

Table 4: Relation between level of asthma control and anthropometric measures, lipid profile and between fat percent and age, sex, clinical characteristics of the studied group

Variable		**Asthma control**			**P value**
		Controlled	**Partially controlled**	**Uncontrolled**	
Height (cm)	Normal	57 (82.6%)	7 (58.3%)	2 (22.2%)	< 0.001*
	Short	12 (17.4%)	4 (33.3%)	7 (77.8%)	
	Tall	0 (0%)	1 (8.3%)	0 (0%)	
Weight (kg)	Normal	54 (78.3%)	9 (75.0%)	7 (77.8%)	0.051
	Underweight	13 (18.8%)	1 (8.3%)	0 (0%)	
	Overweight	2 (2.9%)	2 (16.7%)	2 (22.2%)	
BMI (kg/m²)	Normal	47 (68.1%)	2 (16.7%)	0 (0%)	< 0.001*
	Underweight	9 (13.0%)	1 (8.3%)	1 (11.1%)	
	Overweight	6 (8.7%)	4 (33.3%)	0 (0%)	
	Obese	7 (10.1%)	5 (41.7%)	8 (88.9%)	
Fat%	Healthy	32 (46.4%)	6 (50.0%)	4 (44.4%)	< 0.001*

		Under fat	34 (49.3%)	2 (16.7%)	0 (0%)	
		Over fat	1 (1.4%)	3 (25.0%)	3 (33.3%)	
		Obese	2 (2.9%)	1 (8.3%)	2 (22.2%)	
Fat% interpretation		No high fat	66 (95.7%)	8 (66.7%)	4 (44.4%)	< 0.001*
		High fat	3 (4.3%)	4(33.3%)	5 (55.6%)	
Cholesterol(mg/dl)		Acceptable	67 (97.1%)	7 (58.3%)	0 (0%)	< 0.001*
		High	2 (2.9%)	5 (41.7%)	9 (100.0%)	
TG (mg/dl)		Acceptable	68 (98.6%)	8 (66.7%)	3 (33.3%)	< 0.001*
		High	1 (1.4%)	4 (33.3%)	6 (66.7%)	
HDL (mg/dl)		Acceptable	54 (78.3%)	8 (66.7%)	2 (22.2%)	0.003*
		Abnormal	15 (21.7%)	4 (33.3%)	7 (77.8%)	
LDL (mg/dl)		Acceptable	68 (98.6%)	10 (83.3%)	2 (22.2%)	< 0.001*
		High	1 (1.4%)	2 (16.7%)	7 (77.8%)	
		Fat percent				
Age in years		Correlation Coefficient			0.407	
		P value			< 0.001*	
Sex	Male	12.57 ± 6.05			< 0.001*	
	Female	18.49 ± 5.82				
Atopic manifestations		16.36 ± 7.06			0.010*	
Seasonal variation of asthma		15.22 ± 6.64			0.467	
Asthma control	Controlled	13.68 ± 5.75			< 0.001*	
	Partially controlled	18.39 ± 7.16				
	Uncontrolled	23.22 ± 5.24				
Asthma severity	Mild intermittent	13.66 ± 5.95			0.003*	
	Mild persistent	16.30 ± 5.96				
	Moderate persistent	22.00 ± 7.51				
	Sever persistent	0 (0)				
Inhaled steroids	Given	15.79 ± 7.05			0.276	
	Not given	13.43 ± 4.49				

Data in this table is expressed by mean & SD. TG: Triglycerides, HDL: High density lipoprotein, LDL: low density lipoprotein. *: P value < 0.05 is considered significant

Body fat percentage of the studied group is significantly related to height, weight, BMI, abdominal skin fold thickness, sub scapular skin fold thickness, triceps skin fold thickness, the sum of three skin folds and with upper arm circumference. body fat percent is significantly affected by serum cholesterol level and serum LDL level with P value <0.001 while it is not significantly related to TG and HDL (Table 5).

Table 5: Correlation between fat% and anthropometric measures, lipid profile of the studied group, Comparison between high fat percent group and no high fat percent group regarding clinical data and Relation between degree of fat percent (high fat or no high fat) and lipid profile of the studied group

Variable		**Fat percent**		**P value**
		Correlation coefficient		
Height (cm)		0.297		0.004*
Weight (kg)		0.471		< 0.001*
BMI (kg/m²)		0.496		< 0.001*
Abdominal skin fold thickness (cm)		0.662		< 0.001*
Sub scapular skin fold thickness (cm)		0.804		< 0.001*
Triceps skin fold thickness (cm)		0.698		< 0.001*
The sum of 3 skin fold thickness		0.86		< 0.001*
Upper arm circumference (cm)		0.673		< 0.001*
Cholesterol (mg/dl)		0.398		< 0.001*
TG (mg/dl)		0.15		0.157
HDL (mg/dl)		0.091		0.395
LDL (mg/dl)		0.384		< 0.001*
		High fat %	No high fat%	
Family history of asthma		12 (100.0%)	78 (100.0%)	------
Other atopic manifestations		12 (100.0%)	56 (71.8%)	0.034*
Seasonal variation of asthma		12 (100.0%)	77 (98.7%)	1
Asthma control	Controlled	3 (25.0%)	66 (84.6%)	< 0.001*
	Partially controlled	4 (33.3%)	8 (10.3%)	

	Uncontrolled	5 (41.7%)	4 (5.1%)	
Asthma severity	Mild intermittent	3 (25.0%)	54 (69.2%)	0.001*
	Mild persistent	4 (33.3%)	19 (24.4%)	
	Moderate persistent	5 (41.7%)	5 (6.4%)	
	Sever persistent	0 (0%)	0 (0%)	
Inhaled steroids	Given	11 (91.7%)	59 (75.6%)	0.287
	Not given	1 (8.3%)	19 (24.4%)	
Cholesterol (mg/dl)		245.92 ± (116.12)	152.41 ± (71.78)	< 0.001*
TG (mg/dl)		111.67 ± (62.8)	81.69±(41.11)	0.168
HDL (mg/dl)		39.75 ± (18.65)	44.58 ± (16.97)	0.155
LDL (mg/dl)		117.13± (33.25)	89.59 ± (65.08)	0.003*

Data in this table is expressed by mean & SD or number & %. P value. *: P value < 0.05 is considered significant.

Discussion

Asthma is a chronic inflammatory disorder of the airways. Chronically inflamed air way are hyper responsive; they become obstructed and airflow is limited (by bronchoconstriction, mucus plug, and increased inflammation) when air ways are exposed to various risk factors. Asthma cause recurrent episodes of wheezing, breathlessness, chest tightness, and coughing particularly at night and early morning [13].

As regards clinical characteristics of the studied group, this study showed that all children included had positive family history of asthma. Also, a study from Colombia reported the same finding [14]. In this study, atopic manifestations were presented in 75.6% of children, while, in a study from Denmark the atopic manifestations were presented in 38% of 296 children [15].

Regarding the level asthma control for the group of children included in this study, it was controlled in 76.7%, partially controlled in 13.3%, and uncontrolled in10%.Also,in a study from Netherlands the level of asthma control was controlled in 69.3%, partially controlled in 17.7% and uncontrolled in 13% [16].

As regard to BMI, the children in this study were normal in 54.4%, underweight in 12.2%, overweight in 11.1%, and obese in 22.2% of cases. According to percentage body fat, they were healthy in 46.7%, under fat in 40%, over fat in 7.8%, and obese in 5.6%. While, in a study from UK the BMI was normal in72.4%, underweight in 4.5%, over weight in17.6%, and obese in 5.5% and percentage body fat was over weight in 26.9%, and obese in 2.9% [17].

Regarding skin fold thickness, the results of this study showed that the mean ± SD of triceps skin fold thickness was 12.25 ± 3.89, abdominal skin fold thickness was 11.33 ± 4.68, and sub scapular skin fold thickness was 10.53 ± 4.51. While, another Egyptian study showed that the triceps skin fold thickness was 10.5±19.1, abdominal skin fold thickness was 13.5 ±27.6 and sub scapular skin fold thickness was 10.6 ±23.9 [18].

This study shows that females have a higher serum cholesterol level than males.

Similarly, a study from Denmark on 411 children at age between 5:7 years and was concerned with studying lipid profile in relation to asthma in children and reported that hypercholesterolemia more in girls than boys [15].

The results of this study showed that, the degree of asthma severity was significantly associated with increasing body fat percentage with P value 0.002. Similarly, a study was conducted in UK showed that the increase in percentage body fat is associated with increase in asthma severity [17].

In the current study, asthma severity is directly proportion to total serum cholesterol level with P value 0.006. These results came in agreement with those of a study carried out in New York which found that hypercholesterolemia is associated with higher asthma frequency particularly in overweight and obese children [19].

This study showed that there is a significant statistical difference between level of asthma control and BMI with P value < 0.001. Approximately similar results were reported in more than one study, a prospective American study followed 368 adolescent using BMI & percentage body fat as markers for adiposity which found that higher BMI & high body fat percent were associated with more symptom days and exacerbations. So, they concluded that adiposity is associated with poorer asthma control [14].

This study showing that high serum levels of (cholesterol, TG, HDL, LDL) worse asthma control. Also, a study was conducted in China on 3,458 patients to explore the role of serum total cholesterol, TG, HDL and LDL in asthmatics, they found that LDL and total cholesterol worse asthma control [20].

In this study skin fold thicknesses (triceps, abdominal, sub scapular) are directly proportion to degree of asthma severity with P value 0.001. Similarly, a study from Poland showed that BMI and skin fold thicknesses (triceps, abdominal, sub scapular) were significantly high in sever asthmatics in comparison to their healthy pears [21].

Also, these results show that the use of inhaled steroids is directly proportion to the degree of asthma severity but not with asthma control with P value of 0.014 and 0.105 respectively. Similarly, a European study on 274 obese patients to assess the use of ICS in obese asthmatics they showed increased need for higher ICS doses with the increase in asthma severity [22].

These results show that inhaled steroid use in asthmatic children may affect the serum level of cholesterol and LDL with P value of 0.007 and 0.002 respectively, while serum TG and HDL not affected by their use. Approximately, the same result was reported by Vinding *et al*., that showed high levels of LDL cholesterol were associated with the use of ICS in concurrent asthma [15].

Conclusions

Obesity increases the risk of subsequent asthma, although the evidence does not support the hypothesis that asthma leads to increased obesity. Adiposity is associated with poorer asthma. High serum levels of (cholesterol, TG, HDL, LDL) worse asthma control.

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References

1. Lung NH, Institute B. Global Initiative for Asthma (GINA). Global strategy for asthma management and prevention. Revised 2006. Available from: http://www.ginasthma.org
2. Tunde-Ayinmode MF. Children with bronchial asthma assessed for psychosocial problems in a teaching hospital in Nigeria. Afr Health Sci. 2015;15(2):690-700. DOI:10.4314/ahs.v15i2.49
3. Georgy V, Fahim HI, El-Gaafary M, Walters S. Prevalence and socioeconomic associations of asthma and allergic rhinitis in northern [corrected] Africa. Eur Respir J. 2006;28(4):756-762. DOI:10.1183/09031936.06.00089005
4. Basu M, Sarkar K, Shahbabu B, Ray S, Barik G, Chatterjee S, *et al*. Pattern and determinants of overweight and obesity among medical students of Kolkata. Int J Pharm Sci Res. 2016;7(1):377-386.
5. Brashier B, Salvi S. Obesity and asthma: physiological perspective. J Allergy (Cairo). 2013;2013:198068. DOI:10.1155/2013/198068
6. Peters U, Dixon AE, Forno E. Obesity and asthma. J Allergy Clin Immunol. 2018;141(4):1169-1179. DOI:10.1016/j.jaci.2018.02.004
7. Forno E, Han YY, Libman IM, Muzumdar RH, Celedón JC. Adiposity and asthma in a nationwide study of children and adults in the United States. Ann Am Thorac Soc. 2018;15(3):322-330. DOI:10.1513/AnnalsATS.201709-723OC
8. Umławska W. Adipose tissue content and distribution in children and adolescents with bronchial asthma. Respir Med. 2014;109. DOI:10.1016/j.rmed.2014.11.006
9. Fang LJ, Huang CS, Liu YC, Su YM, Wan KS. The lipid profile in obese asthmatic children compared to non-obese asthmatic children. Allergol Immunopathol (Madr). 2016;44(4):346-350. DOI:10.1016/j.aller.2015.12.004
10. Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, *et al*. Youth risk behavior surveillance United States, 2007. MMWR Surveill Summ. 2008;57(4):1-131.
11. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499-502.
12. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, *et al*. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2019;73(24):3168-3209. DOI:10.1016/j.jacc.2018.11.002
13. Lung NH, Institute B. Global Initiative for Asthma (GINA). Global strategy for asthma management and prevention: NHLBI/WHO workshop report; 2006. Available from: http://www.ginasthma.com
14. Kattan M, Kumar R, Bloomberg GR, Mitchell HE, Calatroni A, Gergen PJ, *et al*. Asthma control, adiposity, and adipokines among inner-city adolescents. J Allergy Clin Immunol. 2010;125(3):584-592. DOI:10.1016/j.jaci.2010.01.053
15. Vinding RK, Stokholm J, Chawes BLK, Bisgaard H. Blood lipid levels associate with childhood asthma, airway obstruction, bronchial hyperresponsiveness, and aeroallergen sensitization. J Allergy Clin Immunol. 2016;137(1):68-74.e4. DOI:10.1016/j.jaci.2015.05.033
16. van der Meer V, van Stel HF, Bakker MJ, Roldaan AC, Assendelft WJ, Sterk PJ, *et al*. Weekly self-monitoring and treatment adjustment benefit patients with partly controlled and uncontrolled asthma: an analysis of the SMASHING study. Respir Res. 2010;11(1):74. DOI:10.1186/1465-9921-11-74
17. Wang R, Custovic A, Simpson A, Belgrave DC, Lowe LA, Murray CS. Differing associations of BMI and body fat with asthma and lung function in children. Pediatr Pulmonol. 2014;49(11):1049-1057. DOI:10.1002/ppul.22927
18. Ismail NF, Aly SM, Abdu MO, Kafash DN, Kelnar CJ. Study of growth in prepubertal asthmatics. Indian J Pediatr. 2006;73(12):1089-1093. DOI:10.1007/bf02763051
19. Vijayakanthi N, Greally JM, Rastogi D. Pediatric obesity-related asthma: the role of metabolic dysregulation. Pediatrics. 2016;137(5). DOI:10.1542/peds.2015-0812
20. Su X, Ren Y, Li M, Zhao X, Kong L, Kang J. Association between lipid profile and the prevalence of asthma: a meta-analysis and systematic review. Curr Med Res Opin. 2018;34(3):423-433. DOI:10.1080/03007995.2017.1384371
21. Umławska W. Adipose tissue content and distribution in children and adolescents with bronchial asthma. Respir Med. 2015;109(2):200-207. DOI:10.1016/j.rmed.2014.11.006
22. Savas M, Wester VL, Staufenbiel SM, Koper JW, van den Akker ELT, Visser JA, *et al*. Systematic evaluation of corticosteroid use in obese and non-obese individuals: a multi-cohort study. Int J Med Sci. 2017;14(7):615-621. DOI:10.7150/ijms.19213

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