

Lipid Profile in Patients with Asthma and Allergic Rhinitis

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ABSTRACT:

Background: Asthma is a chronic inflammatory disease of the respiratory airways; its prevalence has increased worldwide. The disease may be associated with metabolic changes that could be either induced by treatment or may be due to inflammatory process.

Aim: To clarify the status of lipid profile in Iraqi patients with asthma and allergic rhinitis.

Patients and Methods: The study was performed on asthmatic patients (190), allergic rhinitis patients (110) and healthy control subjects (48). Age of subjects included were from 16-60 year, with a mean of 34.34 ± 11.58 . At time of study inclusion, they all underwent full clinical examination after full history taking, pulmonary function tests, measuring body mass index and blood sampling.

Results: Serum cholesterol was significantly higher in asthmatic with overweight (mean 219.93 ± 60.02 mg/dl, $P < 0.001$), asthmatic with metabolic syndrome (mean 290.08 ± 90.95 mg/dl, $P < 0.001$), allergic rhinitis with overweight patients (mean 233.69 ± 81.59 mg/dl, $P < 0.001$) than in control subjects (mean 145.95 ± 36.52 mg/dl). It was of no significant difference in asthma with normal weight patients (mean 139.09 ± 50.36 mg/dl, P -value 0.4291) and allergic rhinitis with normal weight patients (mean 155.81 ± 49 mg/dl, P -value 0.2610) than in control subjects. Serum triglycerides was significantly higher in asthma with normal weight patients (mean 109.65 ± 33.75 mg/dl, P -value 0.0123), asthma with overweight (mean 184.02 ± 60.11 mg/dl, $P < 0.001$), asthma with metabolic syndrome (mean 222.82 ± 65.12 mg/dl, $P < 0.001$) and allergic rhinitis with overweight patients (mean 173.62 ± 54.27 mg/dl, $P < 0.001$) than of control subjects (mean 92.63 ± 35.37 mg/dl). It was of no significant difference in allergic rhinitis with normal weight patients (mean 101.56 ± 36.29 mg/dl, P -value 0.2340) than of control subjects. High density lipoprotein-cholesterol was significantly lower in asthma with overweight group (mean 33.32 ± 4.82 mg/dl, $P < 0.001$), asthma with metabolic syndrome (mean 36.03 ± 3.51 mg/dl, $P < 0.001$), allergic rhinitis with overweight (mean 35.82 ± 4.84 mg/dl, $P < 0.001$) than in control healthy group (mean 40.28 ± 6.05 mg/dl). It was of no significant difference between asthma with normal weight patients (mean 42.92 ± 10.56 mg/dl, P -value 0.1215), allergic rhinitis with normal weight (mean 41.45 ± 4.44 mg/dl, P -value 0.2600) than control subjects. Low density lipoprotein-cholesterol was significantly higher in asthma with overweight (mean 151.31 ± 63.53 mg/dl, $P < 0.001$); asthma with metabolic syndrome (mean 181.15 ± 88.27 mg/dl, $P < 0.001$); allergic rhinitis with normal weight (mean 108.42 ± 38.05 mg/dl, $P < 0.01$); allergic rhinitis with overweight (mean 163.49 ± 80.92 mg/dl, $P < 0.001$).

It was significantly lower in asthma with normal weight patients (mean 68.76 ± 32.66 mg/dl. $P < 0.05$) than in control subjects (mean 85.84 ± 39.39 mg/dl).

Conclusion: Asthma and / or allergic rhinitis in Iraqi population were associated with dyslipidemia, whether in the presence of metabolic syndrome, or the patients were with normal or abnormal weight.

Key words: Asthma, Allergic rhinitis, dyslipidemia, cholesterol, Triglycerides, HDL, LDL, VLDL, Iraq.

Introduction:

Asthma is one of the most common chronic diseases worldwide.[1] A large percentage of children and adults with asthma also have allergic rhinitis (AR) [2]. The link between AR and asthma has long been of interest to physicians. Recently, extensive research has established that epidemiologic and therapeutic links exist between AR and asthma. [3] A number of epidemiologic studies have shown an association between asthma and allergic rhinitis. In a review of five large studies that included populations of children and adults, [4] the prevalence of asthma ranged from 3.6% to 5% in subject without rhinitis versus 10.8% to 32% in subject with rhinitis. In a 23 year follow-up study in university students, [5] asthma developed in 10.5% of subjects with AR, whereas it developed in only 3.6% of subjects without AR. In addition, the reported lifetime prevalence of AR among adults with asthma ranges from 50% to 100%, varying by study design and geographical areas. [6]

Asthma and AR are both inflammatory and immunological diseases of the airways. The similarities between AR and asthma in epidemiologic and pathophysiological features suggest that AR and asthma represent the same syndrome, the chronic allergic respiratory syndrome.[7] Seventy eight percent of asthmatic patients were with nasal symptoms and thirty eight percent of subjects with allergic rhinitis were with asthma as reported by the American Academy of Allergy, Asthma, and Immunology [8]. Although, there are several surveys assessing the association between AR and asthma in different geographical areas worldwide, however, none was performed in large scale study. One study was performed to clarify this association between asthma and AR in Iraq population [9]. The study findings indicated that the frequency of allergic rhinitis was 61.6% among individuals with asthma versus 6% among non-asthmatic (control) subjects (Odd Ratio [OR] = 25.5; $P < 0.0001$). All studies indicated a significant frequency of AR among asthmatic patients in comparison with non-asthmatic subjects, whether the patients were adults or children (OR for adults = 14.9 and 22.5, for children 34.7 and 48.4; $P < 0.001$ for all). Furthermore, the high frequency of AR in asthmatic patients was seen whether the study was a community based study (CBS) (OR = 14.9 and 48.4; $P < 0.0001$) or a hospital based study (HBS) (OR = 22.5 & 34.7; $P < 0.0001$). The frequency of current asthma was 51.8% among individuals with AR versus 5.4% among control subjects (OR = 23.1; $P < 0.0001$).

Because of these relations, various researchers have referred to allergic rhinitis and asthma as allergic united airway disease [10-14] and recently as combined AR and asthma syndrome (CARAS) [15]. Since, AR and asthma are both mediated by similar allergic inflammatory mechanism; we aimed to

evaluate changes of serum lipid concentration in subjects with allergic rhinitis and asthma.

Materials and Methods.

Study Design:

A case-control design was used. The method involves three groups, the cases which were two groups (asthmatic individuals group and individuals with allergic rhinitis group) and control group which includes normal persons who are non-asthmatic and non-allergic rhinitis persons (non-atopic).

Study Population:

The study was performed on asthmatic, allergic rhinitis patients and non asthmatic non allergic control subjects. A total of 348 persons included in the study, and their age range was from 16 to 60 years. The subjects included in the study were outpatients from asthma and allergy units in Baquba Teaching Hospital outpatient clinic at the period from 10/9/2010 to 10/5/2012. The diagnosis of asthma and allergic rhinitis was performed by specialist's physicians and was established according to American Thoracic Society Criteria [16]. Patients are considered atopic (asthmatic and allergic rhinitis) by positive skin test to at least one common aeroallergen. Normal volunteers were also involved in the study as healthy control. None of them had any previous history of lung or allergic disease and were not using any medications. They had (most of them) normal lung function test ($FEV_1 > 80\%$) and negative skin allergy test.

Asthmatic patients were classified into asthmatic with normal weight, asthmatic with overweight and asthmatic with metabolic syndrome [17]. Allergic rhinitis patients are classified into allergic rhinitis with normal weight and allergic rhinitis with overweight.

Exclusion Criteria:

Patients were excluded if they were smokers, if they had respiratory infection within the month preceding the study, malignancy, diabetic, heart failure, history of venous embolism, coronary heart disease, chronic obstructive pulmonary disease(COPD)(chronic bronchitis & emphysema) , patients on steroid treatment and liver or kidney disease. At enrolment, all individuals included in the study underwent full clinical examination, pulmonary function test, prick skin test, body mass index and blood sampling.

Sampling and Sampling Techniques:

The study sample consisted of two groups: one case group and another is control group. The case group is sub divided into five sub groups which are asthmatic patients with normal weight, asthmatic patients with overweight, asthmatic patients with metabolic syndrome, allergic rhinitis with normal weight and allergic rhinitis with overweight. Asthma group consist of 190 patient (66 male & 124 female), allergic rhinitis group consist of 110 patient (44 male & 66 female) and the control group consist of 48 patient (26 male & 22 female).

Data Collection:

For this research questionnaire interviews and face to face interviews were used to collect data from individuals (case and control groups). Most questions were one of two types: the multiple choice question which offers several fixed alternatives and yes/no question which offers a dichotomous choice. The interviewer explained to the all individuals the importance, aim and purpose of the research study. Also all questions are ideally asked in the same way during the data collection to achieve a high degree of validity and reliability. The questionnaire included issues about personal information (age, gender, address, marital status, environment, school, vaccination state). In addition it included socio-economic information.

The questionnaire was piloted by using a small sample consisting of 20 case individuals (10 men and 10 women) and 20 control individuals (10 men and 10 women). This gives the researcher ideas about the length of questions and whether all the respondents understand the questions in the same way. In addition, the pilot study was important for directing and guiding the qualified interviewers to adjust and improve performance in data collection.

Blood Sampling and Processing:

Twelve hours fast blood samples were collected from the cases and controls by well trained and experienced co-researchers. About 6.0 ml of venous blood was drawn from each individual. The serum samples were rapidly separated by centrifugation for 10 minute at room temperature at 3500 rpm. The separated serum was divided into two plastic tubes, one stored at 2-5°C for no more than 24 hours prior lipid profile determination.

Determination of Cholesterol:

Determination of cholesterol was carried out after enzymatic hydrolysis and oxidation using a commercially available diagnostic system (Biolab.Fr, France) test kits. The colorimetric indicator is quinoneimine which is generated from 4-aminoantipyrine and phenol by hydrogen peroxide under the catalytic action of peroxidase [18].

Determination of High Density Lipoprotein-cholesterol (HDL-c)

Determination of HDL-c after serum precipitation was carried out by adding phosphotungstic acid and magnesium ions, using a commercially available diagnostic system (Biolab.Fr, France) test kits. Chylomicrons, very low density lipoprotein, and low density lipoprotein are precipitated by adding phosphotungstic acid and magnesium ions to the sample. Centrifugation leaves only the HDL in the supernatant. Their cholesterol content is determined enzymatically using Ecoline S+ cholesterol [19]

Determination of Low Density Lipoprotein-cholesterol (LDL-c)

Determination of LDL-c was calculated from the primary measurements using the empirical equation of **Friedewald et al. (1972)** [20]

$$\text{LDL-c (mg/dl)} = \text{total cholesterol} - \text{triglyceride}/5 - \text{HDL-c}$$

Determination of Triglyceride

Determination of triglyceride was carried out after enzymatic splitting with lipoprotein lipase using a commercially available diagnostic system (Biolab.Fr, France) test kits. Indicator is quinoneimine which is generated from 4-aminoantipyrine and 4-chlorophenol by hydrogen peroxide under the catalytic action of peroxidase [21]

Measurement of BMI

The body weight of each individual dressed in light clothing without shoes using a carefully calibrated electrical balance (*e-Accura*), the height of each individual was measured using vertical measuring rod. BMI was calculated as weight (kg) divided by squared height (m²) [22].

Data Analysis

Data were analyzed using Statistical Package of Social Sciences (SPSS) system (version 20). Descriptive statistic and frequency distributions were generated to make comparison among the variable and health profile of the case group and the control group. A conditional logistic regression analysis was used to determine the risk factors, which contribute to the development of obesity. Analysis of variance (ANOVA) was used to evaluate the differences in leptin, case and control groups. It used to test the significant of differences between means. A student *t*-test was used to investigate the effect of gender on serum leptin at all individuals and chi-square was used to measure the differences between nonparametric frequency data.

Results:

Our study population is composed of 348 individuals; 300 individuals are case groups and 48 individual is control group. The mean age of the patients included in the study was 34.34 ± 11.58 years, the mean age for male was 32.21 ± 12.58 years and for female was 37.26 ± 8.72 years. A total of 348 individuals are included in this study from them 136(39%) individuals were males and 212 (61%) individuals were females.

Cholesterol:

The mean of cholesterol is significantly higher in the groups of asthma with overweight, asthma with metabolic syndrome, allergic rhinitis with over weight than in control group (219.931 ± 60.029 mg/dl; 290.08 ± 90.952 mg/dl; 233.695 ± 81.593 mg/dl, respectively; $P < 0.001$ for all). While it is not of significant statistical difference in asthma with normal weight and in allergic rhinitis with normal weight groups than in control (139.09 ± 50.364 mg/dl, 155.814 ± 49 mg/dl and $P = 0.4291, 0.2610$ respectively), Table 1.

Table 1 Serum Cholesterol in case and control groups:

	Asthma With Normal weigh	Asthma With Overweight.	Asthma With Metab. Syndrome.	Allergic rhinitis with normal Weight.	Allergic R. with overweight	Control
	Total(70)	Total(64)	Total(56)	Total(62)	Total(48)	Total(48)
Mean	139.09	219.93	290.08	155.82	233.69	145.95
Median	138.5	218.95	325	156.8	216.3	142.8
S.D	50.36	60.03	90.95	49	81.59	36.53
P.V	0.42	<0.001	<0.001	0.26	<0.001	

Table 2 shows that the study of serum cholesterol in case and control groups according to gender distribution, mean of cholesterol was significantly higher in male and female individuals of the groups asthma with overweight, asthma and metabolic syndrome and allergic rhinitis with over weight than of control group and mean was 242.16 ± 72.932 mg/dl (male) , 208.28 ± 49.077 mg/dl (female) 348.35 ± 18.809 mg/dl; 275.51 ± 89.597 mg/dl; 263.75 ± 118.18 mg/dl, 223.64 ± 64.302 mg/dl and $P < 0.001$, while the mean was not with a statistical significant difference between male and female individuals in the groups of asthma with normal weight and allergic rhinitis with normal weight than that of the control subjects. Mean was 146.66 ± 56.322 mg/dl (male) 131.20 ± 43.12 mg/dl (female); 161.8 ± 48.188 mg/dl; 149.84 ± 49.948 mg/dl, respectively.

Table 2 Serum Cholesterol in case and control group according to gender distribution:

	Asthma With Normal weight.		Asthma With Overweight		Asthma With Metabolic Syndrome		Allergic rhinitis with normal Weight		Allergic Rhinitis with overweight		Control	
	M(34)	F(36)	M(20)	F(44)	M(12)	F(44)	M(32)	F(30)	M(12)	F(36)	M(26)	F(22)
Mean	146.66	131.28	242.16	208.28	348.35	275.51	161.8	149.84	263.85	223.64	144.3	147.49
Median	152.3	133.3	247	214.2	374.95	309.2	169.65	140.7	258.25	216.3	152.25	142.8
S.D	56.32	43.12	72.93	49.077	18.809	89.597	48.188	49.948	118.180	64.302	34.67	39.16
P. V	0.85	0.16	<0.001	<0.001	<0.001	<0.001	0.15	0.86	<0.001	<0.001		

Triglyceride

The mean serum of triglyceride was significantly higher in asthma with normal weight, asthma with overweight, asthma with metabolic syndrome and allergic rhinitis groups than in control group (109.651 ± 33.757 mg/dl,

184.02±00.114 mg/dl, 222.826±65.127 mg/dl, 173.621±54.27 mg/dl, respectively and mean for control group was 92.631±35.376 mg/dl. P= 0.0125 for asthma with normal weight group and <0.001 in other groups), Table 3. Additionally, the mean of serum triglyceride at allergic rhinitis group was 101.564±36.298 mg/dl and without significant difference from the value in control group, P=0.23, Table 3.

Table 3 Serum Triglyceride in case and control groups:

	Asthma With Normal weigh	Asthma With Overweight	Asthma With Metabolic Syndrome	Allergic rhinitis with normal Weight	Allergic Rhinitis with overweight	Control
	Total(70)	Total(64)	Total(56)	Total(62)	Total(48)	Total(48)
Mean	109.651	184.02	222.826	101.564	173.621	92.631
Median	105	186	211.6	90.4	161	90
S.D	33.757	60.114	65.127	36.298	54.27	35.376
P. V	0.0125	<0.001	<0.001	0.2340	<0.001	

The mean of serum triglyceride was higher in asthma with overweight, asthma with metabolic syndrome and allergic rhinitis with over weight than in control groups (181.33±61.868 mg/dl (male), 185.3±59.908 mg/dl (female); 219.88±33.12 mg/dl, 223.86±73.579 mg/dl; 163.16±37.944 mg/dl. 177.48±59.134 mg/dl and P<0.0001). There is no significant difference between mean of triglyceride for the groups of asthma with normal weight and allergic rhinitis with normal weight than that of the control groups (104.36± 29.42 mg/dl (male), 114.05± 36.81mg/dl (female); 102.7± 34.36 mg/dl, 100.33± 38.99 mg/dl respectively, P>0.05), Table 4.

Table 4 Serum Triglyceride in case and control group according to gender distribution:

	Asthma With Normal weigh		Asthma With Overweight		Asthma with Metabolic Syndrome		Allergic rhinitis with normal weight		Allergic Rhinitis with overweight		Control	
	M(34)	F(36)	M(20)	F(44)	M(12)	F(44)	M(32)	F(30)	M(12)	F(36)	M(26)	F(22)
Mean	104.36	114.05	181.33	185.3	219.88	223.86	102.7	100.33	163.16	177.48	88.53	96.81
Median	105	103	196	185	202.95	218.5	90.4	85.3	146	168	90	104
S.D	29.42	36.81	61.868	59.908	33.12	73.579	34.36	38.99	37.944	59.134	31.975	38.930
P. V		0.0959	<0.001	<0.001	<0.001	<0.001	0.1485	0.7610	<0.001	<0.001		

High Density Lipoprotein-cholesterol study:

The study results revealed that mean of high density lipoprotein-cholesterol was significantly lower in case group's asthma with overweight, asthma with metabolic syndrome and allergic rhinitis with over weight than in control group. (33.32 ± 4.824 mg/dl, 36.034 ± 3.517 mg/dl, 35.822 ± 4.849 mg/dl for the case groups and 40.283 ± 6.037 mg/dl for the control group. $P < 0.001$ for asthma with overweight and asthma with metabolic syndrome group and equal to 0.0002 for allergic rhinitis with overweight group). However, the mean was of no significant difference for asthma with normal weight and allergic rhinitis with normal weight than of control group; mean was 42.926 ± 10.367 mg/dl, 41.453 ± 4.449 mg/dl and P-value was 0.1215 and 0.2600. Table 5.

Table 5 HDL-C in case and control groups:

	Asthma With Normal weigh	Asthma With Overweight	Asthma With Metabolic Syndrome	Allergic rhinitis with normal Weight	Allergic Rhinitis with overweight	Control
	Total(70)	Total(64)	Total(56)	Total(62)	Total(48)	Total(48)
Mean	42.926	33.32	36.034	41.453	35.822	40.283
Median	40.2	33	36.7	40.9	34.6	38.5
S.D	10.567	4.824	3.517	4.449	4.849	6.057
P. V	0.1215	<0.001	<0.001	0.2600	0.0002	

In study of HDL-c in case and control groups according to gender our results revealed that HDL-c mean is significantly lower in asthma with overweight; asthma with metabolic syndrome and allergic rhinitis with overweight than of control group; mean was 32.98 ± 3.156 mg/dl (male), 33.35 ± 5.465 mg/dl (female); 32.5 ± 2.065 mg/dl, 36.89 ± 3.418 mg/dl; 32.38 ± 2.298 mg/dl, 31.11 ± 4.495 mg/dl for case groups and it was 38.04 ± 4.509 mg/dl(male), 42.98 ± 6.62 mg/dl(female) for the control. $P < 0.001$ for male and female in asthma with overweight group, equal to 0.0003(male), less than 0.001(female) for asthma and metabolic syndrome, and equal to 0.0002(male), less than 0.001(female) for allergic rhinitis and overweight group. While the mean was of no significant differentness in asthma with normal weight and allergic rhinitis with normal weight than of control group, mean was 40.42 ± 11.044 mg/dl (male), 45.62 ± 9.48 mg/dl(female); 39.25 ± 2.505 mg/dl(male), 43.66 ± 4.89 mg/dl(female). P-value was 0.3055 (male), 0.2604(female); 0.2233 and 0.6779, (female) respectively. Table 6.

Table 6 HDL-C in case and control group according to gender distribution:

	Asthma With Normal weight		Asthma With Over weight		Asthma With Metabolic Syndrome		Allergic rhinitis with normal weight		Allergic rhinitis with over weight		Control	
	M(34)	F(36)	M(20)	F(44)	M(12)	F(44)	M(32)	F(30)	M(12)	F(36)	M(26)	F(22)
Mean	40.42	45.62	32.98	33.35	32.5	36.89	39.25	43.66	32.38	31.11	38.04	42.98
Median	40	45	33	33.3	32.3	37.8	39.6	41.95	31.35	37.85	37	39.8
S.D	11.044	9.480	3.156	5.465	2.0655	3.418	2.505	4.890	2.293	4.945	4.509	6.62
P. V	0.3055	0.260	<0.001	<0.001	0.0003	0.001	0.2233	0.6779	0.0002	<0.001		

Low Density Lipoprotein-cholesterol Study

The study of LDL-c as total groups without gender distribution had shown that all the case groups has significant difference in mean than of control group but of different significances. Mean of case groups:, asthma with overweight, asthma with metabolic syndrome, allergic rhinitis with normal weight and allergic rhinitis with overweight were significantly higher than in control mean was 151.318 ± 63.534 mg/dl, 181.151 ± 88.279 mg/dl, 108.422 ± 38.05 mg/dl, 163.49 ± 80.92 mg/dl respectively while it was significantly lower in asthma with normal weight than in control .mean of control group was 85.842 ± 39.391 mg/dl. P-value was 0.0179 for asthma with normal weight, 0.0083 for allergic rhinitis with normal weight and less than 0.0001 for the others. Table 7.

Table 7 LDL-c in case and control groups:

	Asthma With Normal weigh	Asthma With overweight	Asthma With Metabolic Syndrome	Allergic rhinitis with normal Weight	Allergic Rhinitis with overweight	Control
	Total(70)	Total(64)	Total(56)	Total(62)	Total(48)	Total(48)
mean	68.769	151.318	181.151	108.422	163.49	85.842
median	61.7	152.4	161.7	114.4	147	92.8
S.D	32.665	63.534	88.279	38.05	80.92	39.391
P. V	0.0179	<0.001	<0.001	0.0083	<0.001	

In study of LDL-C according to gender the results revealed that LDL-C mean is significantly higher at asthma with overweight, asthma with metabolic

syndrome, allergic rhinitis with overweight groups than for control groups,; mean of these case groups was 176.78±76.02 mg/dl (male) (P<0.001), 137.94±52.07 mg/dl (female) (P<0.001); 256.5± 69.601 mg/dl (P<0.001), 154.65±78.865 mg/dl(P=0.0004); 198.8± 113.805 mg/dl, (P <0.001), 153.6±60.501 mg/dl (P<0.001), of control group it was 86.09±20.542 mg/dl (male), 85.59± 38.553 mg/dl (female). Table 8

For asthma with normal weight there is no significant difference between male and female groups compared with that of control subjects, mean was 68.1±39.26 mg/dl (male), (P=0.0661), 69.39± 25.61 mg/dl (female) (P=0.0689). Allergic rhinitis with normal weight there is significant difference between mean of male group and male group of control, mean was 115.05± 26.323 mg/dl (P=0.001), while there is no significant difference between female group and that of female control subjects and mean was 90.7± 54.16 mg/dl (P-value=0.7164). Table 8.

Table 8 LDL-c in case and control group according to gender distribution:

	Asthma with Normal weight		Asthma With Over weight		Asthma With Metabolic Syndrome		Allergic rhinitis with normal weight		Allergic rhinitis with over weight		Control	
	M(34)	F(36)	M(20)	F(44)	M(12)	F(44)	M(32)	F(30)	M(12)	F(36)	M(26)	F(22)
Mean	68.1	69.39	176.78	137.94	256.5	154.65	115.05	90.7	198.8	153.6	86.09	85.59
Median	68.1	59.87	185	139.8	272.55	144.7	119.5	90.7	190.35	160.1	96.55	78.3
S.D	39.26	25.61	76.02	52.07	69.60	78.86	26.32	54.16	115.80	60.50	20.54	38.55
P. V	0.066	0.068	<0.001	0.001	<0.001	0.0004	<0.001	0.716	<0.001	<0.001		

Discussion:

Lipid and Lipoprotein assays (LPA) form one of the special investigations in vascular disease conditions and in most chemical pathology laboratories worldwide [23-26]. Dyslipidemia has been found to occur as a result of change in diet to high fatty foods (high levels of saturated fats elevate serum lipids) Caucasian lifestyle [27-29]. LPA and its application in the management of patients with cardiovascular diseases (including ischemic heart disease) and monitoring of diabetic patients has been emphasized [30,31] ; while on the contrary there is paucity of information on LPA levels in asthmatics. The effect of hyperlipidaemia (HL) on asthma has not been fully addressed; and concluded that hypercholesterolemia is a potential risk factor independent of obesity [32]. Recent literature implicates a proinflammatory role for hypercholesterolemia in asthma with multiple cell types involved in the pathophysiology of asthmatics.

Hyperlipidaemia, a combination of hypertriglyceridaemia (HT) and hypercholesterolemia (HC) may either primarily be due to genetically determined disorders or secondary as a result of acquired causes [33-37]. These include obese individuals, alcoholics, individuals of high social status as well as

malnourished children [38]. Several studies, however, have shown beneficial effects of reversing hyperlipidaemia by either primary or secondary prevention [39-41]. This has been well established in various disease conditions and consequently the need to have a separate reference value for asthmatics in our community. Hence this study was designed to assess the haematological and LPA order to evaluate it as a possible risk factor. The outcome of this study will enhance our perception of dyslipidemia as a current and actively progressive medical problem that can be contained by different measures to prevent its complications.

The present study results shows a significant increase in serum cholesterol in the groups asthma with overweight, asthma with metabolic syndrome, allergic rhinitis with overweight than of control subjects. This increase was seen in both genders. Cholesterol was of no significant difference in asthma with normal weight and allergic rhinitis with normal weight groups.

Serum triglycerides was significantly higher in the groups (male and female) asthma with overweight, asthma with metabolic syndrome and allergic rhinitis with overweight, while it is of no significant change in the groups asthma with normal weight and allergic rhinitis with normal weight (male and female) than of control individuals.

In this study HDL-c was significantly lower (male and female) in the groups of asthma with overweight, asthma with metabolic syndrome and allergic rhinitis with overweight than of control group. While it is of no significant difference at asthma with normal weight and allergic rhinitis with normal weight groups. Additionally, LDL-c was significantly higher in the groups asthma with overweight (male and female) asthma with metabolic syndrome (male and female) and allergic rhinitis with normal weight (male only) and allergic rhinitis with overweight (male and female) than in control subjects, while it is of no significant difference in the groups asthma with normal weight (male and female), allergic rhinitis with normal weight (female) than in control subjects.

Extensive research on lipids and lipoprotein profile has been carried out in various disease conditions especially cardiovascular/coronary artery disease (CAD) [42-44] and diabetes mellitus [45, 46]. Current epidemiologic studies have even shown that dyslipoproteinaemia with low concentration of HDL and elevated serum TG is associated with a particularly high incidence of CAD [47, 48]. However, there is paucity of information on the role and pattern of LPA in asthmatics in Africa and in the Asia. The findings of this study could therefore serve to evaluate the pattern of lipid profile in relation to age and sex distribution in asthmatics. Dyslipidemia in asthmatics and/or allergic rhinitis subjects with overweight and obese is a risk factor that need follow up as they are prone to develop cardiovascular diseases.

The values of LPA in adults are consistent with the general knowledge on factors affecting biochemical analytes worldwide. Factors such as age, geographical location, and diet, extent of exercise regimen, obesity lifestyle and genetics influence the serum lipid levels in all categories of people [49, 50]. The common denominator would be lack of physical activity associated with unhealthy eating habits; as dietary modification has been reported to contribute to the overall health of asthmatics [51]. The serum lipid concentrations from an early age of one to 15 years fluctuate after which they remain stable through to old age [52]. The mean age of the studied asthmatics was 52 ± 13 years with

females predominating (57.5%). The LP concentrations have also been noted to be different at the gender level [53]. The female LP concentrations in this study were all higher than the male values probably because of the endogenous sex hormonal variations in women.

A previously reported study [54] revealed a combined hypertriglyceridaemia (HT, >2.3mmol/L) and a significant hypercholesterolemia (HC, >5.2mmol/L) according to the ATP III definition. The recommendation of ATP III adopted "the lower, the better" for cholesterol level; therefore a cut-off value of 5.2 mmol/L was adopted to define HC. From the analysis of data obtained here, it shows that hyperlipidaemia is common in asthmatics thereby putting them at increased risk for the development of CAD as well as other disorders related to excess lipids. In a related study combination of CVD with asthmatics was observed in 88.3% of patients [55]; and another study detected various CVD of varying severity in asthmatic patients [56]. There was also a low HDL-C level and elevated serum TG in this study which is associated with a high risk of CAD; thus providing an additional risk factor in asthmatics as seen in cardiovascular diseases. It has even been suggested that HC is a significant factor in the development of atherosclerosis and that there is a correlation between the HC in childhood and the stage and extent of atherosclerotic lesions [57, 58]. This has led various organizations (National Cholesterol Education Program and American Pediatric Academy) to improve cholesterol screening programs to determine individuals in early stage who were predisposed to atherosclerosis; and to recommend a reduction in the amount of cholesterol and saturated fat in the diet of the whole population [59,60]. Although high plasma HDL-C levels prevent the deposition of vascular cholesterol and development of atherosclerosis, the elevation of LDL-C level (>3.0mmol/L) which is the major carrier of cholesterol leads to the early development of atherosclerosis. The elevated LDL-C as recorded in the present study has been known to be susceptible to lipid peroxidation which increases its atherogenic potential. This is due to the fact that the oxidative and peroxidative forms of LDL-C could not be removed from the plasma via LDL receptors in normal cells and accumulates in the atherosclerotic lesions via alternative receptors of the macrophages and endothelial cells, thus stimulating the hypertrophy of blood vessel wall.

In conclusion, in overweight and obese subjects with asthma and / or allergic rhinitis, there was high rate of dyslipidemia which may lead to development of cardiovascular diseases. This finding suggests a need for monitoring lipids profile in individuals with asthma and allergic rhinitis.

References:

1. Masoli M, Fabian D, Holt S, Beasley R. The global burden of asthma: executive summary of the GINA Dissemination Committee report. *Allergy* 2004; 59:469-78.
2. Price D, Holgate S. Improving outcomes for asthma patients with allergic rhinitis: the MetaForum conferences. *BMC Pulm Med* 2006; 6 (Suppl 1):S7.
3. Volcheck GW. Does rhinitis lead to asthma? Evidence for the one-airway hypothesis. *Postgrad Med* 2004; 115:65-8.
4. Leynaert B, Neukirch F, Demoly P, Bouquet J. Epidemiologic evidence for asthma and rhinitis comorbidity. *J Allergy Clin Immunol* 2000; 106:S201-5.

5. Settipane RJ, Hagy GW, Settipane GA. Long-term risk factors for developing asthma and allergic rhinitis: a 23-year follow-up study of college students. *Allergy Proc* 1994; 15:21-5.
6. Gaugris S, Sazonov-Kocevar V, Thomas M. Burden of concomitant allergic rhinitis in adults with asthma. *J Asthma* 2006; 43:1-7.
7. Pawankar R. Allergic rhinitis and asthma: are they manifestation of one syndrome? *Clin Exp Allergy* 2006; 36:1-4.
8. Casale TB, Amin BV. Allergic rhinitis_asthma interrelationships. *Clin Rev Allergy Immunol* 2001; 21:27-49.
9. Alsamarai AG, Ammar M Alwan, Amina Hamed Ahmad, et al. The Relationship between Asthma and Allergic Rhinitis in the Iraqi Population. *Allergology International*. 2009; 58:549-555.
10. Taramarcaz P, Gibson PG. The effectiveness of intranasal corticosteroids In combined allergic rhinitis and asthma syndrome. *Clin Exp Allergy* 2004; 34:1883-9.
11. The Allergy Report. Overview of allergic diseases: diagnosis, management, and barriers to care. Vol 1. Milwaukee: American Academy of Allergy, Asthma, and Immunology, 2000.
12. Leynaert B, Neukirch F, Demoly P, et al. Epidemiologic evidence for asthma and rhinitis comorbidity. *J Allergy Clin Immunol* 2000; 106:201-5.
13. Bugiani M, Carosso A, Migliore E, et al. Allergic rhinitis and asthma comorbidity in a survey of young adults in Italy. *Allergy* 2005; 60:165-70.
14. Koh YY, Kim CK. The development of asthma in patients with allergic rhinitis. *Curr Opin Allergy Clin Immunol* 2003; 3:159-64.
15. Passalacqua G, Canonica GW. Impact of rhinitis on airway inflammation: Biological and therapeutic implications. *Respir Res* 2001; 2:320-3.
16. Bush A, Menzies-Gow A. Phenotypic differences between pediatric and adult asthma. *Proc Am Thorac Soc* 2009; 6 (8): 712-9.
17. Kahn BB, Flier JS: Obesity and insulin resistance. *J Clin Invest* 2000; 106:473.
18. Sigurs N, Bjarnason R, Sigurbergsson F, Kjellman B. Respiratory syncytial virus bronchiolitis in infancy is an important risk factors for asthma and allergy at age 7. *Am J Respir Crit Care Med* 2000; 161(5):1501-7.
19. Stein RT, Sherrill D, Morgan WJ, Holberg CJ, Halonen M, Taussig LM, Wright AL, Martinez FD. Respiratory syncytial virus in early life and risk of wheeze and allergy by age 13 years. *Lancet* 1999; 354(9178):541-5.
20. Illi S, von Mutius E, Lau S, Bergmann R, Niggemann B, Sommerfeld C, Wahn U, MAS Group. Early childhood infectious diseases and development of asthma up to school age: a birth cohort study. *BMJ* 2001; 322(7283):390-5
21. Shaheen SO, Aaby P, Hall AJ, Barker DJ, Heyes CB, Shiell AW, Goudiaby A. Measles and atopy in Guinea-Bissau. *Lancet* 1996; 347(9018):1792-6.
22. Torday JS, Sun H, Wang L, Torres E. Pre- and Postnatal Lung Development, Maturation and Plasticity. Leptin mediates the parathyroid hormone-related protein paracrine stimulation of fetal lung maturation. *Am J physiol Lung Cell Mol Physiol* 2002; 282:405-10
23. Stamler J, Daviglus ML, Garside DB, Dyer AR, Greenland P, Neaton JD. Relationship of baseline serum cholesterol levels in 3 large cohorts of younger men to long-term coronary, cardiovascular and all cause mortality and to longevity. *JAMA*. 2000; 284:311-318

24. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation*. 1998; 97:1837-1847.
25. Grundy SM, Balady GJ, Criqui MH, Fletcher G, Greenland P, Hiratzka LF, et al. Guide to primary prevention of cardiovascular diseases. A statement for healthcare professionals from the Task Force on Risk Reduction. American Heart Association Science Advisory and Coordinating Committee. *Circulation*. 1997; 95:2329-2331.
26. Hokanson JE, Austin MA. Plasma triglyceride level is a risk factor for a meta- analysis of population based prospective studies. *J Cardiovasc Risk*. 1996; 3: 213-219.
27. Agboola-Abu CE, Akinlade AK, Ohwovoriole AK. Pattern of lipidaemia in newly diagnosed Nigerian patients with non-insulin dependent diabetes mellitus. *Nigerian Journal of Internal Medicine*. 1998; 1:1-6.
28. Jarikre AK, Mommoh JAF. Plasma total cholesterol, high density lipoprotein cholesterol and low density lipoprotein cholesterol levels in liver cirrhosis in Nigerians. *Nigerian Quarterly Journal of Hospital Medicine*. 1996; 6: 157-159.
29. Holman R. Atherosclerosis: a paediatric nutrition problem. *Am J Clin Nutr*. 1961; 9:565-569.
30. Vinter Repalust N, Jurkomo L, Katie M, Simunovic R, Petric D. Disease duration, patient compliance and presence of complications in diabetes patients. *Acta Med Croatica*. 2007; 61(1) : 57-62.
31. Gustafsson I, Brendorp B, Siebaek M, Burchardt H, Hildebrandt P. Influence of diabetes and diabetes gender interaction on the risk of death in patients hospitalized with congestive heart failure. *J Am Coll Cardiol*. 2004; 43(5) : 771-777.
32. Al-Shawwa B, AlHuniti N, Titus G, AbuHasan M. Hypercholesterolemia is a potential risk factor for asthma. *J Asthma*. 2006; 43(3) : 231-233.
33. Hopkins PN, Stephenson S, Wu LL, Riley WA, Xin Y, Hunt SC. Evaluation of coronary risk factors in patients with heterozygous familial hypercholesterolemia. *Am J Cardiol*. 2001; 87:547-553.
34. Wiegman A, Rodenburg J, De Jongh S, Defesche JC, Bakker HD, Kastelein JJ, et al. Family history and cardiovascular risk in familial hypercholesterolemia: data in more than 1 000 children. *Circulation*. 2003; 107:1473-1478.
35. Lee MH, Lu K, Hazard S, Yu H, Shulenin S, Hidaka H, et al. Identification of a gene, ABCG5, important in the regulation of dietary cholesterol absorption. *Nat Genet*. 2001; 27:79-83.
36. Osser DN, Najarian DM, Dufresne RL. Olanzapine increases weight and serum triglyceride levels. *J Clin Psychiatry*. 1999; 60:767-770.
37. Ferrieres J, Lambert J, Lussier-Cacan S, Davignon J. Coronary artery disease in heterozygous familial hypercholesterolemia patients with the same LDL receptor gene mutation. *Circulation*. 1995; 92:290-295.
38. Buemann B, Tremblay A. Effect of exercise training on abdominal obesity and related metabolic complications. *Sport Med*. 1996; 21: 191-212.
39. Downs JR, Clearfield M, Weis S, Whitney E, Shapiro DR, Beere PA, et al. Primary prevention of acute coronary events with lovastatin in men and

- women with average cholesterol levels: results of AFCAPS/TexCAPS. Air Force/Texas Coronary Atherosclerosis Prevention Study. JAMA. 1998; 79: 1615-1622.
40. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. The Long Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group. N Engl J Med. 1998; 339:1349-1357.
 41. Gould AL, Rossouw JE, Santanello NC, Heyse JF, Furberg CD. Cholesterol reduction yields clinical benefits: impact of statin trials. Circulation. 1998; 97:946-952.
 42. Ford ES, Mokdad AH, Giles WH, Mensah GA. Serum total cholesterol concentration and awareness, treatment and control of hypercholesterolemia among US adults: findings from the National Health and Nutrition Examination Survey, 1999 to 2000. Circulation. 2003; 107:2185.
 43. Study Group, European Atherosclerosis Society. Strategies for the prevention of coronary heart disease: A policy statement of European Atherosclerosis Society. Euro Heart J. 1987; 8:77-88.
 44. Pirzado ZA, Sangi SA, Malik R. High density lipoprotein cholesterol (HDL) metabolism and its role in the ischemic heart disease. Pak J Med Res. 1999; 38(1) : 38-41.
 45. Abdul RAN, Olufunsho F. Hyperlipidaemia among Saudi diabetic patient's pattern and clinical characteristics. Ann Saudi Med. 1995; 15(3) : 240-243.
 46. Akbar DH. Hyperlipidaemia in diabetic patients in Saudi Arabia. Diabetes International. 2001; 11(1) : 17-18.
 47. Halle M, Berg A, Baumstark MW, Konig D, Huonker M, Keul J. Influence of mild to moderately elevated triglycerides on low density lipoprotein sub fraction concentration and composition in healthy men with low high density lipoprotein cholesterol levels. Atherosclerosis. 1999; 143(1) : 185-192.
 48. Al-Nozha MM, Arafah MR, AlMazrou YY, AlMaatouq MA, Khan NB, Khalil MZ, et al. Coronary artery disease in Saudi Arabia. Saudi Med J. 2004; 25:1165-1171.
 49. Laner RM, Lee J, Carte WR. Factors affecting the relationship between childhood and adult cholesterol: the Muscatine study. Pediatrics. 1988; 82:309-318.
 50. Webber LS, Hunter SM, Johnson CC, Srinivasan SR, Berenson GS. Smoking, alcohol and oral contraceptives. Effects on lipids during adolescence and young adulthood Bogalusa Heart Study. Ann NY Acad Sci. 1991; 623:135-154.
 51. Spector SL, Surette ME. Diet and asthma: has the role of dietary lipids been overlooked in the management of asthma? Ann Allergy Asthma Immunol. 2003; 90(4) : 371-377.
 52. John B. Screening of hypercholesterolemia in childhood. In Hypercholesterolemia of the child. Annales Nestle. 1994; 52: 14-24.
 53. Patrizia B, Giancarlo T, Franca E, Loreta P, Salvatore D, Mario M, et al. Lipoprotein metabolism during normal pregnancy. Am J Obstet Gynecol. 1999; 181(2) : 430-434.

54. Omotil CE, Elizabeth EE. Haematological and lipid profile assays in Nigerian asthmatics. www.jylw.com/guest/wzhtml/53/wz419117.htm.
55. Demko IV, Gordeeva NV, Petrova MM, Artiukhov IP. Clinical picture and treatment of bronchial asthma comorbid with cardiovascular diseases. Ter Arkh. 2007; 79(9) : 60-65.
56. Chicherina EN, Shipitsyna VV. Cardiovascular system in patients with bronchial asthma of varying severity. Probl Tuberk Bolezn Legk. 2003; 8:25-28.
57. McGill HC, McMahon CA, Herderick EE. Origin of atherosclerosis in childhood and adolescence. Am J Clin Nutr. 2000; 72(5suppl) : 1307-1315.
58. Franklin FA, Dashti N, Franklin CC. Evaluation and management of dyslipoproteinemia in children. Endocrinol Metab Clin North Am. 1998; 27(3) : 641-654.
59. NCEP expert panel on blood cholesterol levels in children and adolescents. Pediatrics. 1992; 89:495-501.
60. The American Academy of Pediatrics Committee on Nutrition Indications for cholesterol testing in children. Pediatrics. 1989; 83:141-142.