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Original Research Article

Study of lipid profile, calcium and iron status in patients with asthma

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Abstract

Background and Objective: The research will link these parameter levels and predict asthma severity to assist clinicians diagnose and treat asthma and avoid abrupt and severe airway blockage in patients with hyperirritability of airways and other allergic disorders, which are at high risk for deadly asthma.

Method: Department of Biochemistry, Osmania general hospital, Hyderabad, Telangana, India, conducted the investigation on 80 asthmatics and 40 controls who participated in the research. Total Cholestrol, Triglycerides, HDL, LDL, VLDL, Calcium, and Iron were measured. SPSS 17.0 was used to statistically examine the data.

Result: In stable asthma patients, Cholesterol, LDL, Triglycerides, Calcium, Iron, and HDL were significantly correlated. Exacerbating asthma had lower serum very low density lipoprotein, higher serum calcium levels and has higher serum iron concentrations than stable asthma.

Conclusion: In order to help doctors in the diagnosis and treatment of asthma and avoid abrupt and severe airway obstruction in hyperirritable airways, the current research examined lipid profile, calcium, and iron in aggravating and stable asthma patients.

Keywords: Asthma, lipid profile, triglycerides

Introduction

The hallmark of the asthma syndrome, which can appear in a variety of ways both on its own and in response to treatment, is airflow obstruction. People with asthma are more susceptible than the general population to a wide range of triggers because of a special type of inflammation in their airways. This increased sensitivity leads their airways to constrict excessively, which results in restricted airflow, wheezing, and other symptoms. Some persons with persistent asthma may have a lifelong airflow limitation, despite the fact that airway narrowing is frequently reversible [1-3].

Studies have shown a connection between asthma and the metabolic syndrome. Several biochemical and physiological risk factors, including as abdominal obesity, dyslipidemia, elevated blood pressure, and insulin resistance with or without glucose intolerance, are collectively known as the metabolic syndrome. It is currently unknown whether metabolic syndrome or one or more of its components are a risk factor for the airway illness asthma [4]. Uncertain underlying processes underlie the association between metabolic syndrome and asthma. It is believed that there may be a two-way interaction between the two illnesses. Objective diagnosis is usually confirmed by pulmonary function testing. Spirometry may not always pick up on airflow blockage because of its variability. In addition, the underlying disease mechanism responsible for various phenotypes could not be reflected in lung function tests. There is already widespread use of biological markers in the detection and management of cancer and cardiovascular disease. Biomarkers show significant promise as a noninvasive

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tool for improving clinical diagnosis, tracking disease development, and developing personalized therapy plans [5-7].

Additionally, hyperglycemia, insulin resistance, and other metabolic syndrome markers such dyslipidemia and dyslipidemia are linked to asthma. High levels of triglycerides and low-density lipoproteins (LDLs) have been linked to wheezing in adults, and low levels of serum high density lipoproteins (HDLs) have been linked to an increased risk of asthma in adolescents. Several studies have shown that the iron present in antioxidant enzymes like catalase is a critical element in many oxidative reactions, and free iron as a transition metal contributes to the generation of free radicals and catalyzes the transformation of HO into a highly reactive hydroxyl radical, both of which play a role in the development of asthma [8, 9].

Material and Method

The research was carried out between August 2011 and April 2012 at Department of Biochemistry, Osmania general hospital, Hyderabad, Telangana, India.

Inclusion criteria

1. Patients with asthma were further classified into two groups: those with stable asthma and those with worsening asthma

The Control Subjects:

1. spirometry revealed normal lung function (FEV,>80%);
2. skin allergy testing was negative;
3. patients were not taking any medications;
4. they did not have diabetes mellitus;
5. with no h/o liver disease;
6. with no h/o kidney disease;
7. h/o any other systemic illness.

Exclusion criteria

1. Patients having a history of rheumatological sickness, neuromuscular disease, cardiovascular disease, venous embolism, heart failure, coronary heart disease, or any other major medical condition were not included in the research.

Methodology

The purpose of the research was outlined to all 120 individuals (Cases + Controls)/Participants. Consent was received from each of the 120 participants. Eighty asthma patients were split into two categories: those with stable asthma and those with worsening asthma Forty healthy participants served as the study's "controls." Before sample collection, all participants fasted for 12 hours. All subjects were placed in the supine position for at least 5 minutes prior to venipuncture. Plasma (for lipid profile): 3 ml venous whole blood in 5 mg% EDTA collection container; serum: 3 ml venous whole blood in plain bottle and allowed to clot. The samples were analyzed in this way to get the information we needed. Within an hour of sample collection, serum was carefully separated to prevent hemolysis. Following parameters were examined in serum samples from all 120 subjects:

Spectrophotometric measurement of lipid profile in serum. Cholesterol oxidase and a spectrophotometer for measuring total cholesterol. HDL-L spectrophotometer enzymatic cholesterol oxidase technique. The enzymatic approach using triacylglycerol, a spectrophotometer, and GPO-DAP. low-density lipoprotein calculated using Friedwald's equation. VLDL-C-TG/5, for short. Test for complexone end-pint in calcium-o-cresolphthalein-depleted serum. Ferrozine-based serum iron detection.

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Result

Table 1: One way analysis of Variance was used for testing the significance difference among the three groups

Parameters	Group	N	Mean	SD	Minimum	Maximum
	GI	40	184.85	19.132	116	225
Cholesterol	G2	40	179.55	12.928	160	220
	G3	40	126.78	9.393	110	148
	Total	120	163.73	29.946	110	225
	GI	40	119.05	22.588	82	184
Triglycerides	G2	40	110.75	21.363	72	174
	G3	40	65.25	9.737	51	97
	Total	120	98.35	30.197	51	184
	GI	40	39.70	7.511	23	52
HDL	G2	40	32.20	7.367	20	46
	G3	40	54.25	8.915	40	72
	Total	120	42.05	12.117	20	72
	GI	40	122.33	22.482	82	184
LDL	G2	40	I 19.50	59.605	35	460
	G3	40	59.90	14.965	27	96
	Total	120	100.58	47.317	27	460
	GI	40	23.35	4.458	16	36
VLDL	G2	40	23.43	16.773	15	125
	G3	40	12.95	1.867	10	19
	Total	120	19.91	11.148	10	125
	GI	40	9.618	.8614	8.0	11.2
Calcium	G2	40	8.940	1.2322	3.2	10.8
	G3	40	20.893	2.3254	15.0	23.8
	Total	120	13.150	5.7284	3.2	23.8
	GI	40	111.08	12.219	80	135
Iron	G2	40	IO 1.48	10.789	78	120
	G3	40	201.73	11.594	180	220
	Total	120	138.09	46.779	78	220

Table 2: ANOVA was used for testing the multiple comparison of significance difference between groups (A) and with in groups (B).

Parameters		Sum of Squares	df	Mean Square	F	Sig
	A	82479.950	2	41239.975	199.104	$p<0.01$
Cholesterol	B	24233.975	117	207.128		
	Total	106713.925	119			
	A	67114.400	2	33557.200	94.847	$p<0.01$
Triglycerides	B	41394.900	117	353.803		
	Total	108509.300	119			
	A	l 0055.400	2	5027.700	79.317	$p<0.01$
HDL	B	7416.300	117	63.387		
	Total	17471.700	119			
	A	99426.950	2	49713.475	34.828	$p<0.01$
LDL	B	167004.375	117	1427.388		
	Total	266431.325	119			
	A	2905.217	2	1452.608	14.303	$p<0.01$
B 11882.775 117 IO 1.562						
VLDL						
	Total	14787.992	119			
	A	3605.959	2	1802.979	705.416	$p<0.01$
B 299.042 I 17 2.556						

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Calcium						
	Total	3905.000	119			
	A	244795.267	2	122397.633	917.704	$p<0.01$
Iron	B	15604.725	117	133.374		
	Total	260399.992	119			

Table 3: S Serum Levels of CHO in Controls, Stable asthma (A,case-1) and Exacerbating asthma (B,case-2)

Parameter	**Control**	**Case**		**Control**	**Case**	
		SA(A)	EA(B)		SA(A)	EA(B)
S.CHO	184.85	179.55	126.78	19.13	12.92	9.39

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The mean values of Serum Cholesterol are lower in exacerbating asthma cases compared to stable asthma cases and controls and it is statistically significant.

Table 4: S Serum Levels of TG in Controls, Stable asthma (A,case-1) and Exacerbating asthma (B,case-2)

Parameter	**Control**	**Case**		**Control**	**Case**	
		SA(A)	EA(B)		SA(A)	EA(B)
S.TG	119.05	110.7	65.25	22.58	21.36	9.73

The mean values of Serum triglyceride are lower in exacerbating asthma cases compared to stable asthma and controls and it is statistically significant

Table 5: Serum Levels of HDL in Controls, Stable asthma (A,case-1) and Exacerbating asthma (B,case-2)

Parameter	**Control**	**Case**		**Control**	**Case**	
		SA(A)	EA(B)		SA(A)	EA(B)
HDL	39.7	72.2	54.25	7.51	7.36	8.91

The mean values of Serum High density lipoprotein are higher in exacerbating asthma cases compared to stable asthma.

Table 6: Serum Levels of LDL in Controls, Stable asthma (A,case-1) and Exacerbating asthma (B,case-2)

Parameter	**Control**	**Case**		**Control**	**Case**	
		SA(A)	EA(B)		SA(A)	EA(B)
LDL	122.33	119.50	59.90	22.48	59.60	14.96

The mean values of Serum Low density lipoprotein are lower in exacerbating asthma cases compared to stable asthma.

Table 7: Serum Levels of VLDL in Controls, Stable asthma (A,case-1) and Exacerbating asthma (B,case-2)

Parameter	**Control**	**Case**		**Control**	**Case**	
		SA(A)	EA(B)		SA(A)	EA(B)
VLDL	23.35	23.43	12.95	4.45	16.77	1.86

The mean values of Serum Very Low density lipoprotein are lower in exacerbating asthma cases compared to stable asthma.

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Table 8: Serum Levels of Calcium in Controls, Stable asthma (A,case-I) and Exacerbating asthma (B,case-2)

Parameter	Control	Case		Control	Case	
		SA(A)	EA(B)		SA(A)	EA(B)
Ca	9.6	8.94	20.89	0.86	1.23	2.32

The mean values of Serum Calcium are higher in exacerbating asthma cases compared to stable asthma.

Table 9: Serum Levels of Iron in Controls, Stable asthma (A,case-I) and Exacerbating asthma (B,case-2)

Parameter	Control	Case		Control	Case	
		SA(A)	EA(B)		SA(A)	EA(B)
Fe	111.08	101.48	201.73	12.21	10.78	11.59

The mean values of Serum Iron are higher in exacerbating asthma cases compared to stable asthma.

Discussion

Although atopy remains the most significant risk factor for the onset of asthma, obesity is now being recognized as a contributor. Inhalational allergen exposure worsens airway inflammation, airway hyper-responsiveness, and symptoms in susceptible people [10]. According to the results of the current research, those with asthma whose symptoms are worsening tend to have lower blood Triglycerides concentrations than those with stable asthma. Serum HDL levels were found to be significantly higher (p<0.01) in patients with asthma exacerbation (54.25$\pm$8.91) compared to a control group (39.70$\pm$ 7.51) in the current investigation. Patients with stable asthma (32.20 $\pm$7.36) did not vary significantly from the control group. Serum HDL concentration was shown to be greater in individuals with worsening asthma compared to those with stable asthma. The current research found that the mean value of serum LDL was lower in patients with asthma exacerbation (59.90$\pm$ 14.96) compared to the control group (122.33$\pm$22.48). This difference was statistically significant (p<0.01). Still, neither the stable asthma patients (119.50$\pm$59.60) nor the control group showed statistically significant differences [11, 12].

Findings from the current research reveal that people with asthma whose symptoms are worsening had lower blood LDL concentrations. The current research found that the mean value of serum VLDL was lower in patients with asthma exacerbation (12.95$\pm$1.86) compared to the control group (23.35$\pm$4.45), with a statistically significant difference (p<0.01). In contrast, there was no statistically significant difference between the stable asthma patients (23.43$\pm$4.45) and the control group. According to the results of the current investigation, those with asthma whose symptoms are worsening tend to have lower blood VLDL concentrations compared to those with stable asthma. To synthesize surfactants, fatty acids must be readily available. The lipoprotein lipase enzyme may convert triglycerides in the bloodstream into free fatty acids. Triglycerides are conveyed in very low density lipoprotein (VLDL). VLDL does promote the production of surfactant phospholipids ". Exacerbation-related desease may have an effect on surfactant lipid production, leading to potential changes in surfactant composition and functions [13].

And Zimmerman and Myer "altered surfactant composition and function was described in a variety of inflammatory illnesses affecting the airways or the lung parenchyma, including asthma. Surfactant lipid production relies heavily on low-chain fatty acids for two reasons: first, they are a substrate for the biosynthesis of phospholipids, and second, they are activators of important enzymes involved in the synthesis of phosphotidylcholine ". Lung fatty acids needed for these functions may come mostly from plasma fatty acids or endogenous synthesis, depending on the stage of lung development. Surfactant metabolism is also

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influenced by fatty acids produced from circulating triglyceride rich lipoproteins such very low density lipoprotein (VLDL). The researchers Mallampall *et al*. found that very low density lipoprotein (VLDL) increases surfactant formation through interacting with lipoprotein lipase and, to a lesser extent, with cells' surface lipoprotein receptor. This imbalance between oxidants and antioxidants may then alter VLDL formation, suggesting a direct link between inflammation and this process [14, 15].

Asthma is more common among the obese, and being overweight may even make the condition worse, but losing weight might help those with asthma breathe easier. Obese people had a slightly higher incidence and prevalence of asthma, according to both cross-sectional and prospective cohort studies. Serum levels of cholesterol, triglycerides, LDL, and VLDL were all lower in asthma patients than in controls, whereas HDL-C levels were greater. This research found that the mean value of blood calcium was significantly higher in patients with asthma exacerbation (20.89±2.32) compared to controls (9.618±0.86). But there is no statistically significant difference between the stable asthma patients (9.618±0.86) and the control group. According to the results of the current research, people with asthma whose symptoms are worsening have a greater blood calcium content compared to those with stable asthma [16].

One of the first studies to show this was conducted by Gugger *et al*., who found that the average concentration of plasma ionized calcium was substantially lower than in healthy control. According to their findings, a change in calcium metabolism may play a role in bronchial asthma. Because calcium is the primary second messenger controlling ASM contraction, it was postulated that changes in calcium homeostasis, such as an increased calcium flow or altered calcium regulatory proteins, may play a crucial role in triggering ASM hyper reactivity in asthma. Initiation and maintenance of ASM cell contraction are dependent on calcium ions (Ca), and recent advances in our understanding of signal transduction pathways related to intracellular Carelease have broadened our understanding of coupling mechanisms in ASM. In addition, these advances serve as points of departure for the creation of new medications, with a different mechanism of action, to treat asthma. No matter the kind of asthma, the severity of the episode, or the degree of bronchial obstruction, patients with bronchial asthma had elevated serum calcium concentrations ", Higher blood calcium levels were seen during exacerbation compared to stable asthma, suggesting a positive relationship between serum calcium levels and disease severity [17].

Serum calcium was shown to be lower in treated asthma patients, which may be attributable to hyposensitization's role in regulating cytokine production and restoring the balance between Thl and Th2, inhibiting phospholipase C, and/or modulating oxidant activity created by effector cells. Patients with asthma who are experiencing an exacerbation had a significantly higher mean blood iron value (201.73 ± 11.59) compared to the control group (111.08± 12.21), as shown by the current research (P <0.01). Nonetheless, comparing the stable asthma patients (111.08± 12.21) to the control group shows no statistically significant differences. According to the results of the current research, people with asthma whose symptoms are worsening tend to have a greater blood iron content compared to those with stable asthma. This finding is consistent with those of others "[18].

Whereas, Vural *et al*. found no statistically significant difference between the asthmatic group and the control group in terms of blood iron levels. The biology and pathology of the lower respiratory tract rely heavily on iron metabolism ". Maintaining a healthy iron balance is crucial, as it is with many other aspects of inflammation. Cells involved in the inflammatory response, tissue healing, and the manufacture of mediators may all be stunted by a local deficit. To the contrary, an overabundance of iron, particularly unbound from an iron-binding protein, might lead to the production of harmful hydroxyl radicals [19].

Conclusion

The cellular and molecular mediators that may serve as biological indicators of lung damage have been the focus of research into the pathogenic process of lung injury. The results of the research point to a correlation between lower airway disorders and chronic inflammation

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lasting for a longer period of time. Therefore, the results of the current research imply that airway inflammation and hyperirrtability of airway mucosa contribute to attack remission when exposed to allergens and limit the success rate of therapy and recovery of patients. Ultimately, the present study measured serum lipid profile, calcium, and iron in both exacerbating and stable asthma cases to correlate these levels and predict the risk of severity of asthma, aid the clinician in diagnosis and implementation of appropriate treatment, and prevent the risk of sudden and severe airway obstruction in those with hyperirritability of airways and asthma. As a result, beta-receptor antagonists, theophylline, anticholinergics, cromylyn, nedocromil, or systemic or inhaled corticosteroids may improve the treatment and prognosis of the disorders.

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