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Original research article

Study of serum creatine kinase, lactate dehydrogenase in patients with asthma

[1]Dr. Lavanya Kurakula, [2]Dr. Audi Bhagya Lakshmi, [3]Dr. Asra Naweed

[1, 3]Assistant Professor, Department of Biochemistry, Mallareddy Institute of Medical Sciences, Suraram, Hyderabad, Telangana, India.

[2]Associate Professor, Department of Biochemistry, Mallareddy Institute of Medical Sciences, Suraram, Hyderabad, Telangana, India

Corresponding Author:
Dr. Lavanya Kurakula

Abstract

Background and Objective: The research measures blood creatine kinase, lactate dehydrogenase, in asthmatics with exacerbation and stable asthma. The research will link these levels and predict asthma severity to assist clinicians to diagnose and treat asthma and avoid abrupt and severe airway blockage in patients with hyperirritability of airways and other allergic disorders, who 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. 40 instances were stable asthma and 40 aggravating asthma. SPSS 17.0 was used to statistically examine the data.

Result: LDH and CK are higher in exacerbating asthma patients compared to stable asthma cases and controls.

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 blood creatine kinase, serum lactate dehydrogenase, in aggravating and stable asthma patients.

Keywords: Asthma, lactate dehydrogenase, serum creatine kinase

Introduction

Airflow blockage is a hallmark of the asthma syndrome, which manifests in a wide range of ways both on its own and in response to therapy. Due to a unique kind of inflammation in their airways, people with asthma are more sensitive than the general population to a broad variety of triggers, which in turn causes their airways to constrict excessively, resulting in decreased airflow, wheezing, and other symptoms. Although airway narrowing may often be reversed, some people with persistent asthma may have permanent airflow restriction [1, 2, 3].

In terms of chronic pediatric illness, asthma is at the top of the list. A child's hospital admission is often labeled as being due to asthma. Hyperactivity of the airways in response to a wide range of stimuli; and a highly reversible obstructive process that may develop on its own or as a consequence of therapy suggest that asthma may be classified as a diffuse obstructive lung disease. At least 10% of males and 7%-10% of girls may develop asthma. Nearly twice as many boys as girls are afflicted before puberty, but both sexes are equally impacted thereafter. A family's mental health may be severely disrupted by asthma. But with the right care, symptoms may typically be managed to a reasonable degree. The asthma complex consists of wheezy bronchitis, viral-associated wheezing, and atopic-related asthma and is also known as reactive airways disease [3, 4]. Other than bronchoconstriction, inflammation is a crucial player in the pathophysiology of asthma. Variable degrees of involvement of both big (>2 mm) and tiny (2 mm) airways are possible. This hyper reactivity shows itself as bronchoconstriction after exercise, on exposure to strong smells or irritating gases like sulfur dioxide, cigarette smoke, or cold air, and on exposure to histamine or parasympathomimetic drugs like methacholine in a controlled laboratory setting (Mecholyl) [4, 5].

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

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Asthma patients often have abnormally high levels of blood creatine kinase, lactate dehydrogenase. Whether or whether these shifts are the actual reason for the sickness is unclear. The following modifications were identified as an essential strategy that aids doctors in monitoring, diagnosing, treating, and elucidating the illness pathophysiology in light of the rising worldwide incidence of asthma, the significant burden being imposed on patients, and the high expense of health care [6, 7].

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: Patients with severe asthma who were seen in the medical wards or as outpatients at Hospital were included in the research.

Exclusion criteria

1. Patients with a habitual use of tobacco products or alcohol were not allowed to participate in the research.
2. Patients who had a history of pulmonary infections, pneumonia, chronic bronchitis, emphysema, diabetes, renal insufficiency, or hepatic dysfunction were not allowed to participate in the trial.

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 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 creatine kinase in the serum. Spectrophotometric determination of lactate dehydrogenase in serum.

Result:

Table 1: Mean, SD and Significance of CK and LDH in Asthma patients and controls

Parameter control	SA			EA			Significance
	Mean	SD	Mean	SD	Mean	SD	
Creatine Kinase	125.10	25.65	115.53	18.75	281.50	20.64	$p<0.01$
Lactate dehydrogenase	309.93	42.48	289.40	53.35	480.98	18.41	$p<0.01$

Table 2: 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	125.10	25.656	84	182
Creatine Kinase	G2	40	115.53	18.759	87	162
	G3	40	281.50	20.641	250	324
	Total	120	174.04	79.425	84	324
	GI	40	309.93	42.483	210	386
Lactatedehydrogenase	G2	40	289.40	53.359	22	399
	G3	40	480.98	18.411	438	512
	Total	120	360.10	95.254	22	512

Table 3: ANOVA was used for testing the multiple comparison of significance difference between stable asthma groups (A) and with in exacerbating asthma groups (B).

Parameters	A	Sum of Squares	df	Mean Square	F	Sig.
		694671.217	2	347335.608	725.533	$p<0.01$
Creatine Kinase	B	56011.575	117	478.731		
	Total	750682.792	119			
	A	885071.450	2	442535.725	266.005	$p<0.01$
Lactatedehydrogenase	B	194645.350	l 17	1663.635		
	Total	10797 I 6.800	ll9			

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Table 4: Post hoc analysis (multiple comparison tests) done between control (Gl) stable asthma (G2) and exacerbating asthma (G3) groups

Parameters	Pair wise Comparisons				
	(I) Group	(J) Group	Mean Difference(I-J)	Std. Error	Sig.
	GI	G2	9.575	4.893	$p>0.05$
Creatine Kinase	GI	G3	-156.400	4.893	$p<0.01$
	G2	G3	-165.975*	4.893	$P<0.01$
	GI	G2	20.525	9.120	$p<0.05$
Lactatedehydrogenase	Gl	G3	-171.050	9.120	$p<0.01$
	G2	G3	-191.575	9.120	$P<0.01$
	GI	G2	5.300	3.218	$p>0.05$

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

Parameter	Control	Case 1		Control	Case 2	
		SA(A)	EA(B)		SA(A)	EA(B)
CK	125.10	115.53	281.50	25.65	18.75	20.64

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

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

Parameter	Control	Case1		Control	Case2	
		SA(A)	EA(B)		SA(A)	EA(B)
LDH	309.93	289.40	480.98	42.48	53.35	18.41

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

Discussion

Disease of the airways characterized by persistent inflammation; asthma. 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. House dust mites (often found in pillows, mattresses, upholstered furniture, carpets, and draperies), cockroaches, cat dander, and seasonal pollens are also common allergies [7-10].

Goblet cell hyperplasia and inflammatory cell infiltration (namely eosinophils, neutrophils, and lymphocytes, particularly T lymphocytes) are histopathological hallmarks. Hypertrophy of bronchial smooth muscle and airway epithelium; collagen deposition underneath the basement membrane; occasional clogging of small airways with thick mucus. Chronicity of the condition may be traced back to airway inflammation, which in turn leads to airway hyper-responsiveness, airflow restriction, and respiratory symptoms including wheezing, shortness of breath, chest tightness, and cough [11-13].

Asthma's defining physiological anomaly is airway hyperresponsiveness (AHR), which is characterized by an exaggerated bronchoconstrictor response to a combination of inhaled irritants that normally would not cause a reaction. Reducing AHR is a major goal of treatment since it is correlated with the severity of asthma attacks. Direct bronchoconstrictors like histamine and methacholine compress airway smooth muscle, causing an increase in bronchoconstrictor reactivity. Many indirect stimuli, such as those that cause mast cells to produce bronchoconstrictors or those that stimulate sensory neurons, also exhibit this pattern. The case of a patient who went into status asthmaticus and afterwards had rhabdomyolysis and severe renal failure has been described by Chugh and colleagues. Since creatine kinase is the most sensitive enzyme marker of muscle damage, they hypothesized that the increased respiratory effort associated with acute asthma and hypoxemia was responsible for the increase in muscle-related enzyme levels [14, 15].

The current research found that the mean value of serum Lactate dehydrogenase was significantly higher in patients with asthma exacerbation (480.98$\pm$18.41) compared to the control group (309.93$\pm$42.48). This difference was statistically significant ($p<0.01$). Stable asthma patients (289.40$\pm$53.3) were compared to a control group, and no statistically significant differences were found. According to the results of the current investigation, individuals with asthma whose symptoms are worsening have a greater blood Lactate dehydrogenase content than patients whose symptoms are constant [16, 17].

Mucosa infiltration by inflammatory cells such mast cells, eosinophils, and neutrophils is more strongly linked to these results. airway hyperresponsiveness and mucous secretion cause structural alterations and bronchospasm caused by lymphocytes, macrophages, and inflammatory mediators such histamine, leucotriene, cytokines, and chemokines. The cellular and biochemical mediators thought to be possible biological indicators of lung damage have attracted attention as part of the pathogenic process of lung

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injury. Although lactate dehydrogenase (LDH) and other cytoplasmic enzymes serve no metabolic purpose in the extracellular environment, they may be useful as indications of cellular integrity disruption caused by pathogenic circumstances. When a cell lyses, enzymes from the cytoplasm, such as lactate dehydrogenase (LDH), are released into the surrounding medium. Therefore, LDH detection is based on its presence in the extracellular space after cell damage or death has occurred [18].

Conclusion

Inflammation of the airways persists in those who suffer from asthma. This illness is complex, displaying a wide range of manifestations, and it is usually induced by a number of interactions between genes and the environment. Limitation of airflow in asthmatics is caused by bronchoconstriction, but also by airway edema, vascular congestion, and luminal blockage with exudate. This heightened sensitivity to environmental stimuli is related to the unique form of inflammation that develops in asthmatics' airways. Atopy, indoor allergens, outdoor allergens, airway hyper responsiveness, occupational sensitizers, passive smoking, gender, obesity, ethnicity, respiratory tract virus infections, exercise, and hyperventilation are just few of the many factors that might set off an asthma attack. Allergens that are inhaled are a typical cause of asthma attacks in those who are allergic sensitized. Childhood exposure to home dust mites increases the likelihood of developing asthma and other allergic reactions.

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 blood creatine kinase, serum lactate dehydrogenase, in aggravating and stable asthma patients which is found to be significant.

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