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Correlation of IgE, sputum and peripheral Epinophilia in Bronchial Asthma in Tamil Nadu Population - Retrospective Study

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Abstract

Background: Bronchial Asthma most common chronic, non-communicable diseases in adults due to involvement of various cellular populations and important cause of morbidity and mortality in India and abroad. Due to wide variations among different asthmatics there is a need for a specific bio-marker to diagnose and prognoses the asthma conditions.

Method: 80 (eighty) Bronchial asthma patients aged between 35 to 65 were studied and compared with healthy voluntary of same number. Pulmonary function test, Blood examination for peripheral Eosinophlia, IgE and Sputum for Eosinophia chest X-ray was performed to confirm and diagnose.

Results: History of smokers, allergic rhinitis and family history were higher in Bronchial asthma patients. The parameters like IgE, peripheral Eosinophilia %,

sputum Eosinophilia, FVC% predicted FEV_1, FEV_1/FVC predicted, post FVC/post FEV_1 predicted had significant P value (P<0.001) when compared with controlled group.

Conclusion: This correlative study of peripheral Eosinophilia, Sputum Eosinophilia, IgE, and FEV_1 will be the tolls for the clinician to predict the prognosis and treat efficiently to avoid morbidity and mortality.

Keywords: IgE, Sputum Eosinophilia, FEV_1/FVC, Spirometry, PBS

Introduction

It is well established that one of the main features of Asthma is the presence of air-way inflammation with involvement of several cellular populations and the release of many inflammatory mediators [(1)]. Eosinophils in particular with the release of cytotoxic proteins such as eosinophil cationic protein (ECP), are good marker of airway inflammation in Asthma. In the last few years induced sputum has been increasingly adopted at this purpose as a non-invasive safe and reproducible method [(2)]. An extensive eosinophilic inflammation of the bronchi has been described as bronchial asthma even with mild clinical or sub-.clinical disease [(3)]. If an allergic trigger is suspected, an allergy diagnosis consisting of medical history and/or definition of the immunoglobulin E (IgE) should be performed [(4)]. IgE is a type of antibody produced by plasma cells located in lymph nodes draining the site of antigen entry or locally, at the sites of allergic reactions by plasma cells derived from germinal centres developing within the inflamed tissue IgE is pathogenic in allergic diseases such as asthma allergic rhinitis, atopic dermatitis and food allergy [(5)]. Hence IgE sputum and peripheral eosinophilia are correlated to evaluate the severity of the diseases in adults.

Material and Method

80 Asthmatic patients aged between 20-75 years regularly visiting to SRM Medical college Hospital and research centre Trichy Chennai High way, Irungular Village Tiruchirapalli – 621105, Tamil Nadu.

Inclusion Criteria: The patients having the clinical symptoms of bronchial Asthma were selected for study.

Exclusion Criteria: The patients below 20 years, having cardio vascular. Diabetic mellitus, immune compromised patients were excluded from study.

Method: Same number 80 (Eighty) healthy adults volunteers were also compared with these bronchial asthma patients. Each patients undergone pulmonary function test by spirometry – patients were subjected to PFT which flow sensing MIR spirobank II and were assessed for post broncho dilator reversibility after administrating 200 Mg of inhaled salbutamol by repeating the test after 15 minutes from the base line. The degree of reversibility to forced expiratory volume 1s, (FEV_1) of 12% and 200 ml from the pre bronchodilator value was considered for diagnosis of asthma as per Global Initiative for Asthma (GINA) guide lines but out study observed severe bronchial asthma only. Blood examination included peripheral Eosinophil count, serum IgE levels, and sputum samples for count. For eosinophilia. The sputum was homogenised by adding phosphate buffered saline (PBS), vortexed for 30's and centrifuged for 10 minutes, then 0.1% dithiothreitol to the cells in ratio of 4:1, which was agitated for 20 minutes to break up the disulfide bounds and disperse the cells. Cells are washed once again with PBS and re-suspended. The cell suspension was aspirated and filtered to remove the remaining debris; supernatant was separated from cell pellate, sputum sample was transferred to the slide and distributed thinly and evenly over the slide staining was done by haematoxylin and Eosin stain and analysed using microscope to count for eosinophillis. The sputum eosinophilic count≥3% was considered as normal. Chesty X-ray was taken to confirm the bronchial Asthma.

The duration of study was Meach-2015 to December-2016.

Statistical analysis: Variations in the clinical manifestations in Bronchial asthma were classified with percentage; various parameters of bronchial asthma were compared with controlled group with t test. The statistical analysis was performed in SPSS software. The ratio of male and female was 2:1

Observation and Results

Table-2: Clinical parameters are compared in controlled and bronchial asthma patients-

1) Serum IgE (IU/ml) in normal (controlled group) was 720.2 (± 210) in asthma brachial asthma patients, 1130.24 (±105) t test value was 5.76 p<0.001.
2) Peripheral Eosinophilia: 7.3 (± 3.20) in controlled, 9.04 (± 4.30) in Asthma group t test: 2.90 p<0.001.
3) Sputum Eosinophilia: 3.8 (± 2.80) in controlled, 4.58 (± 7.58) in Asthma group t test -0.85 p<0.001.
4) FVC % predicted: 8.7 (± 11.3) in controlled 60.6 (± 9.5) in Asthma group t test 15.9 p<0.001.
5) FEV_1 % Predicted: 78.6 (± 0.85) in controlled 48.8 (± 5.88) in Asthma group t test 44.8 p<0.000
6) FEV_1 / FVC % predicted: 89.3 (± 9.24) in controlled, 82.6 (± 11.1) t test 4.15 p<0.001
7) Post FVC% predicted: 95.2 (± 7.30) in controlled, 69.09 (± 10.92) in asthma group t test 17.8 p<0.001
8) Post FEV_1 % predicted: 92.2 (± 0.92) in controlled group, 64.1 (± 9.1) in asthma bronchitis t test 27.4 p<0.001
9) Post FEV1 / FVC% predicted: 95.15 (± 6.64) in controlled group, 87.8 (±20.1) in Asthma group t test was 3.06 p<0.001

Table-1: Clinical Manifestations History of Smokers 12 (15%) in controlled and 54 (67%) in Asthma patients.

Allergic rhinitis 11 (13.7%) in controlled group 29 (36.2%) in Asthma. 14 (17.5 %) Bronchial asthma was observed in family history of patients

Discussion

Present correlation of IgE, sputum and peripheral Eosinophillia in Bronchial asthma patients in Tamil Nadu. The clinical manifestations were 12 (15%) in controlled and 54 (67%) asthma patients had history of patients. Allergic rhinitis was 11 (13.7%) in controlled, 29 (36.2%) in asthma patients, 4 (17.5%) Asthma patients had family history of Bronchial asthma (Table-1). In the comparison of various clinical parameters serum IgE 720.25 (± 210) in controlled 1130.24 (± 110) in Asthma patients t test was 5.75 and $p<0.001$ (p value was highly significant). Peripheral Eosinophilia % in controlled was 7.3 (± 3.20), 9.04 (±4.30) in asthmatic patients t test – 2.90 $p<0.001$. Sputum Eosinophilia 3.8 (±2.80) in controlled 4.58 (±7.68) in Asthma patients t test – 0.85 $p<0.001$ FVC % predicted 87 (± 11.3) in controlled, 60.6 (± 9.58) in Asthma patients + 15.9 $p<0.001$ FEV_1 % predicted 78.6 (± 0.85) in controlled 48.8 (± 5.88) in Asthma patients t test 44.8 $p<0.001$ FEV_1/FVC% predicted 89.3 (± 9.2) in controlled, 82.6 (± 11.1) in Asthma patients t test 4.15, $p<0.000$

Post FVC% predicted 95.2 (± 7.30) in controlled, 69.09 (± 10.9) in asthma patients t test 17.8 $p<0.0001$, post FEV_1 % predicted 92.2 (± 0.92) in controlled, 64.1 (± 9.1) in Asthma patients t test 27.4 $p<0.001$, post FEV_1/FVC predicted 95.1 (± 6.64) in controlled, 87.8 (± 20.1) in asthmatics t test 3.06 and p value was highly significant ($p<0.001$) (Table-2). These findings are more or less in agreement with previous studies [6][7][8].

It is reported that, Asthma is of two types – extrinsic and intrinsic types. Atopic asthma is a sub type of extrinsic asthma in which patient have hyper – responsive airway. This stimulates induction of TH_2 type T cells which release cytokines like IL-4 and IL-5. The released cytokines in turn promote IgE production by B cells, growth of most cells (IL-4) and growth and activation of eosinophils subsequent IgE mediator reaction to inhaled allergens elicits acute and late phase reaction [9]. It is hypothesized that, the levels of IgE are quite high locally at the site of inflammation and the serum levels don't necessarily reflect the levels in lungs and bronchus. It is also known that, IgE is bound to mast cells with high affinity [10]. Increase in IgE may be due to viral infections which is the commonest cause of

exacerbation of symptoms of Asthma. The virus is Epstein Barr virus (EBV), cytomegalo virus (CMV), Measles virus and Rhinovirus.

Eosinophils play an important role in the pathogenesis of asthma. Airway inflammation, involving infiltration of bronchial wall by activated eosinophils, most cells and T lymphocytes is an established future of asthma. These is consideration evidence that, eosinophils also play an important role in bronchial epithelial damage proteins and serum total IgE levels sputum eosinophilia has been shown to be the best, predictor of short term response to corticosteroids. The result confirmed that, a high percentage of eosinophils in blood or sputum as a known marker of airway inflammation Eosinophilic cationic protein (ECP) shown to be significantly higher during acute asthma exacerbations.

Summary and Conclusion

IgE, sputum eosinophils, peripheral Eosinophils are significantly higher in severity of bronchial asthma and act as biomarker to diagnose and treat them bronchial asthma but this study further demands genetic, cytological, patho-physiological, nutritional, pharmacological studies because cytological mechanism which cause bronchial asthma and exact pathogenesis of bronchial asthma is still unclear.

Table-1

Clinical Manifestations in Bronchial asthma patients

Clinical Manifestation	Controlled group 80		Bronchial Asthma patients 80	
	Number	Percentage	Number	Percentage
History of smokers	12	15%	54	67.5%
Allergic rhinitis	11	18.7%	29	36.2%
Family history of Bronchial Asthma	--	--	14	17.5%

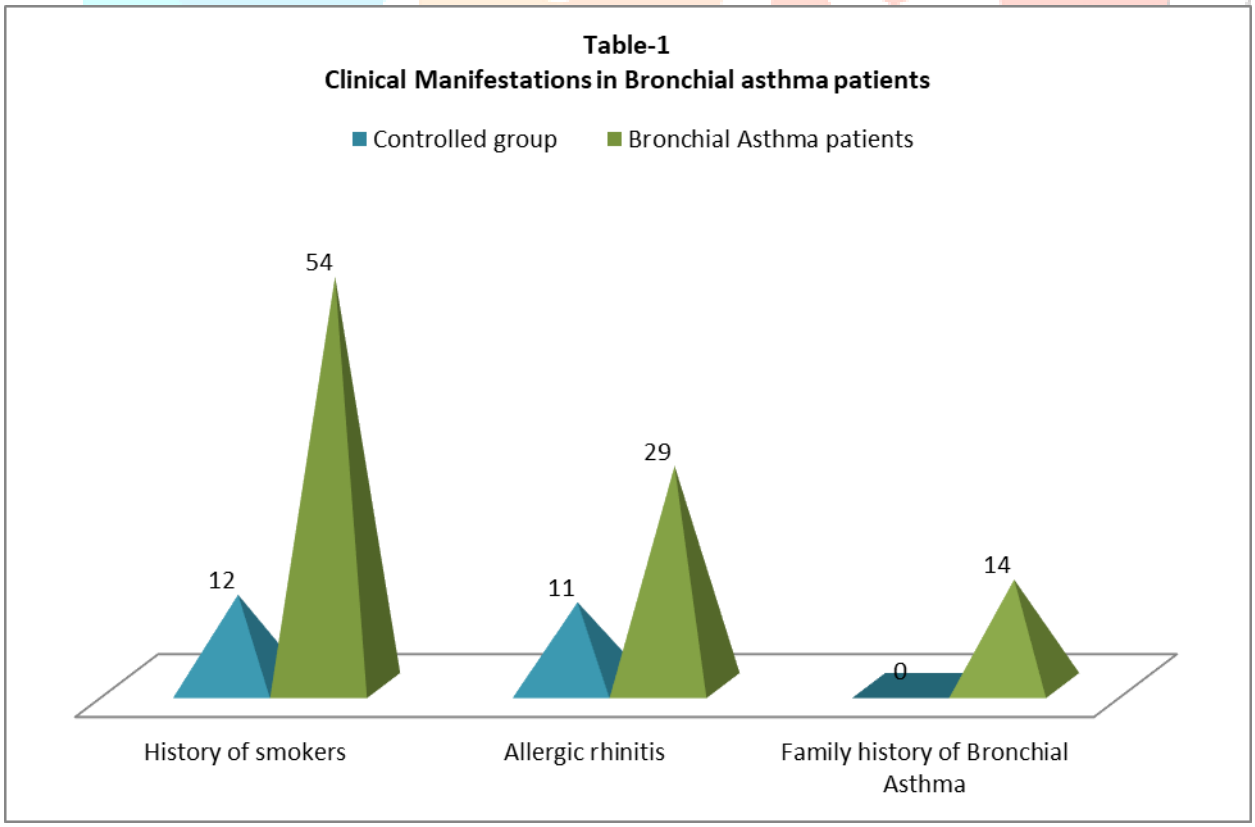


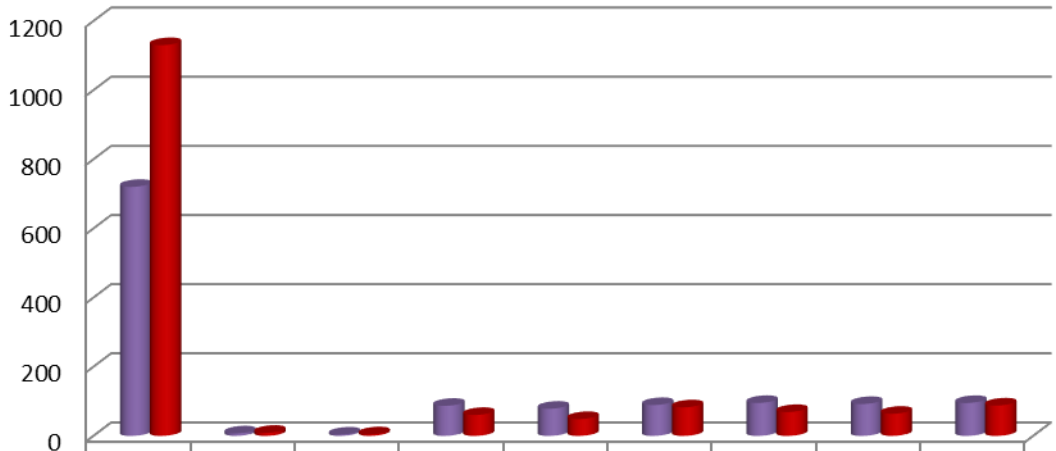
Table – 2

Comparison various parameters severe bronchial Asthma

(80 Controlled, 80- Bronchial Asthma)

Sl No	Clinical Parameters	Normal Controlled 80	Bronchial Asthma 80	t test	p value
1	serum IgE (IU/ml)	720.25 (± 210)	1130.24 (± 101)	5.76	P<001
2	PeripheralEosinophilia %	7.3 (± 3.20)	9.04 (± 4.30)	-2.90	P<0.001
3	Sputum Eosinophilia	3.8 (± 2.80)	4.58 (± 7.68)	-0.85	p<0.001
4	FVC% Predicted	87 (± 11.3)	60.6 (± 9.50)	15.9	P<0.001
5	FEV_1% Predicted	78.6 (± 0.85)	48.8 (± 5.88)	44.8	P<0.000
6	FEV_1/ Predicted	89.3 (± 9.20)	82.6 (± 11.10)	4.15	p<0.000
7	Post FVC% Predicted	95.23 (±7.30)	69.09 (± 10.92)	17.8	P<0.001
8	Post FEV_1% Predicted	92.2 (± 0.92)	64.1 (± 9.1)	27.4	p<0.01
9	Post FEV_1 / FVC% Predicted	95.15 (± 6.64)	87.89 (± 20.1)	3.06	p<0.001

Table – 2
Comparison various parameters severe bronchial Asthma
(80 Controlled, 80- Bronchial)



	serum IgE (IU/ml)	Peripheral Esinophilia %	Sputum Esinophilia	FVC% Predicted	FEV1% Predicted	FEV1/ Predicted	Post FVC% Predicted	Post FEV1% Predicted	Post FEV1 / FVC% Predicted
Normal Controlled	720.25	7.3	3.8	87	78.6	89.3	95.23	92.2	95.15
Bronchial Asthma	1130.24	9.04	4.58	60.6	48.8	82.6	69.09	64.1	87.89

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