Study Specific Immunoglobulin E,G antibodies and Bacterial which Induced Asthmatics

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Abstract:

Background: The study determine if infection with Bacteria triggers the production of pathogen-specific IgE ana IgG in asthma Patients with chronic respiratory diseases which might contribute to inflammation and pathology **Objective**: This study aims at evaluating the parameters immunity including and IgE and IgG antibodies **Methods**: This work was applied on 87 asthma patients admitted to the Babylon center of asthma and 40 apparently health controls with age range (9-60 years). Tonsils Swabs cultures of asthma patients reveals major types of bacteria *,St.pyogens, S.aureus, M. catarrhalis ,Ps.aeruginosa, N.menengenitites H.influenzae*

, S.epidermidis . aureus form high rate of these isolates . S **Results**: The Immunological parameters showing that there is a significantly increased (p < 0.05) in IgE(448.75)) IU/ml compared to control group (50.11) and a significantly increased (p < 0.05) in IgG(56.41) IU/ml compared to control group (29.33). a significantly increased (p < 0.05). **Conclusion**: IgE antibodies play a central role in allergic inflammation; therefore production of bacteria may prove significant in the exacerbation of chronic, allergic airway diseases, thus highlighting a direct role in asthma pathogenesis.

Keywords: Asthma ,IgE Elisa, IgG Elisa, Bacterial isolate

Introduction

Asthma is characterized by acute episodes of airway obstruction precipitated by respiratory infection and the release of IgE dependent mediators ,airway inflammation resulting from an inappropriate response to either infectious or allergic antigens is a finding common to the different manifestations of asthma. (Murphy *etal*.2010)

Airway secretion on the respiratory mucosa is an important part of mucosal immunity(Kay,2009). The presenceofimmunoglobulin (Ig)E and IgG and antibodies in airway secretion has been shown (Brandtzaeg ,1995). In Airway secretion IgA (60-70%) and IgG (30-40%) antibodies account for 99% of total of immunoglobulin (Burnett ,1986). The majority (70-90%) of total IgG is secretory IgG which is locally produced in the respiratory mucosa and majority of IgG antibodies in airway secretion are passively leaked through the surface epithelium from the systemic circulation(Hollams *etal* .2010;Peebles *etal* .2011).

Even less is knownabout the relationship between asthma and bacteria. Recent studies confirm that bacterial respiratory infectionsare frequently associated with increased airway obstructionin patients with bronchial asthma (Bousquet *etal* .2000). While the hygienehypothesis predicts that infections in early life bynon-pathogenic microbes should protect against asthmaand atopy (Ramsey and Celedon ,2005), there is increasing evidence that certainchronic pathogenic infections might also promote airwayhyperresponsiveness and asthma development or exacerbation(Bisgaard *etal*.2007).

Methods

Patients and samples

Bacteriological study includes culturing of Tonsils Swabs with selective and differential media. Biochemical investigations were done for bacterial identification(McFadden, 2000).

A total of 87 asthma patients consisting of 40 health controls were involved in this study. Their age range was from (9-60) years. Case information was taken for each patient include; name, sex, age, residency, duration of infection, and duration of therapy. All asthma cases were clinically diagnosed by a specialist clinician. Those patients were admitted to the Babylon center of asthma .Atotal of 40 apparently healthy subjects were involved as controls group. The age range of controls was matched to the patients (10-60) years.

Three ml of blood were collected by vein puncture into two sterile test tubes , in one of them 2 ml of blood were put and left for (2-4) hours, then the upper layer (serum) was collected in clean test tube and stored at-20 C^o until using it in serological tests and determination of IgEand IgG (Kadooka *et al.*, 2000).

In vitro test which used enzyme –linked immunosorbent technology which measures, bacteria antigens number or enzyme- linked immunosorbent assay (ELISA) technology (Quanti FERON) IgE,IgG production in whole blood (Lazarevic *et al.*, 2005).

Statistical Analysis

T-test (p <0.05) were carried out according to Niazi (2004).

Results and Discussion

Bacteriological study

Bacteriological study of Tonsils Swabs cultures of asthma patients reveals many bacterial isolates, this study concerned with many types of bacteria *M. catarrhalis and S.aureus*. Numbers of bacterial isolates varies with type of specimens and virulence efficacy efficacy, *S.aureus* forms highest rate of these isolates 19.5 % while *M. catarrhalis* form 18.3 % for specimens (table 1). Members of bacterial including *M. catarrhalis* 18.3%, *S.aureus* 19.5%, *Ps.aeruginosa* 8.9%, *St.pyogens* 15.8%, *S. macescence* 0.5%, *K. pneumonia* 7.6%, *S.epidermidis* 12.7% and *St.virdanse* 1.1% genera characterized by their highly ability to cause enteric infection in human and the symptoms of infection appears with certain days as a results of their toxins activity (Armann *et al.*,2010).

Bacterial types	No. of isolates %
St.pyogens	15.8
S.aureus	19.5
M. catarrhalis	18.3
Ps.aeruginosa,	8.9
S. macescence	0.5
<i>H.influenzae</i>	6.1
K. pneumonia	7.6
N.menengenitites	9.5
S.epidermidis	12.7
St.virdanse	1.1
Totale	100

Table 1. Bacterial isolates from Asthma infected

Distribution Asthma with Age, Sex and Geographical

The number increased in the age group of > 31-40 to reach 18 % of the total number of asthma patients. The number of cases were maintained in most at the same level in the age groups of <10 years (22%) and 11-20 years (20.6%).

The results that were expressed in table (2) revealed the wide age range for asthma patients. The result in table (1) reveals that the most predominant age groups (51-60) years of asthma patients. This finding was matched with (Huang *et al.*, 2011) that reported asthma is mainly a disease of older people or of the immunocompromised .The result was matched with that recorded by (Fahy *et al.*, 1995), who mentioned that the aging process has significant and deleterious effects on immune responses in human, resulting in increased susceptibility to bacterial infection because compromised functioning of innate immune responses, at least as much as reduced adaptive responses.

Age group (years)	asthma patients
<10	20: 87 (22 %
11-20	18: 87 (20.6%)
21-30	12: 87 (13 %)
31-40	16: 87 (18 %)
41-50	3:87(3.4%)
51-60	12: 87 (13 %)
>60	6: 87 (6.8 %)
Age range (years)	9-60

Table (2) Age Distribution for asthma Patients

In this study, the asthma patients consisted of 52:87 (59.7%) males and 25:87 (28.7%) females, figure (1). For patients showed that male–female ratio was higher in males than in females. This finding was matched with that recorded by (Lowe *et al.*, 2011)who mentioned that the rateofasthma in male was higher than female for asthma patients attending the Babylon center of asthma. This difference is partly due to the fact that man have less access to diagnostic facilities in some settings, but the broader pattern also reflects real epidemiological differences between men and women, both in exposure to infection and in susceptibility to disease. (Kim *et al.*, 2011).

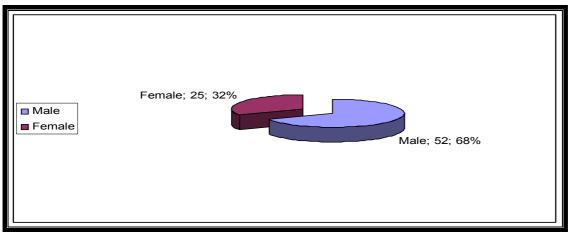


Figure (1) Sex Distribution for asthma Patients

The geographical distribution of 87 asthma patients included in this study is illustrated in figure (2). asthma patients were classified according to the home address into rural52: 87(68%). While the patients' habitat in urbanite 25:87 (32%). For asthma patients, showed that rural-urban ratio was higher in rural than in urban. This finding was matched with that recorded by (Rosenwasser,2011)whomentionedthat tuberculosis cases were commonly found in rural areas 59%.

The higher incidence of asthma among rural could be attributed to non-medical factors such as poor quality of life, poor housing, and overcrowding, population explosion, under nutrition, lack of education, large families, early marriage, lack of a wareness of causes of illness, etc. All these factors interrelated and contributed in the occurrence and spread of asthma. Other studies found that asthma cases finding rate in urban 52% and 28% in rural (Soderstrom, *et al.*,2011).

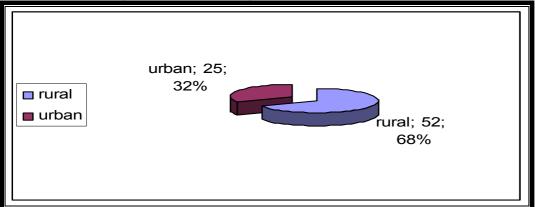


Figure (2) Geographical Distribution for Asthma Patients

Concentration of IgE and IgG in serum Asthma Patients.

In the present study, it was observed that IgE levels in population were higher than control. The allergic patients have an expected IgE concentration up to348.75 IU/ml while in control IgE levels concentration is appears 50 IU/ml significantly p<0.005) (Table 1). Immunoglobulins concentration raised in tissue fluids as a result of an inflammatory agents or other inducing agents , immunoglobulins represent one of specific immune response that elicitand induce B cells proliferations and antibody production, The higher IgE levels in allergic is explained probably by the higher incidence of parasitic infestations and allergic complication (Stokes and Casale, 2004). The biological activities of IgE in allergic patients, has been reported by Rondon and his colleagues (Huang., et al.2011) The biological roleof IgE is inducing humoral and cellular immune responses, increased levels of IgE and inflammatory cells, are related with Th1 andTh2 responses in patients with allergic. (Platts, 1979)

Age group (years)	Group	IgE IU/ml
<10	Patient	33.10±*448.75
	Control	9.09±50.11
11-20	Patient	* 227.42 18.9±
	Control	9.85±78.79
21-30	Patient	27.15±*431.14
	Control	3.56±83.68
31-40	Patient	28.9±*228.58
	Control	1.73±58.48
41-50	Patient	37.18 ±*346.19
	Control	3.50±75.00
51-60	Patient	25.61±*221.52
	Control	3.008± 74.48
>60	Patient	29.18±*344.5
	Control	2.72±87.20

Table (1) Concentration of IgE level IU/ml for asthma Patients andControls

*Standard deviation

In the present study, it was observed that IgG levels in population were higher than control. The allergic patients have an expected IgG concentration up 163.105 whil mg/dl e in control IgG levels concentration is appears 26.3216 mg/dl significantly p<0.005). In this study, the latter method was used because selection of amucus plug provides more viscous samples as well assmaler amounts of specimen for fluid-phase analysis. Thelevels of total IgG and IgG antibodies in induced sputum samples from asthmaticpatients were significantly higher than those in salivary samples.

A significant correlation between sputum eosinophiliaand B-cell counts in asthmatic patients has been reported(Kidney *et al.1996*)

Age group (years)	Group	IgG mg/dl
<10	Patient	163.105±48.4994
	Control	26.32167±21.8478
11-20	Patient	109.305±21.8478
	Control	26.485±16.4696
21-30	Patient	107.9275±26.5996
	Control	42.19±13.6708
31-40	Patient	139.2975±55.8847
	Control	32.465±10.3773
41-50	Patient	136.1683±-45.7561
	Control	26.78833±15.0779
51-60	Patient	120.0825±33.8127
	Control	32.62833±15.8318
>60	Patient	150.664±16.765
	Control	37.654±4.675

Table (2) Concentration of IgG IU/ml for asthma Patients and Controls

*Standard deviation

Conclusions

The role and regulation of IgE and IgG antibodies towards microbial antigens is far from beingelucidated, results from the current study strongly suggest that this obligate intracellular pathogen can induce pathogen-specific IgE production and could thereforelead to mast cell degranulation and release of vasoactive agents. Theorganisms play a direct role in asthma pathogenesis by continuous induction of IgE and IgG, since unlike most aeroallergens that a patient canavoid, thE organisms reside in the lower airways and are continuously secreting bacterial antigens.

References

1- Kay AB. (2009). Asthma and inflammation .J.Allergy .Clin.Immunol.87:893-910.

2-Brandtzaeg P. (1995) The role of humoral mucosal immunity in the induction and maintenance of chronic airway infection .Am.J.Respir. Crit. Care .Med . 151 : 2081-2087.

3- Huang ,Y. ; Nelson, C. and Brodie, E. (2011) Airway microbiota and bronchial hyperresponsiveness in pationts with suboptimally controlled asthma . J. Allergy Clin. Immunol .127(2): 372-381

4- Hollams EM, Hales BJ, Bachert C, Huvenne W, Parsons F, de Klerk NH,

Serralha M, Holt BJ, Ahlstedt S, Thomas WR, et al: Th2-associated immunity

to bacteria in teenagers and susceptibility to asthma. Eur Respir J 2010,

36:509-516.

5-Peebles R., Liu M., Lichtenstein L. and Hammilton R.(2011) IgG, IgM Quantification in bronchoalveolar lavage fluids from allergic asthmatics and normal subjects by antibodt- based immunoenzymetric assays J.Immunol. 179:77-86.

6- Macfaddin, J.F 2000. Biochemical tests for identification of medical bacteria . 3rd ed. Awolters Kluwer Company .

7-Murphy DM, O'Byrne PM: Recent advances in the pathophysiology of

asthma. CHEST 2010, 137:1417-1426.

8- Lazarevic, V. ; Pawar, S. and Flynn, J. (2005). Measuring T-cell function in animal models of asthmatic by Elispot Methods.Mol. Biol. 302: 179-190.

9- Armann J, von Mutius E: Do bacteria have a role in asthma development?

Eur Respir J 2010, 36:469-471.

10 - Bousquet J, Jeffery PK, Busse WW, Johnson M, Vignola AM: Asthma. From

bronchoconstriction to airways inflammation and remodeling. American

Journal of Respiratory and Critical Care Medicine 2000, 161:1720-1745.

11-Huang ,Y.; Nelson, C. and Brodie, E. (2011) Airway microbiota and bronchial hyperresponsiveness in pationts with suboptimally controlled asthma . J. Allergy Clin. Immunol .127(2): 372-381 .

12- Kim, D.; Sato, A.; Fukuyama, S. and Sagara, H.(2011) The airway antigen sampling system : Respiratory m cells as an alternative gaetway for inhaled antigens . Lippincott Williams Wilkins, London, PP. 87 - 104.

13- Lowe , P. and Renard, D. (2011) Omalizumab decreases IgE production in patients with allergic IgE mediated asthma . J. Clin. Pharmacol .10 : 1365-2125.

14- Rosenwasser, L.(2011) Mechanisms of IgE Inflammation .J.Med. Microbiol . 11(2) : 178-830.

15- Soderstrom, L. ; Lilja, G. ; Borres, M. and Nilsson , C. (2011) An explorative study of low levels of allergen specific IgE and clinical allergy symptoms during early childhood . J. Allergy . 10 : 1398-9995.

16- Ramsey CD, Celedon JC: The hygiene hypothesis and asthma. Curr Opin

Pulm Med 2005, 11:14-20.

17- Nahm , D. ; Kim , H. and Park , H. (1998) Elevation of specific immunoglobulin A antibodies to both allergen and bacterial both antigen in induced sputum from asthmatics .J. Respir . 12 : 540-549.

18- Bisgaard H, Hermansen MN, Buchvald F, Loland L, Halkjaer LB, Bonnelykke K, Brasholt M, Heltberg A, Vissing NH, Thorsen SV, et al: Childhood asthma

after bacterial colonization of the airway in neonates. N Engl J Med 2007,

357:1487-1495.

19- Kidney JC, Wong AG, Efthimiadis A, et al. Elevated Bcells in sputum of asthmatics: close correlation witheosinophils. Am J Respir Crit Care Med 1996; 153: 540–544.

20- Fahy J.; Wong H., Liu J., Boushey H., (1995). Comparison of samples collected by sputum induction and bronchoscopy from asthmatic .Am.J. Respir. Crit. Care.Med.152:53-58.

21- Platts T.(1979) Local production of IgE and IgG antibodies in asthmatic Pation .J.Immunol, 22: 2218-2225.

22-Bråbäck L, Forsberg B. (2009). Does traffic exhaust contribute to the development of asthma and allergic sensitization in children: findings from recent cohort studies. *Environ Health*. ;8:17

23- Kadooka Y, Idota T, Gunji H, Shimatani M, Kawakami H, Dosako S, Samori T:

A method for measuring specific IgE in sera by direct ELISA without

interference by IgG competition or IgG autoantibodies to IgE. Int ArchAllergy Immunol 2000, 122:264–269.

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