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Serum Prolactin Level in Individuals Occupationally Exposed to Organic Dust

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Abstract

In the present work eighty wheat workers and twenty unexposed volunteers were studied. Clinical examination together with measuring the level of serum IgE, IgG, and prolactin were done. This had demonstrated that exposed workers may get sensitized to antigens in the wheat dust and give rise to allergic manifestations including respiratory problems. It was found that the level of serum IgE & prolactin were significantly increased in the exposed group as a whole when compared with the controls ($P < 0.01$ & < 0.001). The atopic exposed individuals showed a significantly increased serum prolactin and IgE as as well when compared with the non atopic workers ($P = 0.05$ & < 0.001). There was also a significant positive correlation between the serum prolactin level and the IgE level in the whole exposed group but a weak +ve correlation in the atopic sub-group ($r = 0.368$ & $r = 0.223$). In the asthmatic sub-group there was a —ve insignificant correlation between the prolactin and IgE levels ($r = 0.178$). This proves that exposure more than the atopic trait is behind the increased prolactin level. The mechanism of increased prolactin level is that the IgE-mast cell interaction leads to histamine release which is a potent prolactin stimulant. The lack of correlation between the level of prolactin and IgE in asthmatics was explained.

Introduction

IN his environment man is exposed to many antigens whether by contact, ingestion or inhalation. Some susceptible persons react in an abnormal way when exposed to an offending antigen This reaction manifests itself in a way depending on the organ affected [1].

Although workers with occupational sensitization may have symptoms of rhinitis and/or conjunctivitis, or even they may develop asthma, yet they may have no preceding predisposing factors, i.e. atopy or a positive family history [2]. Atopy is a dominantly inherited trait [3].

The vasoactive amine «histamine» released by the mast cells which are distributed in almost all organs [4], is a potent prolactin stimulant [5,6]. In a previous study, Nicola [7] showed how atopic

asthma in the young was accompanied by hormonal aberrations namely increased prolactin secretion. In fact, there are many evidences which show the intimate relation between the immune system and the neuro-endocrine axis as they mutually affect each other[8,9].

The aim of this work was to find the impact of occupational exposure on the hormonal system and whether it differs from those with atopic trait or not.

Subjects and Methods

Subjects :

This study consisted of two groups of male workers. A group of eighty subjects exposed to wheat dust in a wheat grinding mill at Giza Governorate. Their ages ranged between 22-59 years with mean 44.6 and duration of exposure ranged between 5-33 with mean 12. The second, a control group of twenty healthy male subjects, not exposed to any type of dust. Their ages ranged between 20-56 with mean 43.2 years. All examined workers were of the same socio-economic stratum and were comparable as regards age and smoking habits with no significant difference.

A clinical interview was done to all subjects using a simplified form of the standard Medical Research Council questionnaire on respiratory symptoms[10]. A general clinical examination was done with stress on the chest, heart, liver and skin.

Methodology :

I — Blood samples were withdrawn from all the subjects and stored at —20°C for the following laboratory tests :

1. *Serum Prolactin Level* [11] :

This was done by the radio-immunoassay technique, using Coats-A-Count Prolactin kits. Diagnostic Products Corporation-Los Angeles, Ca, USA.

2. *Serum IgE immunoglobulins* [12] :

The enzyme immunoassay method was adopted, using Behring Enzygnost Kits, IgE micro-Behringwerek AG, Marburg, Germany.

3. *Serum IgG immunogloblins* [13] :

The immunochemical reaction was followed, using the Behring Turbiquant kits of Behringwerke AG, Marburg, Germany.

II — Skin Prick Test (SPT) : This was done using whole grain wheat extract in concentration 1/1000. Histamine was used as a positive control.

Statistical evaluation [14] :

Differences between the mean values of the examined groups were tested using the Student t test for continuous variables. The correlation coefficient (r) was used to measure the linear relationship between two variables.

Results

All the examined subjects were compared as regards age and smoking habits. The exposed group was divided according to the clinical data (symptoms & signs) into 18 (22.5%) asthmatics, 22 (27.5%) chronic bronchitics, 12 (16.25%) with other allergic manifestations such as rhinitis, eye irritation and/or skin irritation, 28 (33.75%)

subjects were exposed but asymptomatic. 24 (30%) worker of the exposed group were atopic (positive SPT to wheat extract).

Among the control group 2 (10%) only had chronic bronchitis, and 1 (5%) had allergic rhinitis (Table 1).

Table 2 showed the levels of the prolactin in ng/ml, the IgE in Iu/ml. and the IgG in mg/dl, in the serum of both exposed and control groups. It is evident that the level of prolactin (although it is still in the normal range), the IgE, and the IgG are significantly higher in the exposed group as a whole when compared with the control ($P < 0.001$, < 0.01, > 0.01) respectively. The IgE was significantly higher in the atopic sub-group than in the non-atopic subjects of the exposed group ($P < 0.001$), the prolactin was just significant in the atopics ($P = 0.05$). Also, the IgE was significantly higher in the asthmatic sub-group when compared with the asymptomatic sub-group ($P < 0.05$).

The correlation coefficient (r) Table 3, showed a significant positive correlation between the level of prolactin and the level of IgE in the exposed group as a whole ($r = 0.368$, $P < 0.01$). This correlation was weak and insignificant in the atopic sub-group ($r = 0.223$) while in the astmatics it was negative and insignificant ($r = -0.0178$).

Table (1) : Clinical Groups Among Examined Population.

Group	Exposed		Control	
	No.	%	No.	%
Asthmatic	18	22.5	—	—
Chr. Bronch.	22	27.5	2	10
Other Allergy	12	16.25	1	5
Asymptomatic	28	33.75	17	85
Total	80		20	
Atopic	24	30	—	—

The correlation studies between serum prolactin level and the IgG level showed a weak positvie correlation in the whole exposed group ($r = 0.242$), and weak positive insignificant in the atopic subgroup ($r = 0.353$). The asthmatic subgroup showed a negative insignificant correlation ($r = 0.152$).

The correlation studies between the IgE & IgG levels showed a very weak insignificant but negative correlation in the different groups ($r = 0.0539$, -0.0264, -0.0333 & $P > 0.05$) respectively.

Table (2) : Level of Prolactin, IgE, and IgG in the Serum of Examined Groups.

	Exposed							Control
	Asthmatic n = 18	Chr.Bronch. n = 22	other allerg. n = 12	Asymptom. n = 28	Atopic n = 24	Non-Atopic n = 56	All group n = 80	n = 20
Prolactin (ng/ml)	7.83 ± 2.7	6.53 ± 2.5	7.16 ± 2.22	7.65 ± 2.5	8.27** ± 2.9	6.9 ± 2.7	7.31* ± 2.6	5.5 ± 0.75
IgE (Iu/ml)	730.5*** ± 480.2	428.2 ± 355	407.1 ± 315.4	418.9 ± 415.9	930.6** ± 428.4	358.4 ± 307.3	530.1* ± 435.8	106.5 ± 41
IgG (mg/dl)	2620.6 ± 909.6	2742.3 ± 907.6	2840 ± 1187.6	3172.5 ± 1173.1	2936.7 ± 1080.3	2838 ± 1066.4	2845* ± 1114	947 ± 118.7

* Student t is significant when compared with controls ($P < 0.001$, < 0.01).

** Student t test significant when compared with non-atopic ($P = 0.05$, < 0.001).

*** Student t test is significant when compared with asymptomatic ($P < 0.05$).

Table (3) : Correlation Coefficient Between Different Variables.

Variables	Exposed n = 80	Atopic n = 24	Asthmatic n = 18
IgE & Prolac.	0.368	0.223	— 0.178
	P < 0.01	P > 0.05	P > 0.05
IgG & Prolac.	0.242	0.353	— 0.152
	P < 0.05	P > 0.05	P > 0.05
IgE & IgG	0.0539	— 0.0264	— 0.0333
	P > 0.05	P > 0.05	P > 0.05

Discussion

For many years investigators have been studying the interrelation among the immune system, nervous system, the endocrine glands and psychologic behavior. There is evidence from clinical observations and animal studies that the immune responses are regulated and modulated in part by neuroendocrine influences. This interaction seems to be bidirectional[**15**].

In the present investigation the role of occupational sensitization by wheat dust on prolactin secretion was studied. Our results showed a significantly increased value of serum prolactin and IgE levels in the exposed group as a whole when compared with the controls ($P < 0.001$, < 0.01). Also there was a significant increase in serum prolactin and IgE levels in the atopic sub-group when compared with the non-atopic subjects ($P = 0.05$, < 0.001). In a previous study[**16**], the significant increase of IgE in subjects exposed to wheat dust was documented in about 38.75% of the exposed subjects by a positive precipitin test to wheat antigen, and about 30% had positive SPT to wheat extract. It is well known that due to occupational exposure workers get sensitized and may develop symptoms of rhinitis and/or conjunctivitis. They may even develop asthma, yet such individuals do not necessarily have a predisposing factor such as atopy or positive family history of atopic disease[**2**]. Atopy is a dominant inherited trait[**3,17**].

It seems that the high IgE in the whole group and atopic sub-group due to exposure, is behind the process of prolactin release in such subjects. The IgE binds to receptors on the mast cells and the offending antigen then cross links the bound IgE antibodies. This process leads to the release of mediators including histamine from the mast cell[**18,19**]. Histamine is a strong, potent prolactin stimulant. As mast cells are found every where in the human body the severity of this reaction depends on the dose of the antigen that gain access to the body[**19**].

The aforementioned view can explain the positive significant correlation between the level of prolactin and IgE in the exposed group as a whole. This means that

as the level of IgE increases the level of released histamine will increase leading to increase in the prolactin level. However, there was a weak positive and insignificant correlation in the atopics and a very weak negative and insignificant correlation in the asthmatics between the level of IgE & prolactin although the level of the IgE was significantly increased in both sub-groups.

This can be explained by the fact that, with repeated exposure to different concentrations & different antigens contained in the wheat dust (fungi, mites, rodent hair, pesticides ... etc)[**20**], and repeated infections will lead to the formation of what is called blocking antibodies. Thus the IgG antibody formed against that antigen may competitively inhibit the binding of that antigen with the IgE antibodies and thereby block the release of the vasoactive amines[**19**]. In fact this is the basis of hyposensitization therapy in allergic patients. Repeated injections by gradually increasing doses of the offending antigen over a prolonged period will induce a state of immunologic tolerance and the level of IgE antibodies gradually fall below the pretreatment level[**17**].

In this study the level of IgG in the asthmatic and the atopic sub-groups was high, even higher than its level in the whole group. In fact, there was a negative correlation between the level of IgE and IgG, although very weak and insignificant in both the atopic and asthmatic sub-groups. This goes with the aforementioned explanation. However our findings among the asthmatics is contrary to what was found by Nicola[**7**] in young asthmatics where atopy was the predominant mechanism[**3**]. Also he found a strong positive correlation between serum prolactin and the level of IgE in his young atopic subjects. In our asthmatic sub-group atopy was found only in 50%.

The insignificant level of the prolactin in our asthmatic sub-group can also be explained by the fact that we compared them with the asymptomatic sub-group which was also exposed to the same antigens and its IgE level was also very high. This condition leads to the stimulation of prolactin secretion in these workers as well.

As mentioned before the severity of histamine release depends on the dose of antigen received by the subjects. In occupational asthma, symptoms can be evoked at ambient levels of sensitizer which are below industrial standards[**2**]. Hence histamine release is minimal. It seems that the level of exposure more than the atopic predisposition is behind the increased hormone level in occupationally sensitized subjects.

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