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Skin Susceptibilty to Antibiotic Contact Allergy or Irritation?

Amal Hamadah Ali Al-Hadithi
Department of Clinical Biochemistry,
College of Pharmacy, Baghdad University, Baghdad –Iraq.

الخلاصة

تمت دراسة شدة الحساسية بصورة مناعية لعشرين شخص من اللذين يعملون في مصنع المضادات الحيوية والذين هم في تماس مباشر مع باودر المضادات الحيوية (امبسلين، اموكسسلين، وسيفالوسبورين).

اظهرت هذه الدراسة باستخدام (Chi-square) زيادة ظاهرية في تركيز مضادات الاجسام (IgG, IgM, IgA and IgE) مقارنة مع التركيز الطبيعي (جدول 1 والصورة 1 ، 2 و3).

هذه الدراسة اظهرت ان زيادة التفاعل لهذه المضادات الحيوية دليل على زيادة الحساسية اكثر مما هو زيادة في تحفيز جينات الجلد .

Abstract

The clinical manifestations of hypersensitivity in 20 patients, working in an antibiotic industry in contact with antibiotic powder were studied immunologically.

Statistical analysis of the data by using Chi–square shows highly significant increase in the level of (IgG, IgM, IgA and IgE) antibodies than the normal ranges (table 1, Figures 1, 2 and 3).

It was concluding that increased reactivity to an antibiotic powder most likely reflects an allergic response rather than, increased genuine skin susceptibility.

Objective

The purpose of this study is to evaluate the possible potentiation of penicillin skin rash in contact with antibiotic powder in an antibiotics industry.

Introduction

The state of pharmacy in the united kingdom, including the start of allergic season, that association with exposure to the specific allergens, which are typically found in the environment such as pollens of different plants, house dust, fish and meat foods like shellfish and nuts, orange, strawberry, animal wool, tobacco, feed intended for fish dry Daphnia, and Cyclopes, cosmetic dye, polish creams, some drugs (iodine), bromine, antibiotics, sulfanilamide's, arsenic drug, bacteria, viruses, and products of their vital activity [1, 2, 3].

During an allergy, the human body produces strictly specific antibodies, unfortunately being protective antibodies with respect to those substances, they may be harmful for organs and tissues of the body itself, injuring the cells. They are contributed to the release of biologically active substances, enzymes having been accumulated in the blood in excess amounts, that may produce an irritating and stimulating effect on different organs and tissues, thereby causing what is called the allergic reaction of the immediate type.

At the same time a special factor is formed in white blood cells–lymphocytes. Thus the appearance of the allergen in the blood gives rise to an allergic response, delayed type develops, one or several days after allergen ingress in to host [4, 5].

The common clinical manifestation includes hay fever, asthma, eczema and urticaria [6].

Hypersensitivity:

Is an in appropriate reaction harmful to the host, which can be subdivided in to 4 main groups:

Type I: (Immediate (Anaphylactic Hypersensitivity)
An antigen induces the formation of IgE antibody which binds firmly by its Fe portion to basophilic and mast cells [7].

Type II: (Cytotoxic Hypersensitivity)
Antibody (IgG or IgM) directed at antigens of the cell–membrane, activate complement, and damage those cells [8].

Type III: (Immune–complex Hypersensitivity)
When antibody and antigen are coexist, immune complexes are formed like auto-immune disorders [9].

Type IV: Delayed (Cell–Mediated Hypersensitivity)
This is a function of T lymphocyte, not antibody, can transferred by T cells not by serum. The response is delayed (starts hours, or day after contact) with the antigen and often last for days[10].

Drug Hypersensitivity:

Drugs, particularly antimicrobial agents, are the most common causes of hypersensitivity reactions.

A metabolic product of the drugs, which act as a hapten binds to a body protein and lead to result antibody, which can react with hapten. The intact drug or the carrier protein are cell–bound and give rise to type I hypersensitivity [11].

Materials and Methods

Twenty samples of blood were collected from 20 individuals who work in an antibiotic industry suffering from skin rash, irritation and urticaria, separated of serum and stored bending biochemical analysis.

Methods: The methods used in this study were Elisa microplates (Direct enzyme–linked immuno sorbent assay), kit for the quantitative determination of total serum IgE and Radial immunodiffussion plates [12].

Principle of the Test:

1. Elisa kit: Is a two site enzyme–linked immuno sorbent assay:

* A first mouse monoclonal antibody is immobilized on to the plastic wells.
* A second Goat polyclonal antibody is labeled with the alkaline phosphatase enzyme.

The diluted sample is incubated with the solid–phase antibody –coated well. After the incubation period, the plate is washed–only the antigen from the patient sample remains bound on to the well.

In a second step, the conjugate–labeled antibody is added. If IgE is present in the sample a (Sandwich) complex is formed. After the end of the second incubation the well are washed to remove unbound conjugate and other unbound components.

A third incubation is performed with the chromogen (PNPP). The reaction is then stopped with NaOH, and the plate is read at 405nm. The colour intensity is directly proportionate to the amount of IgE in the sample [13].

2. Radial Immunodiffusion Plates:

One: Plate IgG.
Two: Plate IgM.
Three:Plate IgA.

Radial Immunodiffusion (RID) Plate:

For the accurate quantitative determination of proteins in serum and other biological fluid [14].

Principle: The purpose of all immuno diffusion techniques is to detect the reaction of antigen and antibody by the precipitation reaction Fig. (1, 2, and 3).

Result

1. Plate IgG: After 48 hours of incubation at 23°C. The result appears in table (1) that shows highly significant increase in the level of IgG than the normal range (normal range of IgG was 1190 mg/dL).

2. Plate IgM: After incubation for 72 hours at 23°C. The result shows in table (1) that there is a high significant increase in the concentration of IgM than the normal range (40 to 250 mg/dL). Average 130 mg/dL.
3. Plate IgA: After incubation for 48 hours at 23°C. The result shows in table (1) that there is a high significant level in IgA than the normal range (normal range 90 to 310 mg/dL). Average 220 mg/dL.
4. Elisa microplates: The result shows that there are more than half of all cases with high level than the normal range.
5. On the other hand, statistical analysis of data by chi–square show a significant increase for all plates table (2).
6. Also we measured the results of all plates according to the diameter square versus the concentration as shown in table (3).

Discussion

Statistical analysis of the data by using chi–square for all immunoglubulins showed high increased in the level of most plates than the normal range.

From the results in this study were appeared that drugs, particularly antimicrobial agents are the most common causes of hypersensitivity reactions[15]. So antibody (IgG or IgM) formation in this study means that drugs, e.g. penicillin can attach to the surface proteins on red blood cell and initiated antibody formation which activates complement and damage those cells. In case of our findings appears that all the patients suffering from cytotoxic hypersensitivity.

While the significant increase in the level IgE as shown in table (1) means that the induces formation of IgE antibody (immediate hypersensitivity) binds to basophil and mast cells leads to release of pharmacologically active mediator which can transmitted by serum. So this antigen–antibody complex triggers allergic responses of the immediate (anaphylactic) type [16].

It was concluded that increased reactivity to antibiotic powder most likely reflects an allergic response rather than an increased genume skin susceptibility[17].

While the increased in the level of IgA in this study may be due to that the contact with the antibiotics powder increased the secretions that associated with the increased IgA like saliva, colostram, tears, and respiratory tract secretion [18]. From all results presented in this study appears that the contact with antibiotic powder for many times, the workers may exhibit rashes, or local or systemic anaphylaxis of varying severity. So the recommendations to those workers for minimizing and preventing exposure to allergen in the work place. These recommendations are appeared by the importance of physician's and pharmacist's awareness of contact allergens in topical preparations.

	IgG	IgM	IgA	IgE
< Lowest normal range	0	0	2	5
Within normal range	0	2	3	4
> Lowest normal range	20	18	15	11

Table (1)

	Actual range				Row sum	Expected range			
	IgG	IgM	IgA	IgE		IgG	IgM	IgA	IgE
1	0	0	2	2	4	1	1	1	1
2	0	2	3	5	10	2.5	2.5	2.5	2.5
3	20	18	15	13	66	5.5	5.5	5.5	5.5
Column sum	20	20	20	20	80				

Table (2): Chi–square test between all subgroup of table (1)

P = 0.024779 < 0.05 which means that it is significant.

IgG			IgM			IgA		
D.mm	D.square mm^2	Conc. mg/dL	D.mm	D.square mm^2	Conc. mg/dL	D.mm	D.square mm^2	Conc. mg/dL
7	49	1614.6	6	36	144.6	4	16	48.5
7	49	1614.6	7	49	218.3	4	16	48.5
7	49	1614.6	9	81	399.7	6	36	226.9
8	64	2248	10	100	410^+	6	36	226.9
9	81	2965.9	10	100	410^+	6	36	226.9
9	81	2965.9	10	100	410^+	7	49	343
9	81	2965.9	11	121	410^+	7	49	343
10	100	3042.3	11	121	410^+	8	64	476.8
11	121	3042.3^+	12	144	410^+	10	100	644^+
9	81	2965.9	9	81	399.7	7	49	343
11	121	3042.3	10	100	410^+	7	49	343
9	81	2965.9	11	121	410^+	8	64	476.8
11	121	3042.3	12	144	410^+	10	100	644^+
9	81	2965.9	9	81	399.7	11	121	644^+
11	121	3042.3	12	144	410^+	12	144	644^+
11	121	3042.3	11	121	410^+	11	121	644^+
11	121	3042.3	11	121	410^+	10	100	644^+
9	81	2965.9	9	81	399.7	8	64	476.8
11	121	3042.3	12	144	410^+	11	121	644^+
11	121	3042.3	11	121	410^+	12	144	644^+

Table (3)

Fig. 1

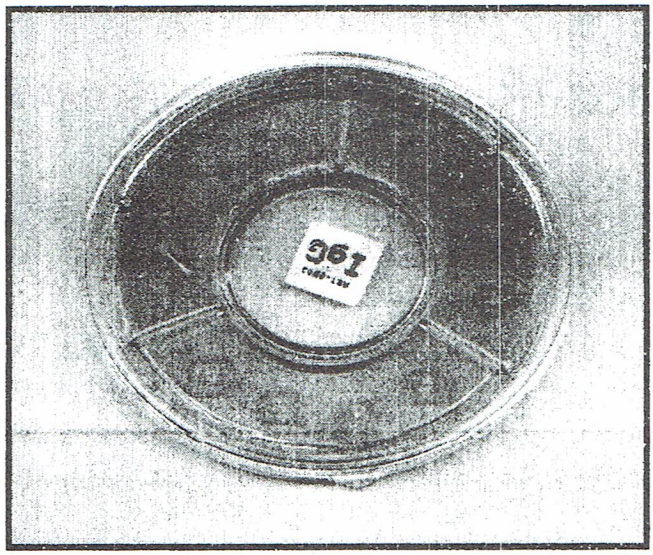


Fig. 2

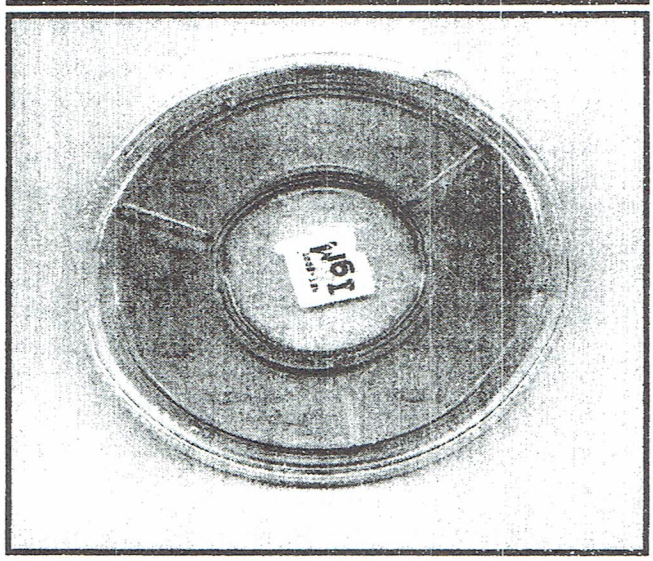
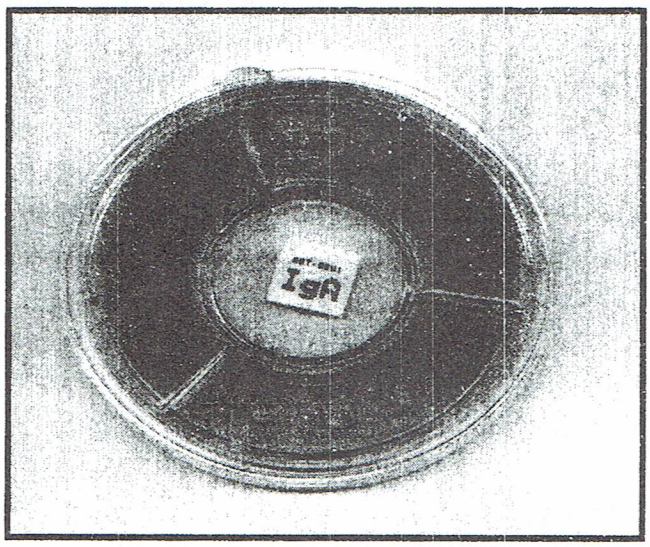


Fig. 3



Immuno diffussion plates of IgG, IgM and IgA

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