

CORRELATION BETWEEN IgE AND DIFFERENT STATES OF DENTAL PULPS

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ABSTRACT

The presence of immune factors in inflamed dental pulps, suggests that immune responses could participate in disease process. With regard to the presence of significant number of mast cells in pulpitis cases, the purpose of this study was to determine the correlation between concentration and presence of IgE in supernatant fluids of explants cultures of different states of dental pulps.

For this purpose, a total of forty-eight pulpal samples were evaluated. They were 10, 10, 8, 11, and 9 pulpal samples from impacted teeth, normal erupted teeth, teeth with carious lesions, irreversible symptomatic pulpitis and irreversible asymptomatic pulpitis, respectively. The samples were maintained in explants cultures for 72 hours. Sandwich ELISA was used to detect and quantitate IgE in supernatant fluids.

It was shown that in impacted and carious samples, there were not any IgE. The presence of IgE in normal, symptomatic and asymptomatic samples were 30%, 45.5% and 55.6%, with average concentration of 0.063 ± 0.02 , 0.24 ± 0.148 and 0.235 ± 20.17 IU/ml, respectively.

Statistical analysis showed significant differences between different states of pulps with regard to presence of IgE ($P=0.0324$). With regard to concentration of IgE, there were significant differences between impacted and symptomatic ($P=0.0325$); and carious and symptomatic ($P=0.0397$).

It is concluded that with increase in antigenic stimuli, there is a proper condition for production of IgE, as the highest presence of IgE was related to irreversible asymptomatic pulpitis. Bacterial by-products are possible allergens, which can induce the production of IgE.

Keywords: IgE, Immunopathogenesis, Dental pulp

IgE and Dental Pulp

INTRODUCTION

Inflammatory diseases of dental pulps are one group of most common diseases of the teeth. Their primary cause is dental caries, which in turn is induced by microorganisms^(1,2).

In addition to microorganisms and their products, it seems that defense reactions of dental pulps may be involved in the disease process. Presence of various immunocompetent cells⁽³⁻⁸⁾, different classes of immunoglobulins such as IgG, IgA, IgM⁽⁹⁻¹¹⁾ and various kinds of inflammatory mediators^(10,12) in inflamed pulps, indicate the possible roles of immune reactions.

With regard to the presence of numerous mast cells in inflamed pulps⁽⁵⁾ and the roles of these cells in the induction of type I hypersensitivity reaction (Allergy), we based our study on determining the correlation between IgE, as the most important factor in triggering this type of hypersensitivity reactions, and different states of dental pulps.

MATERIALS AND METHODS

Sample collection

A total of forty eight pulpal samples, consisted of 10; 10; 8; 11; and 9 pulpal samples of impacted teeth; normal erupted teeth; teeth with carious lesions; with irreversible symptomatic pulpitis; and irreversible asymptomatic pulpitis, respectively, were collected from 46 patients in Dental faculty of Shaheed Beheshti University of Medical Sciences. Among the asymptomatic samples, there were six samples of pulp polyps and three samples of ulcerative pulpitis. All the groups were matched with regard to gender, but we could not match the above five groups with regard to age. The average age and percentages of different sexes in these five groups are shown in table 1.

Table 1. Distribution of different sexes and average of age in 5 different groups

Variables	Gender (%)		Average of Age (yrs.)
	Female	Male	
Impacted	50	50	20.9±2.33
Normal erupted	60	40	24.1±5.28
Carious	37.5	62.5	23.38±7.65
Symptomatic pulpitis	54.5	45.5	30.91±6.52
Asymptomatic pulpitis	66.7	33.3	25.1±9.92

Tissue explants cultures .

Immediately after removing the pulpal tissues, the tissue samples were placed directly into a sterile 7 ml tubes, containing 5 ml of RPMI-1640 tissue culture medium [(10 g/lit), Bahar Afshan Laboratory, Tehran- Iran], supplemented with Fetal Calf Serum [(10%), Bahar Afshan Laboratory, Tehran- Iran], gentamicin sulfate [(100 µg/ml), Zahrvy Laboratory, Tabriz- Iran], and fungizon or amphotericin B [(5 µg/ml), Bristol- Myers Squibb-Paris-France], and refrigerated at 4°C. At the end of each week, all of the collected samples were taken to tissue culture room of Immunology department of Medical School of Shaheed Beheshti University of Medical Sciences, and each was placed on a sterile 58×15 mm petri dish (Haghghat Laboratory, Tehran-Iran) and was washed with RPMI-1640 (10 g/lit) plus gentamicine sulfate (20 µg/ml) and fungizon (2.5 µg/ml). The tissues were blotted on the lid of petri dishes to remove excess fluids. Then the samples were placed on another petri dish and aseptically sectioned into pieces approximately 1 mm in diameter by a # 15 sterile scalpel. Each piece was placed into one well of a 96-well cell culture plate (Greiner - Germany) to which was added 300 µlit per well of RPMI- 1640 (10 g/lit) plus gentamicine sulfate (20 µg/ ml) and fungizon (2.5 µg/ml).

Then, the explants tissue cultures were incubated at 37°C in an atmosphere of 5% CO₂ and 95% humidity for 3 days. At the end of incubation time, the supernatant fluids were aspirated by tuberculin syringes and then divided into microtubes (Haghghat Laboratory, Tehran-Iran) and immediately freezed at -70°C.

Histopathologic examination

In order to examine the tissue culture conditions, the first series of cultured pulpal samples were referred to Oral Pathology Department of Dental School of Shaheed Beheshti University of Medical Sciences. Fortunately, all of the examinations indicated that tissue culture conditions were suitable.

Determination of IgE by ELISA

After collecting all of the samples, the supernatant fluids were defreezed and centrifuged

for 15 minutes at 2000 rpm. Sandwich ELISA were used in order to determine the presence and concentration of IgE, by using DAI-IgE ELISA kits.

Statistical analysis

Statistical analysis was made by analysis of variance, T test, Chi square and Fisher exact test.

RESULTS

We could not detect any IgE in pulpal samples of impacted and carious teeth. The presence of IgE in normal erupted; symptomatic; and asymptomatic pulpitis samples was 30%, 45.5%, and 55.6%, with an average concentration of 0.063 ± 0.02 ; 0.24 ± 0.148 ; and 0.2352 ± 0.17 IU/ml, respectively. The presence of IgE in pulp polyps was 66.7% with an average concentration of 0.353 ± 0.222 IU/ml. Chi square test, showed significant differences between these five groups with regard to presence of IgE ($P=0.0342$). By comparing every two groups with each other by Chi square and Fisher exact test, it was shown that there were significant differences between impacted and symptomatic ($P=0.0277$), impacted and asymptomatic ($P=0.03251$), impacted and pulp polyps ($P=0.00824$), carious and symptomatic ($P=0.0397$), and carious and pulp polyps ($P=0.029$) with regard to presence of IgE.

By using analysis of variance, there was not any significant difference among these 5 groups with regard to the amounts of IgE. However, by comparing every two groups with each other by T test, there were significant differences between impacted and symptomatic ($P=0.0325$) and carious and symptomatic samples ($P=0.0324$).

In Fig. 1 and Fig. 2, the presence and concentrations of IgE in the above five groups are compared with each other.

DISCUSSION

In this study the presence of IgE was seen in 0%, 30%, 0%, 45.5%, and 44.4% of pulpal samples of impacted teeth; normal erupted teeth, teeth with carious lesions, with irreversible symptomatic pulpitis, and with irreversible asymptomatic pulpitis, respectively. The average concentration of IgE in the above samples was 0, 0.063 ± 0.02 , 0, 0.24 ± 0.148 , and 0.235 ± 0.17 IU/ml, respectively. The presence of

IgE in pulp polyps was 66.7% with average concentration of 0.353 ± 0.222 IU/ml.

With regard to the presence of IgE, there were significant differences between these five groups ($P=0.0342$), thus the most abundant cases of presence of IgE were symptomatic and asymptomatic pulpitis.

With regard to IgE concentration, there was not any significant difference between the groups. However, by comparison of every two groups with each other, there were significant differences between impacted and symptomatic ($P=0.0325$), and between carious and symptomatic samples ($P=0.0324$). Therefore, the symptomatic and asymptomatic samples had higher concentrations of IgE.

Based on existing data, there is no study, which has been done for determining IgE or its correlation with different states of dental pulps. However, there are some studies, which have been done for determining the presence or concentrations of other classes of immunoglobulins.

Speer et al (1977), reported that average concentration of IgG and IgA were higher in inflamed pulps than normal pulps⁽³⁾. Although their study was done on different classes of immunoglobulins compared to that of ours, but there are some similarities in that the presence and concentration of IgE were higher in inflamed pulpal samples (symptomatic and asymptomatic pulpitis) too.

Nakanishi et al (1995), found that in symptomatic pulpitis, there were higher amounts of IgG, IgA and IgM than those with normal pulps⁽¹⁰⁾. In our study, although the presence and average concentration of IgE were higher in symptomatic pulpitis than normal erupted teeth, but these differences were not significant. It might be due to the fact that in our study, all of the normal erupted teeth were third molars (wisdom teeth), which because of their eruption into the oral cavity in most cases are associated with pericoronitis, which in turn results in further entrance of antigens into oral mucosa and subsequent synthesis of immunoglobulins. After eradication of inflammation, it takes time for complete degradation of these immunoglobulins. Of course, as we mentioned earlier, our study was done on a different immunoglobulin.

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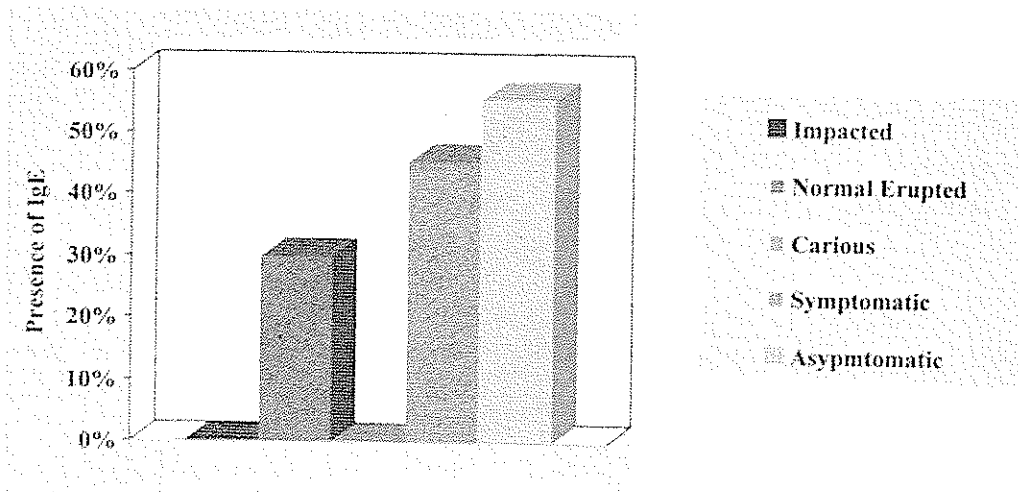


Fig. 1. Comparison of presence of IgE (%)

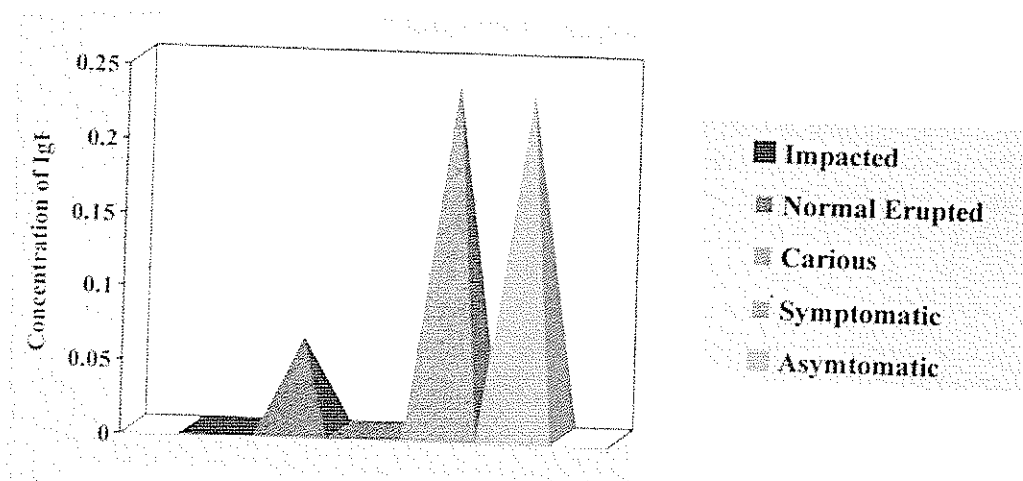


Fig. 2. Comparison of concentration of IgE (IU/ml)

Zerosi et al (1999), found that Russel bodies were found in most cases of chronic pulpitis. They suggested that irritative stimuli caused by the bacteria present in chronically inflamed pulps led to hyper activation of plasma cells and the consequent hyper production of immunoglobulins (13). We also found higher amounts of IgE in asymptomatic samples than impacted, normal erupted and carious teeth. In addition, in most of pulp polyp samples (66.7%), we determined the presence of IgE. Thus, in pulp polyps, IgE exists in its highest amount.

CONCLUSION

The significant amounts of IgE in the pulpal samples of irreversible pulpitis suggest that with continued antigenic stimuli and tissue destruction, the production of IgE could occur. The main causes of the production of IgE are bacterial-by products and toxins and host denatured tissues, which could act as allergens.

IgE could trigger type I hypersensitivity reaction and further destruction of pulpal tissue. More work is needed to determine if a type I hypersensitivity reaction is involved in pulpitis.

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