Concentration of salivary immunoglobulin A, in relation to periodontal disease, plaque, and calculus

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ABSTRACT

Background: It has become apparent that every pathologic process in the body involves the immune system. Periodontal inflammation is not an exception. Periodontal health depends on the interaction of microbial flora and host response. Since long, research is focused on understanding the immunopathologic mechanism operating in the development and maintenance of periodontal inflammatory changes. **Methods:** Patients of age 20-35 were selected. Nonstimulated saliva was collected and assessed using radial immunodiffusion assay to estimate levels of IgA. **Results:** The present study suggests patients with gingival index 0.2–0.5 and periodontal index 0 have a concentration of IgA less than 21.4%. As values of gingival and periodontal index go on increasing, the concentration of salivary IgA also increases. **Conclusion:** the concentration of salivary IgA is directly and positively correlated to severity of inflammation. Also the concentration of IgA depends on the presence of plaque.

Key words: Calculus, immunoglobulins, plaque, saliva

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INTRODUCTION

It has become apparent that every pathologic process in the body involves the immune system. Periodontal inflammation is not an exception.[1] Periodontal health depends on the interaction of microbial flora and host response. Since long, research is focused on understanding the immunopathologic mechanism operating in the development and maintenance of periodontal inflammatory changes.[2] Microbes play an important role in periodontal disease. They may induce destruction by:

1. Direct initiation of inflammatory responses by injurious microbial metabolites; and
2. Initiation of periodontal inflammation by antigens or oral microbes setting immunopathologic processes into action.[3]

The oral microbiota is regulated by pervasive influence of saliva.[4] Local immunity and antibodies may be of primary importance in defense against infections confined to mucosa.

Oral fluid immunology came into existence as an entity when an entirely separate protective system based on local antibody production was recognized for exocrine fluids and transcellular fluids. The external secretions that bathe oral mucosa form

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a unique immunologic panoply, which serves host defense. It is reported that the levels of antibodies in mucosal fluids correlate more directly with resistance to certain infections than did serum antibody titers.[5] IgA is shown to be a predominant immunoglobulin of external secretions against IgG, which predominates in serum and internal secretions. Also IgA secretion is independent of serumal response. These observations are critical features of an immunologic system common to mucosal surfaces and are referred as secretory or mucosal immune system.[6]

Due to quantitative dominance and superior stability IgA is a potentially important secretory immunoglobulin. sIgA inhibits the mucosal penetration and acts as a first line of defense. Antibacterial activity is attributed to its property of coating and agglutinating microbes.

Combination of bacteria with IgA inhibits sticking to epithelial cells and reduces colonization.[7]

Quantification of immunoglobulins in the whole saliva poses problems. Firstly, the contribution to this fluid from minor and major salivary glands varies greatly according to salivary flow rate. Secondly, the flow rate of saliva cannot be accurately measured. Also the fluid has to be cleared by centrifugation leading to variable loss of immunoglobulins.

The study thus uses unstimulated whole saliva for analysis of salivary IgA to establish correlation between sIgA concentration in salivary samples of patients with varying severity of gingivitis and periodontitis.

MATERIALS AND METHODS

Patients attending the outpatient department of Department of Periodontics, GDC Nagpur were examined. A total of 44 patients were selected; and 33 patients had varying grades of gingivitis and periodontitis and were included in test group. Eleven patients had clinically normal gingiva and were included in the control group. Age range was 20–35 years. Thirty were female and 14 were male subjects. Patients with any systemic diseases were excluded from the study.

Patients were selected according to gingival index by Loe and Silness.[8]

The subjects were divided into 4 subgroups depending on gingival index.

- Gr I (G.I.: 0): Females 6, Males 5
- Gr II (G.I.: 1): Females 8, Males 3
- Gr III (G.I.: 2): Females 8, Males 3
- Gr IV (G.I.: 3): Females 8, Males 3

All the groups were assessed for periodontitis using Russel's Periodontal Index.[9]

Whole salivary samples were collected without stimulation. The patient was advised not to consume anything 1 h before collection. The patient was asked to spit in 5 mL flask. Approximately 2–3 mL of saliva was collected from each patient. pH of samples was measured. The samples were then centrifuged in a Remi centrifuge at 3000*g* for 5 min.

The concentration of IgA was estimated using Radial Immunodiffusion assay proposed by Mancini *et al* 1965.[10] Tri-Partigen plates were used for analysis (Hoechst Pharma, India). Along with the plates, control serum was also supplied.

Following steps were done for assay,

- The plates were left open for 5 min to evaporate any possible water that was condensed.
- Well number 1 was filled with 5 μL of control serum. Wells 2–12 were filled with 5 μL of patient's saliva. The saliva used was clear, undiluted, and was obtained after centrifugation with microcapillary tubes. The tube was carried to the center of the well, and by depressing the microplunger each well was carefully filled without damaging the agarose gel.
- The fluid was dispersed and plates were tightly closed and incubated at room temperature. Plates were kept upside down to prevent any

possible evaporation of samples.
- Normal time for IgA diffusion is 50 h and hence the plates were incubated for 50 h.
- The precipitation rings were measured to nearest 0.1 mL with help of a standard Partigen ruler.

With the aid of control serum the quality of plates and test method were controlled. The ring diameter of all wells were measured and compared with values in reference table. Statistical analysis was carried out using Student's t test.

RESULTS AND DISCUSSION

The present study suggests patients with gingival index 0.2–0.5 and periodontal index 0 have a concentration of IgA less than 21.4%. As values of gingival and periodontal index go on increasing, the concentration of salivary IgA also increases.

Secretory IgA is a complex molecule with IgA dimer and a secretory piece with a "J" chain. The presence of secretory component makes the IgA more stable and less susceptible to attack by proteolytic enzymes.[5,11] Two subclasses of IgA are identified as IgA1 and A2. IgA1 is 90% of total serum IgA whereas mucosal IgA has 40%–60% of IgA2 subclass.

IgA class of antibody has antiviral, antitoxin, and antibacterial activity. Studies by Shannon and Suddick state that antibodies can prevent bacterial adhesion. They also have antibacterial activity.[12] This has been supported by Lindstrom and Folke 1973.[13] The rise in concentration of salivary IgA can be supported by studies of Brandtzaeg (1975) and Orstavik (1975) that have shown association between IgA level and periodontal inflammation.[14] Similarly, studies by Brandtzaeg[15] has shown correlation between periodontal inflammation and levels of sIgA. These studies are also supported by work of Shillitoe *et al*,[16] Harding *et al*,[17] Guven and Visscher.[18]

The agglutinating mechanism is explained by the fact that antibodies in the saliva react with bacteria proliferating on surface and impair their attachment. Studies by Schwartz and Buckley[19] showed that patients with IgA deficiency have increased incidence of mucosal infection.

Thus on the basis of the study we conclude that the concentration of salivary IgA is directly and positively correlated to severity of inflammation.

Also the concentration of IgA depends on the presence of plaque.

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