SALIVARY IgG AND IgM IN GINGIVITIS

O. Güven

Department of Oral Surgery School of Dentistry University of Ankara

Key-words: salivary IgG and IgM — gingivitis — multiple radial diffusion method

INTRODUCTION

In recent years a reasonable theory has been advanced for the pathogenesis of periodontal disease. The theory states that periodontal inflammation is due to the reaction of the host’s immune system against products released from plaque. Microbial plaque lying in close proximity to the host’s gingival tissues, may be the primary etiologic factor in gingival inflammation (1—3).

An important stage in the progression of periodontal disease is the onset of flow of serous crevicular fluid. Crevicular fluid complement is rapidly activated by a combination of factors. These include activation of classical pathway by IgG and IgM antibodies to subgingival plaque antigens (4).

IgG in normal external secretions is derived via transudation from serum and by direct synthesis. IgG reaches the interstitial fluid bathing the lamina propria by permeating the capillary walls, but the route across the epitelium is unknown. Fluorescent antibody studies have demonstrated IgG and only a small number of IgG containing plasma cells in the interstitial space of normal individuals. When inflammation occurs there may be both an increase in transudation of IgG from serum and an invasion of the mucosa by IgG producing plasma cells (4).

The detection of large amounts of IgM in secretions and plasma cells in mucosal tissues of IgA deficient patients, suggests that IgM is also produced locally (5). Local IgM synthesis occurs in certain secretory tissues and there is a predominance of IgM compared to IgG in mucosal fluids such as saliva (6, 7).

The IgA in periodontal disease has been discussed in most of the previous literature (8—11). The purpose of this report is to evaluate the possible roles of IgG and IgM in gingivitis.

MATERIALS AND METHODS

Whole-salivary samples from 60 individuals were obtained after breakfast without any stimulation and stored in deep freeze (− 20 °C) until use. The subjects of the study were grouped according to their degree of gingival
inflammation as defined by LÖE's (12) Gingival Index (GI). Each group consisted of 15 subjects.

The qualitative presence of the IgG and IgM were investigated using the method of "Multiple Radial Diffusion" (MRD) as DOMAN (13) described. Technique:

15 g/l agar gel in sodium veronal/veronal buffer of pH 8.6 (sodium veronal 10.3 g/l + veronal 1.83 g/l) was incubated at 50 C. After 3 min, preheated saliva (5 ml) were mixed with the gel to give final volume of 10 ml and poured onto a standard-size plate (70 mm x 80 mm). Antibody reservoirs were prepared. 10μ l of antiserum was put into each of them. After 48 h incubation at room temperature, the plates were rinsed for 24 h in a 0.15 M NaCl solution, dried and stained. The ring diameters were measured and Ig concentrations were calculated by using calibration curves.

RESULTS

Salivary IgG and IgM quantities in relation with the severity of the gingivitis were divided into four groups;

Group 1 (GI 0): IgG was detected in 11 salivary samples. In this group the range of concentrations of IgG were found to be 0.6—9.5 mg%, mean 2.8 mg%, standard deviation (SD) 2.9 mg%. IgM was not present.

Group 2 (GI 1): In the second group, IgG was detected in 6 samples. The range was 0.9—24 mg%, mean 9.9 mg%, SD 9.6 mg%. IgM was detected in 2 samples. These concentrations were 4.1 mg% and 10.5 mg%.

Group 3 (GI 2): In this group IgG was detected in 11 samples. The range was 0.9—21 mg%, the mean value 7 mg% and SD 6.5 mg%. IgM was detected in 3 samples. Minimum concentration was 0.8 mg%, maximum concentration was 6.1 mg%.

Group 4 (GI 3): In the last group IgG was detected in 13 samples. The range was 0.8—25 mg%, mean 9 mg%, SD 7.5 mg%. IgM was detected in 6 salivary samples. Minimum concentration was 1.3 mg%, maximum concentration was 6 mg%.

DISCUSSION

It has been reported previously that the IgG was the principal immunoglobulin fraction found in gingival tissues (14). Its levels appeared to be substantially increased in the inflamed gingiva. (15, 16) Although BYERS et al. (15) reported that IgM can not be consistently demonstrated in inflamed gingiva with the assay they have employed, THONARD et al. (17) concluded that IgM is the dominant fraction detected both intra- and extra-cellularly. BRANDTZAEG et al. (18) showed that whole salivary IgG and IgM concentrations of subjects with gingivitis were higher than the healthy subjects.
Subsequently DICARLO et al. (19) reported the similar elevation for salivary IgG in the patients with gingivitis and their conclusion for IgM were the same as ours. They could not detect IgM in normal human saliva and these results also supported the results of MACH et al. (20).

LAZAREVSKA et al. (21) studying the patients who have periodontal disease, observed higher salivary IgG levels in patients with periodontitis compared to normal controls.

HARDING et al. (22) recently reported that the individuals with acute necrotising ulcerative gingivitis demonstrated no detectable concentration of IgM and a decreased concentration of IgG in whole saliva.

Our statistical analysis of the results showed that there was no significant difference between the IgG concentrations in the last 3 groups, but in each of these groups, IgG concentrations were higher than the first group.

Although IgM was not present in the first group, it was possible to detect IgM in the second group in 2 samples. IgM has been demonstrated in the third group in 3 samples and in the last group in 6 samples. This indicates that there is a positive correlation between the severity of gingivitis and a possible presence of IgM in whole saliva. It is remarkable that IgM was never present alone in a salivary sample. Both IgG and IgM were always found in the same salivary sample.

SUMMARY

Saliva of patients with gingivitis were investigated for IgG and IgM levels with the technique of "Multiple Radial Diffusion". Patients were divided into four subgroups according to the severity of their disease. Salivary samples were collected from 15 patients in each subgroup after breakfast. The samples were frozen and then stored. Salivary IgG levels did not differ significantly between the GI 1, GI 2 and GI 3 patients, but all of these subgroups displayed higher IgG levels than those seen in the GI 0 patients. On the other hand there was a continuous increase of the salivary IgM level with each increased severity step of the gingivitis. IgM was not present alone in the saliva, it was found always together with IgG.

RÉSUMÉ

O. Gitten — La concentration salivaire de la Ig G et de la Ig M au cours de la gingivite

Mots-clé: concentration salivaire de Ig G et Ig M — gingivite — méthode de diffusion radiale multiple

Les niveaux salivaire des Ig G et Ig M ont été déterminés par la méthode de "diffusion radiale multiple".

Les patients ont été répartisés en quatre groupes (15 patients dans chaque groupe) en relation avec la sévérité de la gingivite. Après le petit déjeuner, on a recolté une petite quantité de saliva qui a été ensuite gelée et conservée.

Les niveaux salivaire des Ig G n'ont pas été différents d'une manière significative entre les trois groupes mais ont été plus grands par rapport avec la groupe étalon.

On a observé aussi un haussement continu du niveau salivaire de Ig M conformément à la sévérité de la gingivite.

REFERENCES


Author's address
Dr. Orhan Güven
Yestilyurt Sokak No. 24/18
Asagi Ayränci
Ankara
Turkey