THE EFFECT OF QAT (CATHA EDULIS FORSK) AND TOBACCO CHEWING HABITS ON CHRONIC INFLAMMATORY CELLS IN SAMPLES OBTAINED FROM PATIENTS WITH CHRONIC PERIODONTITIS

Mohammed M. A. Abdullah * and Sherif Y. El-Nagdy **

ABSTRACT

Background: Periodontal disease is mainly caused by interactions between bacteria of the dental plaque and components of the cellular and humoral host immune response, including cytokines and biological mediators released by activated immunocompetent cells.

Aim of the study: The present study was designed to evaluate the effect of qat (Catha edulis forsk) and tobacco chewing habits on chronic inflammatory cells in samples obtained from patients with chronic periodontitis.

Material and Methods: Sixty patients with untreated chronic periodontitis were subjected in the present study and divided into three groups qat chewing, tobacco chewing and non chewing(control group). Plaque index (PLI), gingival index (GI) and clinical attachment level (CAL) were recorded. Gingival biopsies were obtained from selected site for assessing histopathological changes.

Results: The clinical study showed significant differences in plaque index (PLI) , gingival index (GI) and clinical attachment loss between qat chewing and tobacco chewing patients group compared to non chewing patients group (control group). Histopathological study showed increased inflammatory cells in tobacco chewing and qat chewing patients groups compared to non chewing patients group.

Conclusion: The mechanisms of destruction effect of qat chewing habit vary from tissue degradation in chronic periodontitis in tobacco chewing chewers.

KEY WORDS: Qat chewing (Catha edulis forsk). Chronic inflammatory cells, tobacco chewing.

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INTRODUCTION

Periodontal diseases are histologically characterized by an infiltration of several inflammatory cell populations into the gingival epithelium and connective tissue, associated with degradation of extracellular matrix components. It is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both. The mechanisms how periodontitis induces systemic inflammation are still unclear, but may include systemic dissemination of live bacteria and components from lysed bacteria and, as recently suggested, free-soluble components from live planktonic and biofilm bacteria from periodontal pockets. Bacterial colonization and growth on supra- and subgingival tooth surfaces causes chronic inflammation in periodontal tissues. Numerous studies have demonstrated variations in the number and/or density of lymphocyte subpopulations as well as in the number of plasma cells during different stages of periodontal disease. While T lymphocytes represent the predominant inflammatory cell type in early and stable periodontal lesions, B lymphocytes and plasma cells predominate in advanced and progressive lesions, such as chronic periodontitis. In addition to lymphocytes, macrophages represent another important cell type involved in host defense against bacterial aggression, accounting for about 5-30% of the inflammatory infiltrate found in periodontal lesions. Neutrophils, lymphocytes and monocytes were regularly found in samples from clinically healthy gingivae even when histological sections failed to show any inflammatory infiltrates in the gingival connective tissue. The differential counts showed 95–97 % neutrophils, 1–2 % lymphocytes and 2–3 % monocytes. Increased numbers of leukocytes were found in the crevices of chronically inflamed gingivae. However, the proportions of the various leukocytes were the same as those found in the crevices of clinically healthy gingivae. The results of the present investigation support the view that only quantitative differences exist between clinically healthy and chronically inflamed gingivae.

Qat (catha edulis) is a natural stimulant from the catha edulis plant, found in the flowering evergreen tree or large shrub of Celastracea family, which grows mainly in Yemen, Ethiopia, Somalia, Kenya, Saudi Arabia, and at high altitude areas in South Africa and Madagascar. The active ingredient of qat responsible for its psychostimulant effect is an alkaloid chemical known as cathinone, which is structurally and chemically similar to damphetamine, and cathine, a milder form of cathinone. Cathinone is a highly potent stimulant, which produces sympathomimetic and central nervous system stimulation analogous to the effect of amphetamine. Fresh leaves contain both ingredients; those left unrefrigerated beyond 48 hours would contain only cathine, which explains users’ preference for fresh leaves. qat loses its potency after 48 hours. The results of various in vivo and in vitro experiments indicate that the substance could be considered as a “natural amphetamine”. Buccal and gingival mucosa at sites of qat chewing among Yemeni chewers had been extensively studied histopathologically in the past 6 years. The results revealed that histopathological changes found on oral mucosa included increased rete ridges, acanthosis, intercellular edema, orthokeratosis, parakeratosis, epithelial dysplasia, inflammatory cell infiltrate and increased amount of collagen fibers. All results were statistically significant except for results of epithelial dysplasia and inflammatory cell infiltrates. Also the distribution of histopathological cases among smokers and non smokers were statistically not significant. Tobacco smoking is an addictive habit first introduced into Europe. Smoking is now recognized as the most important cause of preventable death and disease. Nicotine is considered the most pharmacologically active compound in tobacco smoke. Most is absorbed through the lung alveoli, but nicotine can also be absorbed, though more slowly, through...
the oral mucosa in sufficient quantities to have a pharmacological effect. Smoking does not normally lead to striking gingival changes. A reduction in clinical signs of gingivitis has been reported in smokers and this effect has been shown to be independent of plaque levels. Heavy smokers may have greyish discoloration and hyperkeratosis of the gingiva: an increased number of keratinized cells has been found in the gingiva of smokers. Changes in the epithelium were described as keratotic, hyperkeratotic and hyperplastic. Bergstrom et al. have found less gingival bleeding in smokers than in non-smokers, due to vasoconstriction of gingival vessels, but may also be attributable to the heavier keratinization of the gingivae in smokers. Cigarette smokers had significantly greater probing depths and bone loss than non-smokers although no difference was found in relation to tooth mobility. Bergstrom et al. found smokers not only to have significantly increased probing depths and alveolar bone loss, but also increased tooth mobility. Some studies have also highlighted the dose relationship between the effect of cigarette consumption and periodontal attachment loss.

The purpose of this study was to evaluate the effect of qat (Catha edulis forsk) and tobacco chewing habits on chronic inflammatory cells in samples obtained from patients with chronic periodontitis.

MATERIALS AND METHODS

A) The Study samples

Sixty patients comprising groups 20 patients in qat chewers, 20 patients in tobacco chewers and 20 patients in non chewers patients group, all in the age groups ranging between 25–50 years were selected from among the patients referred to the out patients clinics of periodontics at the college of dentistry, King Khalid University. The subjects for the study were selected randomly from the patients companyed from untreated chronic periodontitis at least 6 months ago and they should not have had any known systemic conditions that could influence periodontal health taking into consideration only their qat (Catha edulis forsk) and tobacco chewing history since one year or more in the test groups and the patients in control group should not have qat (Catha edulis forsk) or tobacco chewing at anytime in their lives (Fig.1&2).

B) Clinical examination

A thorough periodontal examination was carried out under good artificial light, and parameters selected for the study were carefully recorded as follow:

1. Plaque index of Silness and Loe
2. Gingival index of Loe and Silness, and
3. Clinical attachment level (Fig.3).

Fig. (1) Photograph of qat chewer

Fig. (2) Photograph of tobacco chewer
C) Histopathological Examination:

After the clinical examination preparation was done to take biopsy. Biopsy was obtained by sharp dissection with a Bard Parker blade no. 15 under local anesthesia. The biopsy specimen was immediately transferred to a bottle containing 50% formalo-alcohol (50 mL of 10% formalin and 50 mL of alcohol) and kept for 24 hours for fixation. Slides were prepared by standard histological technique using haemotoxylin and eosin stain. All the slides were viewed under compound microscope attached with a micrometer scale at 20x (objective) magnification to which a camera was attached. Four views of each slide were then photographed with the scale adjusted for each photograph. These photographs were then transferred to a computer and were assessed. Numbers of inflammatory cells were estimated (Fig.4,5 &6).

D) Statistical analysis

All data were collected then analyzed by Chi-square test to test the proportions of study parameters between qat (Catha edulis forsk) and tobacco chewing patients and non chewing patients.

RESULTS

The mean age of qat chewers patients was 37.13±6.33, the mean age of tobacco chewers was 33.56±4.77 and the mean age of non chewers patients (group control) was 35.64±4.65, distribution of patients in the two test groups summarized in table (1).
There were increased number of patients had qat (Catha edulis forsk ) chewing history 11-15 years and more than 15 years 6 and 7 patients compared to qat (Catha edulis forsk ) chewing history 1-5 years and 6-10 years 3 and 4 patients respectively. There were increased number of patients had tobacco chewing history 11-15 years and more than 15 years 5 and 6 patients compared to tobacco chewing history 1-5 years and 6-10 years 4 and 5 patients respectively.

Table (2) and figure (7) summarize the clinical parameters of this study. In qat (Catha edulis forsk ) chewers group the means and ± SD of plaque index (PLI), gingival index(GI) and clinical attachment level were (1.1±0.39), (2.09±0.3) and (4.95±0.33) respectively and the means and ± SD of plaque index (PLI), gingival index(GI) and clinical attachment level in tobacco chewers group were (1.06±0.32), (2.86±0.39) and (3.98±0.37) respectively whereas the means and ± SD of plaque index (PLI), gingival index(GI) and clinical attachment level in non chewers (control) group were (1.44±0.37), (1.09±0.3) and (2.8±0.43) respectively. There were significant differences in plaque index in patients with bad habits groups compared to control patients group. The plaque index (PLI) more value in control group patients than test groups patients due to the self cleaning during qat (Catha edulis forsk ) and tobacco chewing whereas the gingival index (GI) more value in test groups patients than control group patients but it was more in tobacco chewing patients than qat (Catha edulis forsk ) chewing patients due to the degradation effect of nicotine against periodontal tissues($P \leq 0.001$). The clinical attachment level (CAL) more value in test groups patients than control group patients but it was more in qat (Catha edulis forsk ) chewing patients than tobacco chewing patients due to the traumatic effect of qat (Catha edulis forsk ) chewing habit against periodontal tissues.

Table (3) and figure (8) showed increased number of chronic inflammatory cells in test groups patients than control group patients but it is more in tobacco chewers patients than qat (Catha edulis forsk ) chewers patients due to the degradation effect of nicotine against periodontal tissues.

**TABLE (1) Distribution of qat (Catha edulis forsk ) and tobacco chewing**

<table>
<thead>
<tr>
<th>Chewing /years</th>
<th>Qat chewing (20)</th>
<th>Tobacco chewing (20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. P.</td>
<td>%</td>
</tr>
<tr>
<td>1-5</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>6-10</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>11-15</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>&gt; 15</td>
<td>7</td>
<td>35</td>
</tr>
</tbody>
</table>

**TABLE (2) Mean and ± SD of clinical parameters**

<table>
<thead>
<tr>
<th>Groups</th>
<th>PLI</th>
<th>GI</th>
<th>CAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qat chewing</td>
<td>1.1±0.39</td>
<td>2.09±0.3</td>
<td>4.95±0.33</td>
</tr>
<tr>
<td>Tobacco chewing</td>
<td>1.06±0.32</td>
<td>2.86±0.39</td>
<td>3.98±0.37</td>
</tr>
<tr>
<td>Non chewing habit</td>
<td>1.44±0.37</td>
<td>1.09±0.3</td>
<td>2.8±0.43</td>
</tr>
</tbody>
</table>
DISCUSSION

Qat chewing plays a significant role in deterioration of oral hygiene. Complaints such as gingival bleeding, halitosis, difficulty with mouth-opening and swallowing solid food, and the burning sensation in the soft tissues were significantly more common among chewers than among non-chewers. Hill and Gibson reported that qat chewing was associated with a higher prevalence of gingival bleeding. The hardness of the qat and friction mechanism with mucosal and periodontal tissues might be responsible for the poor periodontal status of chewers. Qat, which contains alkaloids, in addition to the presence of pesticides in qat leaves might have a significant causative role in periodontal diseases along with other variables such as the level of oral hygiene, halitosis and gingival bleeding. This lends support to the earlier findings of pigmentation on the qat chewing site which is directly related to mechanical and chemical components in the qat leaves. Macgregor measured the area of stained plaque, and the proportion of gingival margin in contact with plaque in 64 smokers and 64 non-smokers, matched for age and sex. In both sexes, smokers had significantly more plaque than non-smokers, and there was a trend towards increased plaque deposits with increasing cigarette consumption. They report higher plaque and bleeding indices in non-smokers although probing depths and attachment loss were greater in smokers. Young smokers diagnosed with aggressive forms of periodontitis were shown to have more affected teeth and a higher mean loss of periodontal attachment than non-smokers with these conditions.

The present study also indicated deterioration of periodontal condition among qat chewers and tobacco chewing compared to non-chewers patients (control). Gingival index (GI) and clinical attachment loss (CAL) were more prevalent among chewers than non-chewers. Clinical attachment loss (CAL), were more prevalent among qat chewers patients than tobacco chewers but gingival index (GI) more prevalent among tobacco chewers patients than qat chewers. The infiltrate present in

### TABLE (3) The mean and± SD of number of chronic inflammatory cells

<table>
<thead>
<tr>
<th>Number chronic inflammatory cells</th>
<th>Qat chewing</th>
<th>Tobacco chewing</th>
<th>Non chewing habit</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>92.48±7.2</td>
<td>96.12±6.78</td>
<td>55.36±6.48</td>
</tr>
</tbody>
</table>

![Fig. (7) Clinicl parameters](image1)

![Fig. (8) Number of chronic inflammatory cells](image2)
periodontal diseases contains mononuclear cells, mainly transmigrated mononuclear phagocytes and lymphocytes. Whereas T lymphocytes predominate in the established chronic lesion, the proportion of B cells and plasma cells increases with disease progression. Qat containing pesticides might be cytotoxic to periodontal fibroblasts and thus, exacerbate preexisting periodontal disease as well as impair periodontal reattachment. Nicotine metabolites can concentrate in the periodontium and their effects include the promotion of vasoconstriction, and the impairment of the functional activity of polymorphs and macrophages. The numbers of neutrophils in peripheral blood are also increased by tobacco use and their migration through capillary walls. In the present study there increased in number of chronic inflammatory cells in chewers patients groups samples compared to non-chewers patients samples but the number of chronic inflammatory cells more in tobacco chewers than qat chewers.

CONCLUSION

The present study revealed the following:

- Increased plaque index (PLI) in control group patients than test groups patients due to the self cleaning effect of chewing habits against dental plaque.
- Increased gingival index (PLI) in test groups patients more than control group patients due to the chemical degradation effects of chewing habits against periodontal tissues.
- Increased clinical attachment level (CAL) in test groups patients more than control group patients due to the mechanical degradation effects of chewing habits against periodontal tissues.
- Increased number of chronic inflammatory cells in test groups patients more than control group patients due to the chemical degradation effects of chewing habits against periodontal tissues.

REFERENCES


