Evaluation of the Effect of Ginger Supplementation on Stage 2 and Stage 3 Periodontitis in Type 2 Diabetes Mellitus Patients

Sara Hosny
Mai Shafik Attia
Amal Ali Ibrahim
Evaluation of the Effect of Ginger Supplementation on Stage 2 and Stage 3 Periodontitis in Patients with Type 2 Diabetes Mellitus

Sara Hosny a,*, Mai S. Attia b, Amal A. Ibrahim b

a Ministry of Health, Egypt
b Oral Medicine, Periodontology, Oral Diagnosis and Radiology, Faculty of Dental Medicine for Girls, Al-Azhar University, Cairo, Egypt

Abstract

Purpose: The current study aimed to examine the clinical effects of ginger supplementation on type 2 diabetics with stage 2 and stage 3 periodontitis, specifically probing depth, recession, and clinical attachment loss.

Patients and methods: Thirty people took part in the study. Two groups were formed from the participants (group A had only nonsurgical periodontal treatment and group B received nonsurgical periodontal treatment in addition to a ginger supplement).

Results: Between baseline and 12 weeks, there was a statistically significant decrease in each group’s maximum probing depth and clinical attachment loss. Between baseline and 8 weeks, there was no statistically significant difference in recession within each group. But between baseline and 12 weeks, there was a significant decline. There was no statistically significant difference between the study and control groups.

Conclusion: The adjunct use of ginger supplements in diabetic periodontitis patients as host modulation improved the treatment outcomes when used with nonsurgical periodontal treatment.

Keywords: Diabetes mellitus, Ginger, Periodontitis

1. Introduction

Periodontal diseases are a group of oral inflammatory diseases that affect the periodontium [1]. Periodontal disease is a serious oral health problem that affects 20%–50% of adults worldwide [2]. Gingivitis is an acute, reversible inflammation of the gingival tissues that can progress to periodontitis, a more advanced, irreversible, and destructive form of periodontitis if left untreated [3].

Periodontitis, a multifactorial disease, is caused by dysbiosis of the oral microbiota and pro-inflammatory events involving both cells and mediators of innate and adaptive immunity. Its main symptoms are loss of periodontal attachment and progressive destruction of the tooth-supporting apparatus [4,5]. Clinical signs of periodontitis include increased probing depth, increased plaque index, reduced clinical attachment level, bleeding on probing, and apparent bone loss [6].

Diabetes mellitus is a heterogeneous group of disorders characterized by hyperglycemia due to an absolute or relative deficit in insulin production or action. Diabetes' chronic hyperglycemia has been related to end organ damage and dysfunction of many organs, particularly the retina, kidneys, nervous system, heart, and blood vessels. Numerous pathogenic processes can result in diabetes. These can range from abnormalities leading to insulin resistance to autoimmune destruction of pancreatic cells, which results in insulin deficiency. Due to insulin's ineffective action on target tissues, diabetes results in abnormalities in the metabolism of carbohydrates, fats, and proteins [7].

The great majority of diabetes cases can be classified into one of two etiopathogenetic categories. One type of diabetes, type 1 diabetes (T1DM), is
induced by a complete lack of insulin secretion. T2DM, which is much more prevalent, is brought on by insulin resistance and an inadequate compensatory insulin secretory response. The latter category includes hyperglycemia, which is severe enough to affect various target organs pathologically and functionally [7].

Diabetic patients are 2–3 times more liable to have periodontitis than nondiabetic patients, and glycemic control is a major element in finding risk. Poor glycemic control increases the risk of periodontitis, as does the risk of other diabetes complications. Much research is being done on the pathogenic processes that link the two diseases, and mostly the upregulation of inflammation in each of the diseases adversely affects the other [8].

As diabetes progresses, there is an increase in the deposition of advanced glycation end-products in periodontal tissues. Interaction of AGEs with their receptors (RAGEs, mainly on macrophages) triggers local immune and inflammatory responses [9]. As a result of these upregulated responses, increased cytokine production as in interleukin-1 (IL-1), tumor necrosis factor (TNF), and IL-6 occurs. An increase in oxidative stress occurs as well as a disturbance in the receptor activator of the NF-KB ligand/osteoprotegerin (RANKL/OPG) pathway that leads to bone loss [10].

Studies have demonstrated that nonsurgical periodontal therapy (NSPT), which entails oral hygiene instruction, scaling, and root debridement, lowers bacterial challenge and inflammation and may help diabetic patients regain their insulin levels as well as people with periodontal disease [11]. Ginger (Zingiber officinale) is a medicinal plant that is used in many different countries. Bisabolene, shogaol, gingerol, and paradol are some of the active ingredients in ginger; they control blood glucose levels and show anti-inflammatory, antioxidant, anticancer, and anti-obesity properties [12].

In diabetics, ginger can enhance glycemic control and decrease inflammation markers (TNF-, IL-1, IL-6) [12] and increase antioxidant enzymes [superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GPx)] [13].

2. Patients and methods

This study was a randomized clinical trial on 30 patients with T2DM patients stage 2 and stage 3 periodontitis according to the new classification, aged between 30 and 60 years. The patients were selected from the outpatient clinic of the Oral Medicine, Periodontology and Diagnosis Department, Faculty of Dental Medicine Al-Azhar University. Patients were selected according to the following criteria (the patients were free from any systemic disease (other than diabetes) or autoimmune diseases, had no history of allergy or hypersensitivity reaction, nonsmokers, nonpregnant women).

The protocol of the study will be clarified to each patient and after that an informed consent will be signed by each participating patient. Research Ethics Committee approval of the Faculty of Dental Medicine was obtained (REC-ME-22-11).

2.1. Sample size calculator

We used Steven K. Thompson’s equation to calculate the sample size, from the formula

$$n = \frac{N \cdot p \cdot (1 - p)}{\left[ N \cdot p \cdot (1 - p) \right] + p \cdot (1 - p)}$$

where n is the sample size (27.8), N the population size (30), Z the confidence level at 95% (1.96), d the error proportion (0.05), and p is the probability (50%) (Table 1).

2.2. Patient grouping

Patients were divided randomly into two groups. All groups received nonsurgical periodontal therapy (baseline and after 4 weeks).

Group A (16 patients): The first group is the control group, which received only nonsurgical periodontal treatment.

Group B (14 patients): The study group received herbal ginger in the form of oral capsules 2 g/day (4 tablets of 500 mg as two tablets before lunch and dinner) for 8 weeks in addition to the nonsurgical periodontal treatment.

2.3. Clinical evaluation

Clinical findings were reported at baseline, 8 weeks, and at 12 weeks.

2.4. Probing depth

Probing depth is measured from the gingival margin to the sulcus base at 6 points (midbuccal, midlingual, mesiobuccal, mesiolingual, distobuccal, distolingual).

<table>
<thead>
<tr>
<th>Group (A)</th>
<th>Group (B)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size calculator</td>
<td>14</td>
<td>13.8</td>
</tr>
</tbody>
</table>

Table 1. Sample size distribution.
and distolingual) around each tooth. It was obtained by measuring the probing pocket per site using a William periodontal probe with the following graduation (1, 2, 3, 5, 7, 8, 9, and 10 mm) as shown in Fig. 1.

2.5. Gingival recession

It was obtained by recording the distance between the cementoenamel junction and gingival margins using a periodontal probe at 6 points (midbuccal, midlingual, mesiobuccal, mesiolingual, distobuccal, and distolingual) around each tooth.

2.6. Clinical attachment loss (CAL)

It was obtained by measuring the distance from the cementoenamel junction to the pocket base using a William periodontal probe at 6 points (midbuccal, midlingual, mesiobuccal, mesiolingual, distobuccal, and distolingual) around each tooth.

Statistical analysis was performed by applying the Kruskal–Wallis test followed by the Mann–Whitney test for pairwise comparisons between different groups.

3. Results

3.1. Maximum probing depth (Max PD)

The study group achieved the highest percentage change after 8 weeks and 12 weeks, 28.83% and 41.99%, respectively. There was no statistically significant difference between the two groups at the two time intervals (Table 2).

3.2. Clinical attachment loss

The study group achieved the highest percentage change after 8 weeks and 12 weeks, 28.51% and 43.20%, respectively. There was no statistically significant difference between the two groups at the two time intervals (Table 3).

3.3. Gingival recession

Although the study group achieved the highest percentage change after 8 weeks and 12 weeks, 13.81% and 25.53%, respectively, there was no statistically significant difference between the two groups at the two time intervals (Table 4).

4. Discussion

Periodontitis is the sixth chronic disorder that affects more than 743 million people worldwide and has an adverse effect on an individual’s oral capacity, self-assurance, fundamental well-being, and general prosperity. Recently, some systemic disorders and syndromes have been associated with a rise in immune system cell function and the progression of clinical periodontal conditions [14].

Hyperglycemia is a symptom of diabetes, either insulin secretion, insulin action, or both have contributed to it. Organ dysfunction and failure are associated with chronic hyperglycemia [15].

Diabetes patients are more likely to exhibit periodontitis than nondiabetics, and glycemic control is

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Table 2. Percentage of change of maximum probing depth for the two groups at different time intervals.

<table>
<thead>
<tr>
<th></th>
<th>8 Weeks</th>
<th>12 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>28.77%</td>
<td>36.99%</td>
</tr>
<tr>
<td>Study</td>
<td>28.83%</td>
<td>41.99%</td>
</tr>
<tr>
<td>P value**</td>
<td>0.144\textsuperscript{NS}</td>
<td>0.258\textsuperscript{NS}</td>
</tr>
</tbody>
</table>

NS = nonsignificant (P > 0.05) **Overall P-value or intergroup comparison.

Table 3. Percentage of change of clinical attachment loss for the two groups at different time intervals.

<table>
<thead>
<tr>
<th></th>
<th>8 Weeks</th>
<th>12 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>24.63%</td>
<td>33.74%</td>
</tr>
<tr>
<td>Study</td>
<td>28.51%</td>
<td>43.20%</td>
</tr>
<tr>
<td>P value**</td>
<td>0.540\textsuperscript{NS}</td>
<td>0.890\textsuperscript{NS}</td>
</tr>
</tbody>
</table>

NS = nonsignificant (P > 0.05) **Overall P value or intergroup comparison.

Table 4. Percentage of change of Recession for the two groups at different time intervals.

<table>
<thead>
<tr>
<th></th>
<th>8 Weeks</th>
<th>12 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>13.303%</td>
<td>23.76%</td>
</tr>
<tr>
<td>Study</td>
<td>13.81%</td>
<td>25.53%</td>
</tr>
<tr>
<td>P value**</td>
<td>0.981\textsuperscript{NS}</td>
<td>0.984\textsuperscript{NS}</td>
</tr>
</tbody>
</table>

NS = nonsignificant (P > 0.05) **Overall P value or intergroup comparison.
a major factor in determining risk. Periodontitis, as well as other diabetes complications, is increased by poor glycemic control. Much research is being done on the pathologic processes that connect the two diseases, and it is likely that increased inflammation in one has a negative impact on the other [8].

Periodontitis treatment's main goal is to reduce the bacterial challenge [10]. NSPT involves improving oral hygiene and removing plaque and calculus from affected root surfaces with subgingival scaling and root debridement [16]. The formation of a biofilm on the tooth/root surface causes periodontal inflammation, but it is insufficient to cause periodontal tissue damage. Periodontal tissue destruction is caused by the aggravated host inflammatory-immune response to this dysbiotic microbial challenge [17].

As a result, periodontal disease treatment with host modulation therapy (HMT) has become a novel strategy. By modifying or downregulating destructive components of the host response while upregulating protective or regenerative responses, HMT targets the decrease in tissue destruction and stabilizes or even regenerates the periodontium [18]. HMT refers to pharmaceuticals introduced either in a systemic or local pathway as a part of the treatment plan [19]. Many herbal formulations have been shown in the literature to control the production of pro-inflammatory cytokines, allowing them to manage a variety of inflammatory processes [20].

Ginger belongs to the Zingiberaceae family (Z. officinale Rosc.). It is a spice and condiment that originated in Southeast Asia and is now used in a variety of countries [21]. Ginger is well known for its anti-inflammatory and antioxidant properties. It also has antimicrobial properties, which can aid in treating infectious diseases [22]. In this study, 2 g oral supplementation of ginger was used daily for 8 weeks, which was the same used in the study by Zare Javid et al. [11] (2019) and Hasan Gholinezhad et al. (2020) [23].

The results of this study showed that regarding PD, both the study and control groups achieved the highest mean of the mentioned parameter before the start of therapy (baseline), while the lowest mean of CAL was achieved before the start of therapy (baseline), and the overall p-value was highly significant. After ginger therapy, the study achieved the highest percentage change after 8 weeks and 12 weeks.

The results were in agreement with the study performed by Zare Javid et al., 2019 [11]. The study was carried out on 46 T2DM patients with periodontitis, who were randomly allocated to intervention and control groups and received either 4 tablets of 500 mg (2 g) ginger or placebo twice a day for 8 weeks. All patients were treated with NSPT during the intervention period. Periodontal indices including CAL, bleeding on probing, pocket depth (PD), and plaque index were evaluated in all subjects pre- and post-intervention. The results of this study reported that consumption of ginger supplements for 8 weeks along with NSPT significantly reduced the mean of PD and CAL after the intervention. Also, in the control group, NSPT reduced the periodontal parameters, but it was not significant, at the end of the study. There was a significant difference in the mean changes of CAL and PD between the intervention and control groups.

In another study performed by Hasan Gholinezhad et al., 2020 [23], 50 T2DM patients with periodontitis were randomly allocated to intervention and control groups and received either 2 g ginger or a placebo (4 tablets) twice a day for 8 weeks. All patients underwent NSPT during the intervention period. CAL, PD, plaque index, and bleeding on probing were measured in all subjects at baseline and postintervention. There were significant differences observed in the mean changes of CAL and PD between intervention and control groups after the intervention.

4.1. Conclusion

The adjunct use of ginger supplements in diabetic periodontitis patients as host modulation improved the treatment outcomes when used with nonsurgical periodontal treatment.

4.2. Recommendation

Further study of the effect of oral ginger supplement and its effect on periodontitis and diabetes mellitus patients conducted over a longer period and with a larger sample is needed.
Funding
No funding was received for this research.

Conflicts of interest
The authors declare that they have no conflict of interest.

References