

ORIGINAL RESEARCH—ERECTILE DYSFUNCTION

Is There a Relationship Between Chronic Periodontitis and Erectile Dysfunction?

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DOI: 10.1111/j.1743-6109.2012.02974.x

ABSTRACT

Introduction. Chronic periodontitis (CP) is characterized with inflammation of the gingival tissues, which causes endothelial dysfunction in different organs.

Aim. In this study, we investigated the association of CP with the erectile dysfunction (ED).

Methods. The study group included 80 male patients with ED and 82 male patients without ED (control), aged between 30 and 40 years. The International Index of Erectile Function (IIEF) questionnaire was used to assess male sexual function, particularly the presence or absence of ED.

Main Outcome Measures. The patients in the study and control groups were statistically compared according to their plaque index (PI), bleeding on probing (BoP), probing depth (PD), and clinical attachment level (CAL).

Results. In the non-ED and the ED groups, the mean age was 35.7 ± 4.8 and 34.9 ± 4.9 years, respectively. Patients' characteristics including body mass index, household income, and education status were similar in both groups ($P > 0.05$). Nineteen patients (23%) had severe CP in the non-ED group; 42 patients (53%) had severe CP in the ED group. Logistic regression analysis showed a significantly high association between ED and the severity of CP (odds ratio: 3.29, 95% confidence interval: 1.36–9.55, $P < 0.01$). The mean values of PI, BoP, and the percentages of sites with PD >4 mm and sites with CAL >4 mm were significantly higher in the ED group than in the control group ($P < 0.05$). The mean values of PD and CAL were not significantly different in the two groups ($P > 0.05$). The decayed, missing, filled teeth scores were also significantly higher in the ED group than in the non-ED group ($P < 0.05$).

Conclusion. Our results have suggested that CP had a high association with ED in young adults at 30–40 years. We think that it will be of benefit to consider periodontal disease as a causative clinical condition of ED in such patients. **Oğuz F, Eltas A, Beytur A, and Akdemir E. Is there a relationship between chronic periodontitis and erectile dysfunction? J Sex Med 2013;10:838–843.**

Key Words. Erectile Dysfunction; Chronic Periodontitis; Endothelial Dysfunction; Periodontal Disease; Systemic Vascular Disease

Introduction

Erectile dysfunction (ED) is described as a persistent or recurrent inability to have sufficient erection for satisfactory sexual performance [1]. It is a multifactorial condition that is estimated to affect more than 150 million men worldwide [2,3].

It is considered a major public health problem, which seriously affects the quality of life of patients and their partners [4].

ED may result from organic or psychological reasons, or their combination [5]. An organic pathology is found about 65% of ED cases, and vascular dysfunction is the most common cause of ED

[6]. Additionally, emotional stress and depression may cause ED without any vascular failure [7].

Chronic periodontitis (CP) is a group of infectious diseases caused predominantly by anaerobic and microaerophilic bacteria [8,9]. They most often occur with inflammation of the gingival tissues and may follow on the loss of both attachment of the periodontal ligament and the bony support of the tooth [10]. Many studies have reported that CP may induce systemic vascular diseases such as coronary heart disease, cerebrovascular disease, and chronic obstructive pulmonary disease, via inducing endothelial dysfunction [11–13].

Some investigators showed that there was a high prevalence of CP in the patients with ED [14]. In a nationwide study including men at different age groups, a strong relationship between the ED and CP was reported [15]. However, the impact of severity and the particular type of CP on ED is unknown. Therefore, in this case-control study, we aimed to investigate the possible relationship between the severity of ED and CP and to establish the correlation between clinical periodontal parameters and ED.

Materials and Method

Study Population

The present study was designed as a single-blinded, randomized-controlled clinical trial. The study group included 80 male patients with ED and 82 male patients without ED (control) who were admitted to the Urology Department. Before enrollment, informed consent was obtained from all patients. The study protocol was approved by the human ethics committee of Inonu University.

The inclusion criterion for the ED group was presentation with complaints of ED and for the non-ED group (control), presentation without any symptom of ED. All the patients in both groups were aged between 30 and 40 years. After establishing the diagnosis of ED, the patients were referred to the periodontology department for evaluation of their periodontal status.

Criteria for exclusion from the study were the following: the presence of a systemic disease (diabetes mellitus, heart disease, hypertension, etc.), which can affect periodontal health or ED; undergoing periodontal therapy within the last 12 months or taking any systemic antibiotics within the last 6 months; and smoking.

Sample Size

The sample size calculation determined that each group was to be composed of 40 subjects, and the test was calculated to provide 80% power ($\alpha = 0.05$) to detect a difference of 39% between the groups (52% severe periodontal disease [SPD] in the ED group and 23% SPD in the non-ED group). The power of this test was calculated as 92%.

International Index of Erectile Function (IIEF)

Questionnaire

The IIEF is a simple and suitable questionnaire for screening patients with ED. The questions from 1 to 5 and question 15 were used to assess the existence of ED. Subjects with a score higher than 30 were considered to have normal erectile function and those with a score lower than or equal to 25 were considered to have ED. To ensure that the study subjects truly had either completely normal or impaired erectile function, subjects with IIEF score of 26–29 were excluded. The IIEF questionnaire was used to assess male sexual function, particularly the presence or absence of ED. Those who reported very low to medium erectile confidence were considered to have ED, and those who reported high erectile confidence were excluded. The questionnaires were administered in a standard manner, with the initial explanation given by the same clinician to all participants [16].

Periodontal Examination

Each patient underwent a comprehensive periodontal examination. The examination involved assessing the plaque index (PI), bleeding on probing (BoP), probing depth (PD), and clinical attachment level (CAL). The measurement of the state of oral hygiene by PI is based on recording both soft debris and mineralized deposits on the following teeth. BoP that is also known as bleeding gums or gingival bleeding is a sign of inflammation. PD was measured as the distance between the gingival margin and the deepest aspect of the pocket. CAL was measured as the distance between the cemento-enamel junction of the tooth and the deepest aspect of the pocket. Full-mouth bleeding scores were recorded as the presence or absence of bleeding after measurement of PD. All periodontal measurements were taken on six surfaces per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and either the distolingual or the distopalatal surface) in all teeth except the third molars using a Williams probe (PCP-12, Hu-Friedy, Chicago, IL, USA) [17,18].

The periodontal condition was stratified according to the criteria used by Boggess et al. [19]. Periodontal health was defined as the lack of PD ≥ 4 mm. Mild periodontal disease was defined as 1–15 tooth sites with ≥ 4 -mm PD and BoP. Severe periodontal disease was defined as ≥ 15 tooth sites with ≥ 4 -mm PD and BoP.

The examiner recorded the teeth that were decayed, missing, and filled, according to the criteria of the World Health Organization's decayed, missing, filled teeth (DMFT) index.

Periodontal examinations were performed by one previously calibrated periodontist (M.Ö.U.) who had no knowledge about the ED status of patient. Before the start of the study, the examiner was trained to ensure adequate levels of accuracy and reproducibility in recording the clinical parameters and indices.

Statistical Analysis

These results were analyzed using a statistical package (SPSS Statistical Package for the Social Sciences software, V-17.00, Chicago, IL, USA). A descriptive analysis was conducted (mean, standard deviation, and frequency distribution) for the collected data. The Shapiro–Wilk tests were computed for each variable to assess whether the variables were distributed normally. Differences among the groups were determined by the *t*-test (normal distribution). Correlations between the clinical parameters and ED were determined by the Pearson chi-square test. A *P* value < 0.05 was considered statistically significant. Logistic regression analysis was used to determine the association between the severity of periodontal disease and ED. From the logistic-regression analysis, odds ratios (ORs) were calculated with a 95% confidence interval (CI).

Results

The characteristics of both groups are presented in Table 1. In the non-ED group, there were 82 men,

Table 1 Demographic characteristics of participants for both groups

	Non-ED (N = 82)	ED (N = 80)
Age	35.7 \pm 4.8	34.9 \pm 4.9
Age range	30–40	30–40
Body-mass index (kg/m ²)	26.1 \pm 1.9	26.3 \pm 2.1
Education (years)	12.8 \pm 2.1	13.4 \pm 2.2
Household incomes (\$) per month	643 \pm 56	675 \pm 61

ED = erectile dysfunction

Table 2 The compared groups of clinical variables

Clinical parameters	Non-ED	ED	<i>P</i> values
% sites with plaque	43.7 \pm 15.2	65.7 \pm 17.3*	0.0032
% sites exhibiting BoP	38.9 \pm 16.6	59.8 \pm 16.5*	0.0029
Mean PD (mm)	3.21 \pm 0.39	3.34 \pm 0.49	N.S.
% sites with 4 mm <PD	11.94 \pm 2.7	22.68 \pm 4.8**	0.0008
Mean CAL (mm)	3.54 \pm 0.41	3.72 \pm 0.54	N.S.
% sites with 4 mm <CAL	16.1 \pm 3.34	35 \pm 6.47**	0.001
DMFT	4.61 \pm 1.65	7.86 \pm 1.9*	0.0038
SPD (%)	23	53*	0.0035

P* < 0.01; *P* < 0.001; *P* values represent difference between groups.

BoP = bleeding on probing; PD = probing depth; CAL = clinical attachment level; DMFT = decayed, missing, filled teeth; SPD = severe periodontal disease; N.S. = nonsignificant; ED = erectile dysfunction

and their mean age was 35.7 \pm 4.8 years, ranging from 30 to 40 years. In the ED group, there were 80 men, and their mean age was 34.9 \pm 4.9 years, ranging from 30 to 40 years. The mean age, body mass index (BMI), household income, and education status were similar in both groups (*P* > 0.05). None of the subjects smoked, and all the patients were married.

Nineteen subjects (23%) had severe PD in the non-ED group; 42 (53%) had severe CP in the ED group (Table 2). Logistic regression analysis showed a highly significant association between ED and the severity of CP (OR: 3.29, 95% CI: 1.36–9.55, *P* < 0.01). After adjusting for potential confounders (age, BMI, household incomes, and education level), subjects with severe CP were 3.29 (1.36–9.55) times more likely to present with ED than periodontal healthy men. The mean clinical variables for both groups are shown in Table 2. The mean values for PI, BoP, and the percentages of sites with PD >4 mm and sites with CAL >4 mm were significantly higher in the ED group than in the non-ED group (*P* < 0.05). The mean values for PD and CAL were not significantly different in the two groups according to the statistical results (*P* > 0.05). The DMFT scores were also significantly higher in the ED group than in the non-ED group (*P* < 0.05).

The correlations between periodontal parameters and ED were analyzed by Pearson's chi-square test (Table 3). Positive correlations between DMFT, PI, BoP, and the percentage of sites with PD >4 mm with ED and the percentage of sites with CAL >4 mm with ED were identified (*P* < 0.05). There was no correlation found between the mean PD and CAL with ED (*P* > 0.05).

Discussion

The term “endothelial dysfunction” is used to refer to various pathological conditions, including

Table 3 The correlation between the ED and periodontal characteristics of participants

	% sites with plaque	% sites exhibiting BoP	Mean PD (mm)	% sites with 4 mm <PD (%)	Mean CAL (mm)	% sites with 4 mm <CAL (%)	DMFT
ED (<i>R</i> coefficient)	0.162*	0.187*	0.083	0.224**	0.113	0.314**	0.271**
<i>P</i> values	0.04	0.04	N.S.	0.007	N.S.	0.01	0.009

*Correlation is significant ($P < 0.05$).

**Correlation is significant ($P < 0.01$).

BoP = bleeding on probing; PD = probing depth; CAL = clinical attachment level; DMFT = decayed, missing, filled teeth; N.S. = nonsignificant; ED = erectile dysfunction

altered anticoagulant and anti-inflammatory features of the endothelium, impaired modulation of vascular growth, and dysregulation of vascular remodeling. The well-known risk factors for atherosclerosis can predispose to endothelial dysfunction [20].

Erection is a neurovascular phenomenon that ultimately culminates in an increase of arterial flow within the hypogastric-penile bed, followed by the activation of the veno-occlusive mechanism of the corpora cavernosa in a hormonal environment under psychological control [21,22]. The ED may result not only from the occlusion of the cavernosal arteries by atherosclerosis but also from the impairment of endothelial function/smooth muscle relaxation [23,24]. It is characterized by obstructive vascular changes, leading to vascular pathology. Endothelial dysfunction is the first step of vascular pathology [25].

In this study, we aimed to determine the relationship between the chronic periodontal disease and ED, with considering the severity of the dental disease. The findings of the present study support the hypothesis that CP identified by the presence of PD >4 mm and CAL >4 mm and the increased percentage of sites with BoP is associated with an increased risk for ED. Men with severe CP were 3.29 times more likely to present with ED than periodontal healthy men.

To our knowledge, ED and CP in humans are caused by the similar risk factors, such as aging, smoking, diabetes mellitus, and coronary artery disease. Therefore, we excluded the men who had systemic disease and smokers from this study. We particularly selected the men aged between 30 and 40 years to assess the impact of CP on ED without taking into account the potential effect of aging.

Zadik et al. [26] reported that CP was significantly more prevalent among men with ED when compared with men without ED, and they suggested that ED might be associated with CP. In the other study, Sharma et al. [27] found that the prevalence of CP among patients with ED was highest for men who had severe ED (81.8%). In other

words, they reported that CP and vasculogenic ED were correlated with each other positively. Despite the presence of these data, information about the potential role of CP in the pathophysiology of ED remains scarce. Therefore, this study was aimed at shedding further light on this issue.

The periodontitis-induced systemic inflammation contributes to the development and maintenance of atherosclerosis through activation of a biochemical reaction cascade, initiation and development of plaque formation, and injury of the endothelium [28]. Blum et al. [29] suggested that treating periodontitis may improve endothelial function, reverse endothelial dysfunction in patients with severe periodontitis, and prevent future cardiovascular disease.

The results of our study support the idea that CP is present more often in patients with ED than in those without ED. They also suggest that CP may increase endothelial dysfunction. Three mechanisms have been proposed to explain this relationship. First, chronic inflammation in endothelial dysfunction is associated with an increase in reactive oxygen species [30–32]. Excess production of reactive oxygen species leads to an increase in nitric oxide (NO) inactivation, and the damage to the antioxidant system may contribute to endothelial dysfunction in patients with periodontitis [28]. Second, high levels of inflammatory mediators such as tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), IL-8, and IL-18 may be associated with increased risk of endothelial dysfunction [33–35], with the result that levels of inflammatory markers are also increased in subjects with CP [36]. Third, periodontal pathogens or their products could affect endothelial function directly [37]. In a rat model, Zuo et al. [38] have reported that the decrease in the expression of endothelial NO synthase (NOS) and NOS activity in penile cavernous tissue caused by mild systemic inflammatory status in periodontitis may be one of the important risk factors of ED. Though endothelial dysfunction was not assessed in this study, we measured BoP as an indicator of clinical inflamma-

tion. The higher level of BoP seen in the ED group in our study compared with the control group and the correlation between BoP and ED support the earlier reports that a high level of infection results in ED due to endothelial dysfunction.

Our findings relating to the percentage of PD and CAL sites >4 mm, the DMFT scores in the ED group compared with the control group, and the correlation between ED and these parameters support the relation between ED and CP. These results support the relation between ED and CP. Alterations in these parameters are due to the response of the host to periodontal infection resulting from bad oral hygiene.

We have some limitations in this study. Although CP is thought to cause ED via endothelial dysfunction, we did not evaluate endothelial dysfunction. We also did not assess the severity of ED or the underlying cause.

Conclusion

This study is the first to demonstrate the associations between clinical periodontal parameters and ED, and it suggested that periodontal inflammation can be associated with ED. Periodontal diseases must be considered in the etiology of ED in young adults.

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Conflict of Interest: None declared.

Statement of Authorship

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