

Original Article

Comparison of prevalence of periodontal disease in women with polycystic ovary syndrome and healthy controls

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among women of reproductive age, affecting 4-18% of them. Previous studies also showed that periodontal diseases are associated with different components of the metabolic syndrome. The aim of this study is to determine the association between PCOS and periodontal diseases.

Materials and Methods: A total of 196 women (98 with PCOS and 98 healthy controls) were enrolled. PCOS diagnosis was confirmed by history, clinical signs, physical examination, laboratory parameters, and ultrasound studies. Both cases and controls were examined by the same periodontist. Periodontal parameters including bleeding on probing (BOP), probing depth, clinical attachment loss (CAL), plaque index, and tooth loss were investigated in all participants. Pregnant women, smokers, individuals with a history of malignancy or osteoporosis, and those taking prophylactic antibiotics for dental procedures or receiving periodontal treatment during the 6-month period before examination were excluded. Data were analyzed using t-test, Chi-square test, and linear regression. Statistical significance was set at $P < 0.05$.

Results: CAL and sites with BOP were significantly higher in women with PCOS ($P < 0.05$). However, no significant difference was observed in the tooth loss rate between PCOS and non-PCOS participants ($P = 0.384$).

Conclusion: The prevalence of periodontal disease seems to be higher in women with PCOS. This may be related to the role of chronic systemic inflammation in the pathophysiology of both PCOS and periodontal diseases.

Key Words: Dental plaque index, menstrual disturbance, periodontal diseases, polycystic ovary syndrome

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among women of reproductive age, affecting 4-18% of them.^[1,2] In addition to reproductive derangements, patients with

PCOS may develop other metabolic and psychological comorbidities.^[3] Classically, PCOS is characterized by the presence of menstrual abnormalities (oligomenorrhea or amenorrhea), chronic anovulation

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or oligoovulation, clinical/biochemical evidence of hyperandrogenism (hirsutism, acne, or androgenic alopecia), and ultrasound findings.^[4] Patients with this gender-specific form of metabolic syndrome are at higher risk for developing insulin resistance (IR), obesity, dyslipidemia, cardiovascular disease (CVD), and hypertension.^[5,6] Moreover, recent studies have shown the higher prevalence of impaired glucose tolerance (IGT), type II diabetes mellitus (DM), and lipid profile disturbances in women with PCOS.^[7-9]

Periodontal diseases are chronic inflammatory processes that may lead to tooth loss by affecting tooth-supporting tissues, including the gingiva, alveolar bone, and periodontal ligaments. In addition to the role of bacterial infections, earlier studies have demonstrated the association of periodontal diseases and systemic conditions such as dyslipidemia, obesity, IR, DM, and CVD.^[10-14]

Previous studies showed higher levels of oxidative stress and systemic inflammatory markers such as interleukin-6 and C-reactive protein in both periodontal diseases and PCOS.^[14-16] This association may be explained by the role of oxidative stress as the potential link between periodontitis and PCOS based on some recent studies.^[17,18] This might suggest a possible common pathophysiologic mechanism in concur manifestations of these conditions. In this present study, we aimed to compare the prevalence of periodontal disease in women with PCOS and healthy controls.

MATERIALS AND METHODS

In a cross-sectional study (December 2012 to August 2013), a total of 196 women, aged 18-45 years old, visiting the clinics of Fatemeh Women's Hospital, Hamadan, Iran, were enrolled after providing adequate explanation and obtaining informed consent. The study protocol was approved by the Institutional Review Board of Hamadan University of Medical Sciences, Hamadan, Iran. A unique questionnaire containing past medical history, habitual history, and anthropometric parameters was completed for all subjects consisting 98 women with PCOS and 98 systematically healthy subjects. PCOS diagnosis was made based on history, clinical signs, physical examination, laboratory parameters, and ultrasound findings, known as the Rotterdam criteria.^[4] Fasting blood glucose (FBS), high density lipoprotein (HDL), low density lipoprotein (LDL) cholesterol, triglyceride (TG), testosterone, and serum

nitric oxide (NO) levels were assessed in both groups. In order to exclude, other conditions including thyroid diseases, hyperprolactinemia, Cushing's syndrome, androgenic tumors, 21-hydroxylase deficiency, thyroid stimulating hormone, and prolactin were measured when it was appropriate.

Pregnant women, smokers, individuals with history of alcoholic drinks consumption, malignancy, osteoporosis, those took prophylactic antibiotics for dental procedures, and patients who received periodontal treatment during the 6-month period before examination were excluded.

Due to the effects of obesity and IGT on periodontal diseases, PCOS patients who had a body mass index (BMI) >25 or IGT were also excluded.^[19,20]

Both groups of participants underwent examinations by a single investigator using periodontal probe and mirror to determine the periodontal parameters at the Periodontics Clinic of Hamadan University of Medical Sciences. The periodontal parameters included bleeding on probing (BOP), clinical attachment loss (CAL), probing depth (PD), and plaque index (PI).

In order to minimize the effects of hormonal changes during the menstrual cycle on the status of periodontal tissue, the examinations of all the subjects were carried out in the early follicular phase of the menstrual cycle.^[21-24]

The BOP, PD, and CAL were calculated at six sites on each tooth except for the third molars. The BOP was assessed based on NIDCR protocol and after drying gums with dental compressed air. This index was reported in percentage.^[25] According to the classification proposed by the American Academy of Periodontology, PD was calculated in percentages and based on involvement of the gums around the tooth surfaces. The involvement of more than 30% and <30% of all surfaces were described as generalized periodontitis and localized periodontitis, respectively.^[25,26] CAL was determined by measuring the distance from the bottom of the periodontal pocket to the cemento-enamel junction using a periodontal probe. This index was used for describing the severity of periodontitis (slight: 1-2 mm of CAL; moderate: 3-4 mm of CAL; and severe >5 mm of CAL).^[27,28]

To calculate the PI, all participants rinsed for 30 s with a disclosing solution.

The O'Leary index consisted of recording the presence or absence of disclosed plaque on the

mesial, distal, buccal, and lingual surfaces of all teeth. The percentage of disclosed plaque was then calculated for each participant. When 10% or less of the surfaces contain plaques, a reasonable periodontal health exists.^[29]

Statistical analysis

Statistical analysis was done using SPSS software version 20.0 (SPSS Inc., Chicago, IL, USA). For all variables, the Kolmogorov–Smirnov test was used to assess normality. For normally distributed variables, independent sample *t*-tests were applied, and data were presented in mean \pm standard deviation. The Mann–Whitney U-test was applied for nonnormally distributed variables. Pearson's Chi-square test was used to compare qualitative data. If the data in a group were <5 , Fisher's exact test was used. The relationship between PCOS and CAL was investigated using univariate analysis of variance and by controlling for confounding variables such as BMI and PI. Statistical significance was defined at $P < 0.05$.

RESULTS

The mean age in the systematically healthy control group and patient with PCOS group were 29.1 ± 6.6 years (ranging 19-45 years) and 28.6 ± 6.4 years (ranging 19-45 years), respectively. Clinical characteristics and periodontal parameters of both groups are summarized in Table 1. BMI, FBS, TG, LDL, HDL, testosterone, and serum NO levels were not significantly different between two groups. Although two groups were matched, the number of women suffering from hirsutism in the case group was significantly higher ($P < 0.001$).

The mean number of missing teeth in the PCOS group was 2.2 ± 1.9 (range: 0-7). In the non-PCOS group, the mean number of missing teeth was 2.0 ± 1.7 (range: 0-8) which was not statistically different from the PCOS group ($P = 0.384$).

Women with PCOS had significantly higher PI as compared with non-PCOS group ($41.1 \pm 14.2\%$ vs. $32.8 \pm 11.3\%$, $P = 0.0001$) in the PCOS and non-PCOS groups, respectively, which indicated a significantly higher rate of PI in women with PCOS.

The BOP in the case and control groups was $3.0 \pm 2.6\%$ and $1.4 \pm 1.8\%$, respectively, which showed higher rate of BOP in women suffering from PCOS ($P = 0.0001$).

CAL in the PCOS group was 2.2 ± 0.4 mm. The mean CAL for the control group was 2.0 ± 0.3 mm. Mann–Whitney U-test indicated a significant difference between CAL between study groups ($P = 0.001$). Although univariate analysis of variance with regard to confounding factors such as PI and BMI showed that CAL as the main indicator of periodontitis in the two groups was not statistically significant (adjusted $R^2 = 0.408$ $P = 0.201$), this finding reflected the impact of confounding variables.

In the PCOS group, 92 subjects (93.9%) were affected by slight periodontitis and 6 patients (6.1%) were diagnosed with moderate periodontitis. Slight periodontitis was observed in 97 subjects (99%) in the control group while only one subject (1%) was reported to have moderate periodontitis. Fisher's exact test failed to reveal any significant difference between these groups in terms of periodontitis severity. The detailed results of periodontal examination are presented in Table 2.

DISCUSSION

Very few studies are focused on the periodontal parameters in women suffering from PCOS.^[24,30] Our results revealed higher prevalence of periodontal disease parameters in PCOS women comparing to systematically healthy controls who were matched in terms of possible confounding variables such as age

Table 1: Clinical characteristics and periodontal parameters of the PCOS and control groups

Parameters	PCOS (n = 98)		Control groups (n = 98)		P
	Mean \pm SD	95% CI (lower–upper bound)	Mean \pm SD	95% CI (lower–upper bound)	
BMI*	23.81 \pm 4.10	22.98-24.63	24.02 \pm 3.50	23.31-24.72	0.928
Age	29.06 \pm 6.56	27.74-30.38	28.60 \pm 6.37	27.32-29.88	0.506
AL* (mm)	2.16 \pm 0.40	2.08-2.24	1.96 \pm 0.33	1.89-2.02	0
PI %*	41.10 \pm 14.24	38.25-43.96	32.81 \pm 11.30	30.54-35.07	0
BOP %*	3.01 \pm 2.56	2.50-3.52	1.47 \pm 1.81	1.11-1.83	0
Tooth loss*	2.17 \pm 1.86	1.80-2.55	1.95 \pm 1.73	1.60-2.30	0.402

*Mann–Whitney U-test was used. AL, PI, BOP and tooth loss. AL: Attachment loss; PI: Plaque index; BOP: Percentage of bleeding on probing; SD: Standard deviation; CI: Confidence interval; PCOS: Polycystic ovary syndrome; BMI: Body mass index

Table 2: Periodontal disease severity in the PCOS and control groups

Group	Severity	n (%)	P
Slight	Case	92 (93.90)	0.118*
	Control	97 (99)	
Moderate	Case	6 (6.10)	
	Control	1 (1)	

*Fisher's exact test was used. Slight: 1-2 mm of AL, Moderate: 3-4 mm of AL, Severe >5 mm of AL. PCOS: Polycystic ovary syndrome; BMI: Body mass index; AL: Attachment loss

and BMI. These findings are compatible with Dursun *et al.*^[24] study that showed higher periodontal disease indexes including significant among women with PCOS.

The association of gingivitis with hormonal changes during puberty, pregnancy, and menstrual cycles has been studied well.^[31] Increased production of steroid hormones is associated with increased gingival inflammation. The effects of estrogen on the gingival epithelium, collagen synthesis, osteoblasts, and bony tissues are important factors in the development of periodontal disease. Estrogen and progesterone affect the capillary system, inflammation, and angiogenesis processes. These alterations lead to excessive proliferation of vascular endothelial cells and epithelial keratinization in gums.

The hyperandrogenism status in patients with PCOS not only results in menstrual abnormalities and infertility but also may pose an increased risk of periodontal diseases to these patients. Regarding the conversion of testosterone to estrogen in women with PCOS, the paradox of co-excising of high levels of estrogen and testosterone is appreciable.

The increased vulnerability of PCOS patients to periodontal diseases can be explained regarding the influence of altered circulating hormones in on periodontal tissues. These derangements impact gingival tissues through initiating changes in oral flora and pro-inflammatory cytokines. In turn, these changes adversely affect bones, adhesive joints and eventually lead to tooth loss.^[32] Furthermore, enhanced oxidative stress in affected periodontal tissues may participate in the pathology of PCOS by mechanisms such as increasing glucose intolerance and dyslipidemia.^[17]

Our analysis failed to show any significant difference in tooth loss rate and disease severity between study groups. Of note, periodontal disease and their subsequent complications such as tooth loss

and bone loss are chronic in nature.^[28] Our results may be due to relatively low age of PCOS patients (28.8 ± 6.5 years) in our study. Longstanding periodontitis and severe forms of disease accentuate the progression of adverse outcomes. Comparison of tooth loss index in an older study population including women with and without PCOS may reveal greater contribution to the assessment of this index. In a recent study, Porwal *et al.*^[30] showed that the frequency of mild periodontitis in PCOS and healthy women was not statistically significant which is consistent with our study. However, the frequency of moderate periodontitis was higher in PCOS group compare to control subjects. In our study, neither case nor control groups were periodontally healthy which opposed to the Porwal *et al.* study.

Consistent with previous studies, the higher BOP rates in patients with PCOS were compatible with the known impact of hyperandrogenism on vascular flora.^[22] Also, the increased CAL in the patients with PCOS group might be attributed to an increased susceptibility to activation of inflammatory processes. These inflammatory processes play a great role in the development of periodontal disease and involvement of gingival supporting tissues, and subsequent gingival sulcus depth and bone loss.

The PI index was higher in PCOS group which may be considered as the main limitation of our study. The similar periodontitis severity in study groups may be due to the leading role of PI in the pathogenesis of periodontal disease. Future studies should focus on investigations in individuals matched for PI.

In this study, by matching individuals and excluding some factors such as age, obesity (BMI >25), smoking, and antibiotic therapy within the 6 months period prior to the study, we aimed to minimize the effects of confounding factors. In order to eliminate other factors such as socioeconomic status and genetics, subjects were selected from the same family or residential area. It is worth noting that many PCOS patients with abnormal glucose tolerance and metabolic syndrome are obese. In these patients, adipose tissue converts testosterone to estrogen by aromatase enzyme, triggering a vicious cycle of intense hormonal effects on periodontal tissues. These effects may cause greater likelihood for development of more severe forms of periodontitis in these patients.

Therefore, further investigations in obese patients and patients with different degrees of PCOS severity are recommended.

Our study showed higher prevalence of periodontal disease parameters in nonobese women with PCOS comparing to systematically healthy controls. The findings need further confirmation in future studies systematically studying women with PCOS to elucidate any underlying relationships.

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Conflicts of interest

The authors of this manuscript declare that they have no conflicts of interest, real or perceived, financial or non-financial in this article.

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