Short Communication

# Comparative Assessment of Periodontal Status in Subjects with and without Polycystic Ovary Syndrome and its Correlation with Body Mass Index: A Cross-Sectional Study

**Sarah Mariam[1], Amita Mali[1], Saif Khan[2], Neha Agrawal[2], Juhi Gupta[2], Najam Khalique[3]**

[1]Department of Periodontology, Bharati Vidyapeeth Deemed to be University, Dental College and Hospital, Pune, Maharashtra, [2]Department of Periodontics and Community Dentistry, Dr. Ziauddin Ahmad Dental College and Hospital, A.M.U, Aligarh, Uttar Pradesh, [3]Department of Community Medicine, J N Medical College and Hospital, A.M.U, Aligarh, Uttar Pradesh, India

## Abstract

New avenues for research have opened, which assess the influence of systemic disease on periodontium and vice versa. To find the correlation between polycystic ovary syndrome (PCOS) and periodontium by assessing clinical parameters [plaque index (PI), probing depth, periodontal disease index (PDI)] and the anthropological parameter [body mass index (BMI)] and to find the correlation between body mass index and periodontal disease index in subjects with and without PCOS. Sixty females comprising 30 with PCOS and 30 without PCOS were selected. Clinical, anthropological, and radiological assessment was done. Double blinding was incorporated. There was a statistically highly significant difference in mean age, mean PI, and mean PDI ($P < 0.001$) in PCOS group when compared to those without PCOS group by unpaired *t*-test for inter-group analysis. A statistically significant difference was found in mean probing depth and mean BMI ($P < 0.05$) in PCOS group when compared to those without PCOS group by unpaired *t*-test for inter-group analysis. No statistically significant correlation was found between mean PDI and mean BMI in PCOS and non-PCOS group subjects using Spearman's rank correlation. Women suffering from PCOS may be at a heightened risk for developing periodontal disease as our study re-establishes this association with respect to some periodontal parameters. With such a result, general practitioners/gynecologists can be encouraged to refer cases of PCOS to periodontists for early detection, prevention of periodontal disease, and maintenance of periodontal health.

**Keywords:** Body mass index, periodontal disease index, polycystic ovary syndrome

## Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine aberration with metabolic and clinical manifestations among women of a reproductive age group.[1] Rotterdam criteria were proposed for its diagnosis, with the presence of at least two of the following: 1) amenorrhea or oligomenorrhea, 2) increased androgens (testosterone levels) biochemically and clinically, and 3) polycystic ovaries indicated by ultrasonography.[2] PCOS is also called as "XX syndrome" as it has features similar to those of the metabolic syndrome with increased risk for developing insulin resistance.[3]

Periodontitis is chronic inflammatory disease of periodontium, resulting in loss of attachment and subsequent loosening of teeth. Chronic stimulation and production of pro-inflammatory bone resorption factors, which are hallmarks of periodontal disease, also contribute to insulin resistance.[4]

Previous research has signaled a possible inter-relationship between periodontitis and PCOS, hypothetically pointing to an impact of PCOS on periodontal status including gingival inflammation.[5] Females suffering from PCOS have increased concentration of systemic inflammatory markers as well as blood lymphocytes and monocytes, which may

**Address for correspondence:** Prof. Najam Khalique, Department of Community Medicine, J N Medical College and Hospital, A.M.U, Aligarh - 202 002, Uttar Pradesh, India.
E-mail: najam_km@yahoo.com



**Access this article online**

Quick Response Code:

Website: www.ijcm.org.in

DOI: 10.4103/ijcm.ijcm_1012_22

**How to cite this article:** Mariam S, Mali A, Khan S, Agrawal N, Gupta J, Khalique N. Comparative assessment of periodontal status in subjects with and without polycystic ovary syndrome and its correlation with body mass index: A cross-sectional study. Indian J Community Med 2023;48:798-801.

**Received:** 28-12-22, **Accepted:** 17-07-23, **Published:** 07-09-23

increase susceptibility to periodontitis.[6] The presence of insulin resistance in both periodontitis and PCOS could be a converging factor.

Current evidence has linked obesity to increased risk for periodontitis.[7] Adipose tissues secrete adipokines, which play a role in inflammatory pathways similar to those of periodontitis.[8] Most of PCOS women are obese and are in the range of higher body mass indexes (BMIs).[9] These women are at increased risk for periodontitis from both obesity and insulin resistance. Therefore, this study also included the search for any correlation between obesity and periodontitis by comparing BMI and gingival and periodontal components of periodontal disease index (PDI). To the best of our knowledge, this is the first study done to compare PDI and BMI in PCOS subjects.

The five-fold objectives are as follows: 1) to evaluate and compare plaque indexes (PIs) in subjects with and without PCOS, 2) to evaluate and compare probing depths in subjects with and without PCOS, 3) to evaluate and compare PDI in subjects with and without PCOS, 4) to evaluate and compare BMI in subjects with and without PCOS, and 5) to correlate PDI and BMI in subjects with and without PCOS.

## Materials and Methods

This cross-sectional study was conducted in the Department of Periodontology, Bharati Vidyapeeth Dental College and Hospital, Pune, and the Department of Obstetrics and Gynecology, Bharati Hospital, Pune. The duration of the study was March 2018 to April 2019. The research proposal was approved by the Institutional Ethics Committee (BrIII/22-12-2017). Informed written consent was taken from all participants. A total of 60 female subjects comprised the study population. Group A consisted of 30 subjects diagnosed with PCOS. These were selected from the out-patient Department of Obstetrics and Gynecology. Group B consisted of 30 subjects without PCOS and were selected randomly and age-matched.

### Inclusion criteria

**Group A** study subjects were those females of age 18–40 years diagnosed with PCOS, with the minimal presence of 16 natural teeth. Cushing syndrome, congenital adrenal hyperplasia, androgen secreting tumors, and other medical conditions which mimic clinical manifestations of PCOS were excluded.[2]

**Group B** study subjects were regularly menstruating women (18–40 years of age) without any biochemical or clinical sign of androgen excess. Ultrasonography was done for all subjects to aid in the diagnosis of this syndrome.

**Exclusion criteria:** Pregnant women, smokers, alcoholics, women taking prophylactic antibiotics for dental procedures during the past 3 months, or those who received periodontal treatment during the past 6 months or having any other systemic disease.

**Clinical examination:** A proforma was designed for recording findings of clinical parameters. The relevant data comprising chief complaints and preliminary medical and dental history were recorded. Assessment of clinical parameters was carried out in BVDU. The autoclaved diagnostic instruments were utilized, including a graduated periodontal probe (UNC 15). Blinding was incorporated in assessment, with the help of another post-graduate student for both group A and group B. The parameters recorded were 1) PI (Turesky, Gilmore, Glickman modification of Quigley–Hein 1970) using 2% erythrosine dye for all teeth, except third molars; 2) probing depth (PD) with UNC-15 probe involving six-point probing per tooth (midbuccal, mesiobuccal, distobuccal, mid-lingual/palatal, mesiolingual/palatal, disto lingual/palatal) by "walking the probe" for all teeth except third molars; 3) gingival and periodontal components of PDI (Ramjford, 1959) for all teeth except third molars; and 4) BMI, also called as Quetelet index, calculated by the mathematical formula BMI = weight (kg)/(height in meters)$^2$

**Sample Size:** We have calculated the sample size by using Cochran formula

$$n = \frac{2 * (Za + Z(1-\beta))2 * (\text{Combined SD})^{\wedge 2}}{d2}$$

n = sample size

Za = Standard normal variate for a = 0.05 (95% CI)

Z1-β = Standard normal variate for 1-β =0.80 (80%)

Combined SD = 0.38

d = effect size = 0.30

The minimum required sample size is 25 per group.

To avoid loss of follow-up, additional 5% of total sample size was taken into consideration.

### Statistical analysis

Data analysis was performed by using SPSS (Statistical Package for Social Sciences) version 20:0. Quantitative variables were expressed by using mean, standard deviation (SD), median, and so on. Qualitative data variables were expressed by using frequency and percentage (%). Chi-square test/Fisher's exact test was used to compare the qualitative data variables with treatment groups. *P*- value <0.05 considered as significant.

## Results

Taking into consideration there was no loss to follow-up, the mean age for group A was 30.67, while for non-PCOS group, it was 27.17, with SDs of 2.22 and 3.39, respectively. A statistically highly significant difference was found (*P*- value <0.001) [Table 1].

The mean PI for group A was 0.62 with an SD of 0.54, while for group B, it was 0.04 with an SD of 0.05. There was a statistically highly significant difference in PI in the two groups, with higher values in group A [Table 1].

**Table 1: Intergroup comparison of mean PI, PD, PDI, and BMI**

| Parameters | Group A (Mean±SD) | Group B (Mean±SD) | *P* |
|---|---|---|---|
| Mean PI | 0.6202±0.5383 | 0.0428±0.0497 | <0.001** |
| Mean PD | 1.2812±0.2268 | 1.1831±0.1431 | 0.05* |
| Mean PDI | 0.6023±0.4007 | 0.0654±0.1023 | <0.001** |
| Mean BMI | 25.6283±3.6371 | 23.3292±3.9062 | 0.022* |

*P*<0.05 - Significant*, *P*<0.001 - Highly significant**)

The mean PD for group A was 1.28 with an SD of 0.23, while for group B, it was 1.18 with an SD of 0.14. The result was statistically significant [Table 1].

The mean value for PDI for group A was 0.60 with an SD of 0.40. The mean value for PDI for group B was 0.07 with an SD of 0.10. There was a statistically highly significant difference in PDI values in both groups, with higher values noted in group A [Table 1].

The mean value for BMI in group A was 25.63 with an SD of 3.64, while for group B, it was 23.33 with an SD of 3.91. There was a statistically significant difference in BMI in the two groups, with higher values in group A.

For finding the correlation between PDI and BMI using Spearman's rank correlation coefficient for both groups A and B, the z value was found to be 1.30 and 0.56, respectively, with a *P*- value greater than 0.05, which was statistically not significant.

## Discussion

Women with underlying PCOS present with a plethora of features related to ovarian dysfunction and hyperandrogenism. Their quality of life is impacted as they are at risk of multiple dilapidating conditions like diabetes mellitus (type 2), obesity, cardiovascular diseases, and endometrial cancer. Hyperinsulinemia and insulin resistance play pivotal roles in pathogenesis of this syndrome.[10] Higher testosterone levels directly influence insulin in target tissues (e.g., muscles) having roles in contributing to the state of insulin resistance.[11] Obesity (visceral) is linked to hyperlipidemia, glucose intolerance, insulin-resistant state, and hyperandrogenism.[12]

There was a statistically highly significant difference in PI in the two groups, with higher values in group A [Table 1]. The results are in accordance with Dursun E *et al*.[5] and Rahiminejad ME *et al.*[12] Akcalı A *et al*. 2014[13] concluded that counts of periodontal bacteria in salivary secretions and their serum antibody response are increased in PCOS as it might influence oral microbiota by having a colluding response with gingival and periodontal health. However, Porwal S *et al*.[14] did not find any significant differences. It is well known that plaque is the primary etiologic factor in causation of chronic inflammatory periodontal disease.

In our study, marginally higher values of PDs were found in group A, which was statistically significant [Table 1]. The result is synchronous with that found by Dursun *et al*.,[5] Porwal *et al*.,[14] and Akcalı A *et al*. 2017[15] Assessment of PD is an indispensable clinical parameter for determining the level of epithelial attachment. Increased probing values (above the baseline) point to progressive periodontal disease, although the influence of host response and other risk factors remains a subject of investigation in such patients.

The distinct advantage of PDI is its dual nature to find the gingival status as well as periodontal status of individual teeth. In the gingival status, it takes into account the inflammatory status around teeth. In the periodontal status, it takes into account the level of periodontal attachment loss at various levels starting from CEJ up to more than 6 mm apical to CEJ. In our study, there was a statistically highly significant difference in PDI values in the two groups, with higher values noted in subjects with PCOS [Table 1]. In a study by Dursun *et al*.,[5] the GI, BOP, and volume of GCF (sub-clinical sign of gingival inflammation) were increased in PCOS compared to the control group. Therefore, the observations were similar. In the study of Porwal *et al*.,[14] PCOS-N group had a significantly higher CAL and BOP and a higher frequency of moderate periodontitis. In our study, assessment of PDI was done (not directly CAL and bleeding on probing) and higher values of PDI were found in PCOS group compared to non-PCOS group. Akcalı *et al.*[15] concluded that myeloperoxidase and matrix metallo-proteinase-9 levels in serum had higher values in women with gingivitis and PCOS in comparison to women (with periodontal health) and PCOS. It pointed toward an association between gingival inflammation and PCOS.

Many recent studies have pointed toward a link between obesity and periodontitis.[6] Adipokines secreted by fat tissues are the chemical mediators that may contribute to periodontitis.[7] In our study, values for mean BMI were statistically significant ($P < 0.05$) with higher values in PCOS group [Table 1]. These women may be at an increased risk for periodontitis owing to greater concentration of adipose tissues with respect to body mass. The results of our study matched to a study conducted by Dursun *et al*.[5] and by Porwal *et al*.,[14] in which mean BMI (kg/m$^2$) in PCOS-N was 25.01 with an SD of 3.61; in PCOS-MT, it was 24.57 with an SD of 4.38, and in the control group, it was 23.76 with a mean deviation of 4.82. Concurrently, raised periodontitis scores were observed in these patients.

Various studies have linked PCOS patients with increased concentration of inflammatory markers like IL-6, TNF-alfa, and IL-17 and some matrix metalloproteinases in saliva, GCF, and serum, which may point to a positive correlation between periodontal disease and PCOS.[5,15] Our study also re-establishes this association between PCOS and periodontal disease.

No statistically significant correlation was found between PDI and BMI in both group A and B subjects using Spearman's rank

correlation. Our study de-links the hypothesized association between these two clinical entities.

## Conclusion

Women with PCOS had a statistically highly significant difference in mean age, mean PI scores, and mean periodontal index scores compared to ones without PCOS. A statistically significant difference in mean PD and mean BMI was found in subjects with PCOS. Thus, women suffering from this syndrome may be at a cumulative risk for developing periodontal disease. Our study disassociates from relationship between the anthropological parameter of obesity (BMI) and the periodontal parameter (PDI) keeping in consideration that no statistically significant correlation of mean PDI and mean BMI was found in both groups.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

## References

1. Stein IF, Leventhal ML. Amenorrhea associated with bilateral polycystic ovaries. Am J Obstet Gynecol 1935;29:181-91.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 2004;81:19-25.
3. Sam S, Dunaif A. Polycystic ovary syndrome: Syndrome XX? Trends Endocrinol Metab 2003;14:365-70.
4. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, van der Velden U. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol 2000;71:1528-34.
5. Dursun E, Akalın FA, Guncu GN, Cinar N, Aksoy DY, Tozum TF, *et al*. Periodontal disease in polycystic ovary syndrome. Fertil Steril 2011;95:320-3.
6. Saito T, Shimazaki Y, Kiyohara Y, Kato I, Kubo M, Iida M, *et al.* Relationship between obesity, glucose tolerance, and periodontal disease in Japanese women: The Hisayama study. J Periodontal Res 2005;40:346-53.
7. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab 2004;89:2548-56.
8. Ehrmann DA, Barnes RB, Rosenfield RL, Cavaghan MK, Imperial J. Prevalence of impaired glucose tolerance and diabetes in women with polycystic ovary syndrome. Diabetes Care 1999;22:141-6.
9. Baillargeon JP, Iuorno MJ, Nestler JE. Insulin sensitizers for polycystic ovary syndrome. Clin Obstet Gynecol 2003;46:325-40.
10. Kelly CC, Lyall H, Petrie JR, Gould GW, Connell JM, Sattar N. Low grade chronic inflammation in women with polycystic ovarian syndrome. J Clin Endocrinol Metab 2001;86:2453-5.
11. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third report of the national cholesterol education program (NCEP) Expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III): Final report. Circulation 2002;106:3143-421.
12. Rahiminejad ME, Moaddab A, Zaryoun H, Rabiee S, Moaddab A, Khodadoustan A. Comparison of prevalence of periodontal disease in women with polycystic ovary syndrome and healthy controls. Dent Res J (Isfahan) 2015;12:507-12.
13. Akcalı A, Bostanci N, Özçaka Ö, Öztürk-Ceyhan B, Gümüş P, Buduneli N, *et al.* Association between polycystic ovary syndrome, oral microbiota and systemic antibody responses. PLoS One 2014;18:e108074.
14. Porwal S, Tewari S, Sharma RK, Singhal SR, Narula SC. Periodontal status and high-sensitivity C-reactive protein levels in polycystic ovary syndrome with and without medical treatment. J Periodontol 2014;85:1380-9.
15. Akcalı A, Bostanci N, Özçaka Ö, Gümüş P, Öztürk-Ceyhan B, Tervahartiala T, *et al.* Gingival inflammation and salivary or serum granulocyte-secreted enzymes in patients with polycystic ovary syndrome. J Periodontol 2017;88:1145-52.