"CLINICAL AND LABORATORY CHARACTERISTICS OF CHRONIC INITIAL PERIODONTITIS IN WOMEN OF REPRODUCTIVE AGE WITH POLYCYSTIC OVARY SYNDROME AND OPTIMIZATION OF TREATMENT"

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Abstract. *Background: Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disorder among women of reproductive age. In addition to reproductive dysfunction, PCOS is frequently associated with insulin resistance, hyperandrogenism, obesity, oxidative stress, and persistent low-grade systemic inflammation. Periodontitis is a chronic inflammatory disease of the tooth-supporting tissues in which microbial dysbiosis interacts with an altered host immune-inflammatory response.*

Increasing evidence suggests that PCOS and periodontitis may share several biological pathways, including systemic inflammation, impaired metabolic regulation, oxidative stress, and changes in immune responsiveness.

Objective: *To summarize the current understanding of the clinical and laboratory characteristics of periodontitis in women of reproductive age with PCOS and to propose an evidence-based approach for optimizing periodontal treatment in this population.*

Materials and methods: *This article presents a narrative synthesis of scientific literature addressing the association between PCOS and periodontal disease, clinical periodontal parameters, inflammatory and metabolic biomarkers, and contemporary approaches to periodontal treatment. Particular attention was given to systematic reviews, meta-analyses, clinical studies, and international periodontal treatment recommendations.*

Results*: Available evidence indicates that women with PCOS may have a higher prevalence and greater severity of periodontal inflammation than systemically healthy women.*

Increased bleeding on probing, periodontal probing depth, and clinical attachment loss have been reported in women with PCOS. Potential mechanisms include insulin resistance, hyperandrogenism, altered inflammatory signaling, oxidative stress, and increased systemic levels of pro-inflammatory mediators. C-reactive protein, interleukin-6, tumor necrosis factor-α, and other inflammatory parameters may reflect the systemic inflammatory background, although their routine use as diagnostic biomarkers of periodontitis in PCOS has not yet been sufficiently standardized. Treatment should therefore combine individualized periodontal therapy with management of modifiable systemic and behavioral risk factors.

Conclusion: *PCOS may represent an important systemic modifying condition in women with periodontitis. Early periodontal screening, comprehensive clinical assessment, individualized non-surgical periodontal therapy, maintenance care, and interdisciplinary cooperation between dentists, periodontists, gynecologists, endocrinologists, and other specialists may improve the long-term management of these patients. Further prospective studies are required to establish causal relationships and determine whether periodontal treatment produces clinically meaningful improvements in metabolic and reproductive outcomes.*

Keywords: *polycystic ovary syndrome; PCOS; periodontitis; reproductive-age women; periodontal inflammation; insulin resistance; C-reactive protein; interleukin-6; TNF-α; periodontal therapy; systemic inflammation.*

1. Introduction

Polycystic ovary syndrome is one of the most common endocrine disorders affecting women during the reproductive period. Although the syndrome is traditionally recognized through reproductive manifestations such as menstrual irregularity, ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology, its clinical significance extends beyond the reproductive system. Many women with PCOS also demonstrate insulin resistance, metabolic disturbances, increased adiposity, oxidative stress, and chronic low-grade systemic inflammation.

Periodontitis is another chronic inflammatory condition characterized by disruption of periodontal tissue homeostasis, progressive destruction of periodontal attachment, and, in susceptible individuals, alveolar bone loss. The disease develops through a complex interaction between dental biofilm dysbiosis and the host immune-inflammatory response. Contemporary periodontal classification emphasizes that disease severity and complexity should be assessed through a staging system, while disease progression and risk factors are incorporated into grading.

The possible association between PCOS and periodontal disease has attracted increasing scientific attention. Both disorders are characterized by inflammatory and metabolic abnormalities, which raises the possibility of shared pathogenic mechanisms. A systematic review and meta-analysis reported that women with PCOS had a higher risk of periodontitis, while women with periodontitis also demonstrated a higher likelihood of having PCOS.

However, the authors emphasized that the available evidence was predominantly observational and that causal relationships could not be established.

A more recent systematic review published in 2025 similarly concluded that most available studies indicated a higher prevalence and severity of periodontal disease among women with PCOS. At the same time, substantial heterogeneity in diagnostic criteria, study design, confounding variables, and periodontal assessment limited the comparability of the available evidence.

Therefore, the investigation of periodontal health in reproductive-age women with PCOS is relevant not only to dentistry but also to interdisciplinary women's healthcare.

2. Potential Biological Relationship Between PCOS and Periodontitis

The association between PCOS and periodontitis is unlikely to be explained by a single biological mechanism. Instead, several interconnected pathways may contribute to an increased inflammatory burden.

2.1. Chronic low-grade inflammation

Low-grade systemic inflammation is frequently observed in PCOS. Increased inflammatory activity may be associated with adiposity, insulin resistance, and endocrine dysfunction. Periodontitis can also contribute to systemic inflammatory exposure through the continuous interaction between periodontal tissues, microbial products, and immune cells.

C-reactive protein (CRP) is one of the most commonly studied systemic inflammatory markers. Increased CRP concentrations have been reported in both PCOS and periodontal disease, suggesting that the two conditions may contribute to a common inflammatory environment. However, CRP should not be regarded as a specific biomarker for either condition because it is influenced by numerous systemic and lifestyle factors.

2.2. Insulin resistance

Insulin resistance represents an important metabolic feature of PCOS. Hyperinsulinemia may affect inflammatory signaling, endothelial function, oxidative balance, and tissue metabolism.

Periodontal inflammation may also influence systemic metabolic homeostasis.

Consequently, a potential interaction between periodontal inflammation and insulin resistance has been proposed. Nevertheless, current evidence does not justify concluding that periodontitis directly causes insulin resistance in women with PCOS.

2.3. Hormonal factors

Sex hormones influence periodontal tissues through their effects on vascular permeability, connective-tissue metabolism, immune responses, and inflammatory signaling.

Hyperandrogenism and other endocrine abnormalities characteristic of PCOS may therefore modify periodontal tissue responses to bacterial biofilm.

Importantly, hormonal changes should be considered modifying factors rather than independent explanations for periodontal destruction.

2.4. Oxidative stress

Oxidative stress is involved in both metabolic disorders and chronic inflammatory diseases. Excessive production of reactive oxygen species can contribute to tissue damage and amplify inflammatory signaling.

In PCOS, oxidative imbalance may be associated with insulin resistance and endocrine dysfunction. In periodontitis, oxidative stress may contribute to connective tissue and alveolar bone destruction. This shared biological pathway provides a plausible explanation for the potentially greater inflammatory response observed in some women with PCOS.

3. Clinical Characteristics of Periodontitis in Women with PCOS

The periodontal examination of a woman with PCOS should not differ fundamentally from the examination of other patients; however, particular attention should be given to the inflammatory burden and systemic modifying factors.

Important clinical parameters include:

periodontal probing depth (PPD);

bleeding on probing (BOP);

clinical attachment level/loss (CAL);

plaque accumulation;

gingival inflammation;

gingival recession;

tooth mobility;

furcation involvement;

radiographic alveolar bone loss;

number and distribution of periodontal pockets.

The 2017 World Workshop classification recommends diagnosing periodontitis using a multidimensional approach based on severity, complexity, and progression risk rather than relying solely on the historical distinction between “chronic” and “aggressive” periodontitis.

Evidence synthesized in earlier meta-analyses demonstrated that women with PCOS may exhibit greater gingival inflammation, deeper periodontal pockets, and greater clinical attachment loss than women without PCOS. One systematic review found a statistically significant association between PCOS and several periodontal parameters, including clinical attachment loss, probing depth, gingival inflammation, and bleeding on probing.

A larger systematic review and meta-analysis also reported that PCOS was associated with approximately a 28% higher risk of periodontitis, while periodontitis was associated with a higher likelihood of PCOS. However, these findings should be interpreted as evidence of association rather than proof of a bidirectional causal relationship.

4. Laboratory Characteristics and Potential Biomarkers

Clinical periodontal parameters remain the foundation of diagnosis. Laboratory markers can provide additional information regarding systemic inflammatory and metabolic status but should not replace periodontal examination.

Potentially relevant laboratory parameters include:

4.1. C-reactive protein

High-sensitivity CRP may reflect systemic inflammatory activity. Women with PCOS and periodontal disease may have an increased inflammatory burden, although CRP lacks disease specificity.

4.2. Interleukin-6

IL-6 is a multifunctional cytokine involved in the regulation of acute and chronic inflammation. It has been investigated in both PCOS and periodontal disease. Alterations in IL-6 may reflect the interaction between local periodontal inflammation and systemic inflammatory processes.

4.3. Tumor necrosis factor-alpha

TNF-α is a major pro-inflammatory cytokine involved in immune regulation and tissue destruction. Studies evaluating women with PCOS have investigated TNF-α in serum, saliva, and gingival crevicular fluid. These findings support the concept that inflammatory mediators may participate in the interaction between endocrine-metabolic and periodontal disorders.

4.4. Metabolic parameters

Because insulin resistance is a clinically important component of PCOS, periodontal assessment may appropriately be accompanied by consideration of metabolic parameters such as fasting glucose, glycated hemoglobin when clinically indicated, fasting insulin, and calculated indices of insulin resistance.

These parameters should be interpreted by the appropriate medical specialist and should not be used as independent diagnostic criteria for periodontitis.

5. Microbiological Considerations

Periodontitis is fundamentally associated with an ecological imbalance within the subgingival biofilm and an inappropriate or excessive host inflammatory response. PCOS may modify the host environment through metabolic, endocrine, and inflammatory pathways.

However, currently available evidence does not support a specific “PCOS periodontal microbiome” that could be used routinely for diagnosis. Therefore, conventional periodontal diagnosis should remain based on clinical examination, periodontal charting, radiographic findings, and assessment of risk factors.

Molecular microbiological testing may have a role in selected clinical or research situations, but its routine application requires further validation.

6. Optimization of Periodontal Treatment

The treatment of periodontitis in women with PCOS should follow evidence-based periodontal principles while taking the patient's systemic condition into account.

The European Federation of Periodontology recommends a stepwise approach to periodontal treatment.

The major therapeutic components include behavioral and oral-hygiene intervention, professional removal of supra- and subgingival deposits, management of periodontal pockets, and long-term supportive periodontal care.

6.1. Initial periodontal assessment

Before treatment, the clinician should collect:

detailed medical and gynecological history;

current medications;

metabolic risk factors;

smoking status;

oral hygiene practices;

periodontal charting;

radiographic assessment where indicated;

periodontal risk assessment.

The periodontal diagnosis should then be expressed using the current stage-and-grade framework.

6.2. Professional mechanical plaque control

Professional removal of supra- and subgingival biofilm is the cornerstone of periodontal therapy. Patient education should focus on individualized toothbrushing, interdental cleaning, and sustainable daily plaque-control practices.

Because PCOS is a chronic condition, periodontal care should also be regarded as a long-term management process rather than a single course of treatment.

6.3. Non-surgical periodontal therapy

Scaling and root surface instrumentation remain central components of non-surgical periodontal treatment. Treatment should be adapted to disease severity, pocket depth, anatomical complexity, patient tolerance, and response to initial therapy.

A clinical trial involving women with PCOS and chronic periodontitis investigated non-surgical periodontal therapy together with myo-inositol and evaluated inflammatory and metabolic outcomes, including hsCRP and insulin resistance. Such studies are clinically interesting because they raise the possibility that periodontal intervention may influence systemic inflammatory status; however, they do not establish that periodontal therapy is a treatment for PCOS itself.

6.4. Adjunctive treatment

Antiseptic agents, locally delivered antimicrobials, systemic antibiotics, or periodontal surgery should not be prescribed automatically simply because the patient has PCOS.

Adjunctive therapy should be selected according to the periodontal diagnosis, treatment response, risk profile, and evidence-based indications.

This distinction is particularly important for antimicrobial stewardship.

6.5. Supportive periodontal therapy

Long-term maintenance is especially important because periodontal inflammation can recur after successful active treatment.

Follow-up intervals should be individualized according to:

periodontal stage and grade;

previous treatment response;

oral hygiene;

smoking status;

metabolic risk factors;

residual periodontal pockets;

patient adherence.

The EFP treatment framework emphasizes that periodontal therapy should proceed sequentially and that treatment decisions should be based on clinical response rather than on a fixed treatment protocol for every patient.

7. Interdisciplinary Management

Women with PCOS and clinically significant periodontitis may benefit from coordinated care involving several specialists.

A practical interdisciplinary model may include:

Gynecologist/endocrinologist → assessment and management of PCOS, hormonal status, metabolic risk factors and insulin resistance

Periodontist/dentist → periodontal diagnosis, mechanical periodontal treatment and maintenance

Primary-care physician/nutrition specialist → management of lifestyle and metabolic risk factors when appropriate

Such cooperation is particularly valuable when the patient presents with obesity, insulin resistance, diabetes or other metabolic abnormalities.

The objective should not be to attribute one disease to the other, but rather to identify potentially modifiable factors that contribute to the overall inflammatory and metabolic burden.

8. Proposed Clinical-Laboratory Algorithm

For reproductive-age women diagnosed with PCOS, a practical periodontal screening algorithm can include the following sequence:

Step 1. Identify the diagnosis and clinical phenotype of PCOS.

Step 2. Record systemic and behavioral risk factors, including obesity, insulin resistance, smoking and oral hygiene.

Step 3. Perform a periodontal screening examination.

Step 4. If periodontal disease is suspected, perform comprehensive periodontal charting and appropriate radiographic assessment.

Step 5. Classify periodontitis according to the current stage-and-grade system.

Step 6. Assess systemic inflammatory and metabolic parameters when clinically indicated.

Step 7. Initiate individualized non-surgical periodontal therapy.

Step 8. Reassess periodontal outcomes after active treatment.

Step 9. Escalate treatment when residual disease persists despite adequate initial therapy.

Step 10. Establish individualized supportive periodontal care.

This approach allows the periodontal condition to be treated according to established periodontal principles while acknowledging the systemic characteristics of PCOS.

9. Discussion

The relationship between PCOS and periodontitis is biologically plausible but clinically complex. Both conditions may involve chronic inflammatory activity, oxidative stress, altered metabolic regulation, and changes in host immune responses. This shared pathophysiological background provides a reasonable explanation for the higher periodontal inflammatory burden reported in several observational studies.

At the same time, important confounding variables must be considered. Obesity, insulin resistance, smoking, socioeconomic status, oral hygiene, dietary patterns, medication use, and access to dental care may influence periodontal outcomes independently of PCOS.

Consequently, the presence of PCOS should not automatically be interpreted as the direct cause of periodontitis. Similarly, the presence of periodontitis should not be considered sufficient evidence that a woman has PCOS.

The available literature increasingly supports an association between the two conditions.

The 2025 systematic review concluded that most included studies reported greater periodontal prevalence or severity among women with PCOS but also emphasized substantial heterogeneity and methodological limitations.

This means that the most clinically appropriate approach is not to search for a single disease-specific biomarker, but to integrate periodontal clinical findings with the patient's endocrine, metabolic, inflammatory, and behavioral profile.

10. Limitations of Current Evidence

Several limitations should be acknowledged.

First, a considerable proportion of published evidence is observational. Therefore, causal relationships cannot be reliably established.

Second, different studies have used different diagnostic criteria for PCOS and periodontal disease.

Third, obesity, insulin resistance, smoking, age, socioeconomic conditions, and oral hygiene may act as confounding factors.

Fourth, laboratory biomarkers such as CRP, IL-6, and TNF-α are not specific for PCOS-associated periodontitis.

Finally, there is still limited evidence from large, well-designed prospective interventional studies assessing whether periodontal treatment can produce clinically significant improvements in PCOS-related metabolic or reproductive outcomes.

11. Conclusion

Polycystic ovary syndrome and periodontitis represent two chronic conditions that may interact through common inflammatory, metabolic, endocrine, and oxidative pathways. Current evidence suggests that women of reproductive age with PCOS may demonstrate increased periodontal inflammation and, in some populations, greater periodontal tissue destruction.

The clinical assessment of these patients should include comprehensive periodontal examination with measurement of probing depth, bleeding on probing, clinical attachment level, plaque accumulation, tooth mobility, and radiographic bone loss when indicated. Laboratory evaluation may provide supplementary information regarding systemic inflammation and metabolic status, but no currently available laboratory marker can replace clinical periodontal diagnosis.

Optimization of treatment should be based on contemporary periodontal principles: individualized risk assessment, effective oral-hygiene instruction, professional mechanical plaque control, non-surgical periodontal therapy, appropriate management of persistent periodontal lesions, and regular supportive periodontal care.

Interdisciplinary collaboration between dental and medical specialists is particularly important for patients with significant metabolic or endocrine abnormalities.

Future longitudinal and randomized clinical studies are needed to determine whether effective periodontal treatment can influence systemic inflammatory, metabolic, or reproductive outcomes in women with PCOS.

Overall, periodontal screening and preventive care should be considered an important component of comprehensive healthcare for women with PCOS, while the relationship between the two diseases should be interpreted as an association supported by emerging evidence rather than as a definitively established causal pathway.

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