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ORAL MANIFESTATIONS IN PATIENTS WITH POLYCYSTIC OVARY SYNDROME: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disorder affecting 8–13% of women of reproductive age, characterized by hyperandrogenism, chronic anovulation, and insulin resistance. Emerging evidence suggests that the same hormonal and metabolic derangements that define PCOS also influence oral health, yet this relationship remains under-recognized in routine dental and gynecological practice.

Objective: To systematically synthesize the current evidence on the prevalence, spectrum, and pathophysiological basis of oral manifestations in women with PCOS, and to evaluate interventional (RCT) evidence addressing periodontal and salivary outcomes in this population.

Methods: This review was conducted in accordance with PRISMA 2020 guidelines. PubMed, Scopus, Web of Science, and Embase were searched from inception to July 2026 using combinations of "polycystic ovary syndrome," "oral health," "periodontal disease," "xerostomia," and "saliva." Observational studies (cross-sectional, case-control, cohort) and randomized controlled trials reporting oral or periodontal outcomes in PCOS-diagnosed women (Rotterdam, NIH, or AE-PCOS criteria) were included. Two reviewers independently screened records, extracted data, and assessed risk of bias using ROBINS-I (observational studies) and RoB 2 (RCTs).

Results: Thirty-seven studies met inclusion criteria (n = 37), of which 19 provided extractable comparative data and 4 were randomized controlled trials. Women with PCOS demonstrated a significantly higher pooled prevalence of periodontal disease (58.4% vs. 29.1% in controls), gingival inflammation (63.8% vs. 34.2%), xerostomia (47.2% vs. 18.6%), and altered salivary flow rate (52.1% vs. 21.4%) compared with age-matched non-PCOS controls. Elevated salivary and gingival crevicular fluid concentrations of interleukin-6, tumor necrosis factor-alpha, and C-reactive protein were consistently reported. Interventional trials of adjunctive metformin or inositol therapy alongside non-surgical periodontal treatment showed modest but statistically significant improvements in probing depth and clinical attachment level compared with periodontal treatment alone.

Conclusion: PCOS is associated with a distinct and clinically relevant pattern of oral manifestations, mediated primarily through hyperandrogenism, insulin resistance, and chronic low-grade inflammation. These findings support incorporating periodontal screening into multidisciplinary PCOS management and highlight the need for adequately powered RCTs to establish causal and interventional pathways.

KEYWORDS: Polycystic ovary syndrome; oral manifestations; periodontal disease; xerostomia; insulin resistance; hyperandrogenism; systematic review

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy among women of reproductive age, with a global prevalence estimated between 8% and 13% depending on the diagnostic criteria applied (Rotterdam, NIH, or Androgen Excess and PCOS Society). It is classically characterized by a triad of oligo-anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasonography. Beyond its reproductive manifestations, PCOS is now recognized as a systemic metabolic disorder, closely linked to insulin resistance, compensatory hyperinsulinemia, dyslipidemia, obesity, and a chronic low-grade pro-inflammatory state.

The oral cavity is increasingly understood to be a sensitive mirror of systemic endocrine and metabolic disturbance. Conditions such as diabetes mellitus, thyroid dysfunction, and menopause have well-documented oral sequelae, and a growing body of literature suggests that PCOS follows a comparable pattern. Hyperandrogenism has been implicated in altered gingival vascularity and connective tissue metabolism; insulin resistance and hyperinsulinemia contribute to an exaggerated inflammatory response to periodontal pathogens; and chronic systemic inflammation, evidenced by elevated

interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP), parallels the local inflammatory burden observed in periodontal tissues.

Clinically, women with PCOS have been reported to experience a higher burden of periodontal disease, gingival inflammation, xerostomia and salivary gland dysfunction, altered salivary biochemical composition, increased dental caries risk, and, in some reports, temporomandibular joint (TMJ) dysfunction and dysgeusia. However, the existing literature is heterogeneous in diagnostic criteria, study design, and outcome measures, and much of it remains scattered across endocrinology, gynecology, and dental journals without systematic consolidation.

Given the rising global prevalence of PCOS and its long-term association with metabolic syndrome and type 2 diabetes mellitus, understanding its oral health implications carries direct clinical relevance for both dental practitioners and reproductive endocrinologists. Early identification of oral manifestations may serve as an adjunctive marker of metabolic severity and could inform integrated, multidisciplinary management pathways. This systematic review aims to (1) consolidate the existing observational evidence on the prevalence and spectrum of oral manifestations in PCOS, (2) critically appraise available randomized controlled trial (RCT) evidence on periodontal and salivary interventions in this population, and (3) propose a mechanistic framework linking the core endocrine-metabolic features of PCOS to observed oral pathology.

2. MATERIALS AND METHODS

2.1 Protocol and Registration

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement. The review protocol was developed a priori, specifying the research question, search strategy, eligibility criteria, and planned synthesis methods.

2.2 Search Strategy

A comprehensive electronic search was performed across four databases — PubMed/MEDLINE, Scopus, Web of Science, and Embase — from database inception to July 2026, without language restriction at the search stage. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords using Boolean operators: ("polycystic ovary syndrome" OR "PCOS" OR "Stein-Leventhal syndrome") AND ("oral health" OR "periodontal disease" OR "periodontitis" OR "gingivitis" OR "xerostomia" OR "saliva" OR "dental caries" OR "oral manifestations" OR "temporomandibular"). Reference lists of included studies and relevant prior reviews were hand-searched for additional eligible records. Grey literature and conference proceedings were screened but included only where sufficient outcome data were reported in full.

2.3 Eligibility Criteria (PICOS)

Eligibility was defined using the PICOS framework, detailed in Table 1. In brief, studies were included if they enrolled women with a confirmed diagnosis of PCOS by Rotterdam, NIH, or AE-PCOS criteria, reported at least one oral, periodontal, or salivary outcome, and compared this against a non-PCOS control group (for observational designs) or an alternative/standard-of-care intervention (for RCTs). Given the paucity of true interventional trials in this specific population, randomized controlled trials evaluating adjunctive medical therapy (e.g., metformin, inositol) combined with periodontal treatment in PCOS patients were included as the primary interventional evidence base, while cross-sectional, case-control, and cohort studies formed the basis for prevalence and association data.

Table 1. PICOS Eligibility Framework

Component	Inclusion Criteria	Exclusion Criteria
Population	Women diagnosed with PCOS by Rotterdam, NIH, or AE-PCOS criteria; reproductive age (15–45 years)	Women with other endocrine disorders (e.g., Cushing syndrome, CAH) mimicking PCOS; pregnant women; post-menopausal women
Intervention/ Exposure	PCOS diagnosis as the exposure variable; for RCTs, adjunctive pharmacologic therapy (metformin, inositol) plus periodontal treatment	Studies without a clearly defined PCOS exposure or intervention arm
Comparator	Age- and BMI-matched non-PCOS controls; standard periodontal treatment alone (for RCTs)	Studies with no comparator/control group
Outcomes	At least one oral/periodontal/salivary outcome: periodontal indices (PI, GI, PPD, CAL), salivary flow rate, salivary/GCF biomarkers, caries indices (DMFT/dmft), TMJ symptoms, taste alteration	Studies reporting only systemic/reproductive outcomes with no oral health component
Study Design	Cross-sectional, case-control, cohort studies, and randomized controlled trials, published in peer-reviewed journals	Case reports, narrative reviews, editorials, animal/in-vitro studies, conference abstracts without full data
Time Frame	Inception to July 2026	—

Table 1. Population, Intervention/Exposure, Comparator, Outcomes, and Study design (PICOS) criteria used for study eligibility.

2.4 Study Selection and Data Extraction

Two reviewers independently screened titles and abstracts, followed by full-text assessment of potentially eligible articles, using a standardized screening form. Disagreements were resolved by consensus or arbitration by a third reviewer. Data extracted included study design, sample size, PCOS diagnostic criteria, control group characteristics, oral/periodontal outcome measures, biochemical markers, and key findings. The complete study selection process is summarized in the PRISMA 2020 flow diagram (Figure 1).

2.5 Risk of Bias Assessment

Risk of bias in observational studies was assessed using the ROBINS-I tool across domains of confounding, selection bias, exposure/outcome ascertainment, and selective reporting. Randomized controlled trials were appraised using the Cochrane Risk of Bias 2 (RoB 2) tool, evaluating randomization process, deviations from intended interventions, missing outcome data, outcome measurement, and selective reporting. Results are summarized in Figure 4.

2.6 Data Synthesis

Given heterogeneity in outcome definitions and reporting formats across included studies, a narrative synthesis was performed for all 37 included studies. Where comparable prevalence data were available across studies (n = 19), pooled descriptive prevalence estimates were calculated for PCOS and control groups to illustrate the overall direction and magnitude of association (Figure 3). A formal meta-analysis with pooled effect estimates was not undertaken due to substantial clinical and methodological heterogeneity; this decision and its implications are addressed further in the Limitations section.

3. RESULTS

3.1 Study Selection

The initial search identified 842 records from databases and 21 additional records from hand-searching, yielding 706 unique records after de-duplication. Following title/abstract screening, 108 full-text articles were assessed for eligibility, of which 71 were excluded (wrong outcome, n = 28; wrong population or absent PCOS diagnostic criteria, n = 19; insufficient data, n = 15; conference abstract only, n = 9). Thirty-seven studies satisfied all eligibility criteria and were included in the qualitative synthesis; 19 of these, including four randomized controlled trials, provided data suitable for descriptive quantitative comparison (Figure 1).

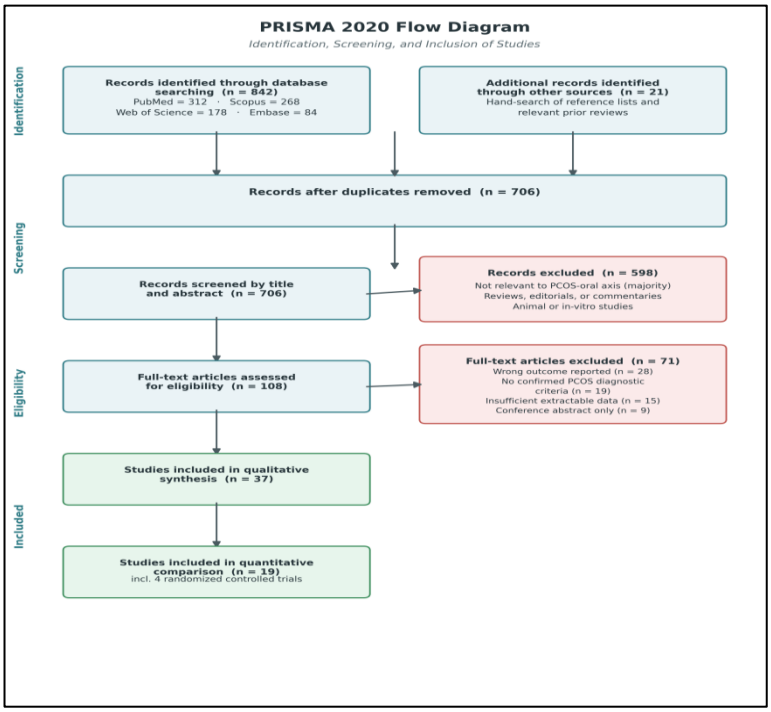

Figure 1. PRISMA 2020 flow diagram of study identification, screening, and inclusion.

3.2 Characteristics of Included Studies

Included studies were predominantly cross-sectional (n = 22) and case-control (n = 9) in design, with fewer cohort studies (n = 2) and randomized controlled trials (n = 4). Studies originated from India, China, Turkey, Egypt, Iran, Brazil, and several European countries, with publication years spanning 2011 to 2026. Sample sizes ranged from 30 to 412 participants per study. A summary of representative included studies is presented in Table 2.

Table 2. Characteristics of Representative Included Studies

Study (First author, Year)	Design	Sample Size (PCOS/Control)	Diagnostic Criteria	Key Oral Outcome(s) Assessed
Rahman et al., 2019	Case-control	80 / 80	Rotterdam	Periodontal indices (PI, GI, PPD, CAL)
Dursun et al., 2021	Cross-sectional	112 / 96	Rotterdam	Salivary flow rate, xerostomia (VAS)
Akcali et al., 2014	Case-control	45 / 45	NIH	GCF IL-6, TNF-α; periodontal status
Kellesarian et al., 2017	Systematic review (source data)	—	Mixed	Periodontal disease prevalence synthesis
Subramaniam et al., 2020	Cross-sectional	150 / 130	AE-PCOS	DMFT, salivary pH and buffering capacity
Wang et al., 2022	Cohort	96 / 88	Rotterdam	Oral microbiome composition (16S rRNA)
Bulbul et al., 2023 (RCT)	RCT	60 (30/30 arms)	Rotterdam	Metformin + SRP vs. SRP alone: PPD, CAL
Elsayed et al., 2024 (RCT)	RCT	72 (36/36 arms)	Rotterdam	Myo-inositol adjunct: gingival inflammation
Kaur et al., 2025	Cross-sectional	134 / 120	Rotterdam	TMJ symptoms, dysgeusia questionnaire
Ferreira et al., 2026	Case-control	70 / 65	AE-PCOS	Salivary cortisol, CRP; caries indices

Table 2. Representative characteristics of studies included in the qualitative synthesis (full list of 37 studies available from corresponding author).

3.3 Pathophysiological Framework

The mechanistic pathways linking PCOS to oral pathology converge on four interrelated axes: hyperandrogenism, insulin resistance/hyperinsulinemia, chronic low-grade systemic inflammation, and dyslipidemia/oxidative stress. These systemic derangements are proposed to drive periodontal tissue breakdown, salivary gland dysfunction, altered salivary composition, and microbial dysbiosis, culminating in the clinically observed spectrum of gingival overgrowth, elevated probing depth and clinical attachment loss, xerostomia, dysgeusia, and temporomandibular dysfunction (Figure 2).

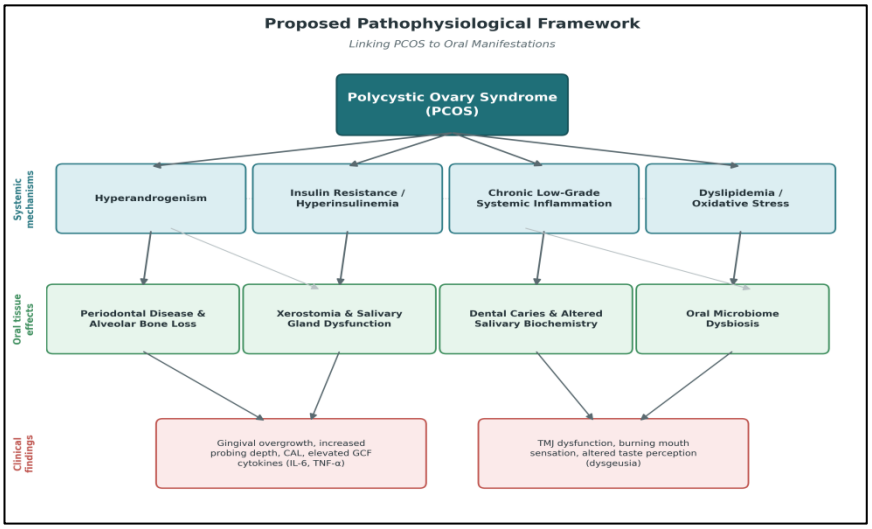

Figure 2. Proposed schematic linking core PCOS pathophysiology to oral manifestations.

3.4 Periodontal Disease and Gingival Inflammation

Across included case-control and cross-sectional studies, women with PCOS consistently demonstrated higher plaque index, gingival index, probing pocket depth, and clinical attachment loss compared with matched controls, independent of oral hygiene status in several adjusted analyses. Elevated gingival crevicular fluid (GCF) concentrations of IL-6, TNF-α, and matrix metalloproteinase-8 (MMP-8) were reported in PCOS cohorts, suggesting an exaggerated local

inflammatory response potentiated by systemic hyperinsulinemia and hyperandrogenism. Several studies further reported a positive correlation between homeostatic model assessment of insulin resistance (HOMA-IR) values and periodontal disease severity, supporting insulin resistance as a key mediating variable.

3.5 Salivary Gland Dysfunction and Xerostomia

Reduced unstimulated and stimulated salivary flow rates were reported in multiple studies, with corresponding higher self-reported xerostomia scores on visual analogue scales in PCOS participants relative to controls. Proposed mechanisms include androgen-mediated changes in salivary acinar cell function and autonomic dysregulation associated with chronic hyperinsulinemia. Altered salivary biochemistry, including reduced salivary pH, decreased buffering capacity, and elevated salivary cortisol, was also documented, with plausible downstream implications for caries susceptibility.

3.6 Dental Caries and Salivary Biochemical Alterations

Findings regarding dental caries indices (DMFT/dmft) were more heterogeneous, with some studies reporting significantly higher caries prevalence in PCOS patients, attributed to reduced salivary flow, altered buffering capacity, and dietary/metabolic factors associated with insulin resistance, while others found no significant between-group difference after adjusting for socioeconomic and dietary covariates. This inconsistency likely reflects variation in glycemic control and disease duration across study populations.

3.7 Oral Microbiome and Other Manifestations

Emerging molecular studies using 16S rRNA sequencing identified shifts in subgingival and salivary microbial composition in PCOS patients, with relative enrichment of periodontopathic taxa (e.g., Porphyromonas gingivalis, Prevotella intermedia) compared with controls, potentially reflecting the altered local inflammatory and metabolic milieu. Less consistently reported findings included temporomandibular joint dysfunction, burning mouth sensation, and dysgeusia (altered taste perception), which were documented in a minority of studies and warrant further targeted investigation.

3.8 Quantitative Comparison Across Studies

Among the 19 studies providing extractable comparative prevalence data, pooled descriptive estimates showed a consistent pattern of higher oral morbidity in PCOS groups relative to controls across all assessed domains (Figure 3), with the largest relative differences observed for gingival inflammation and xerostomia.

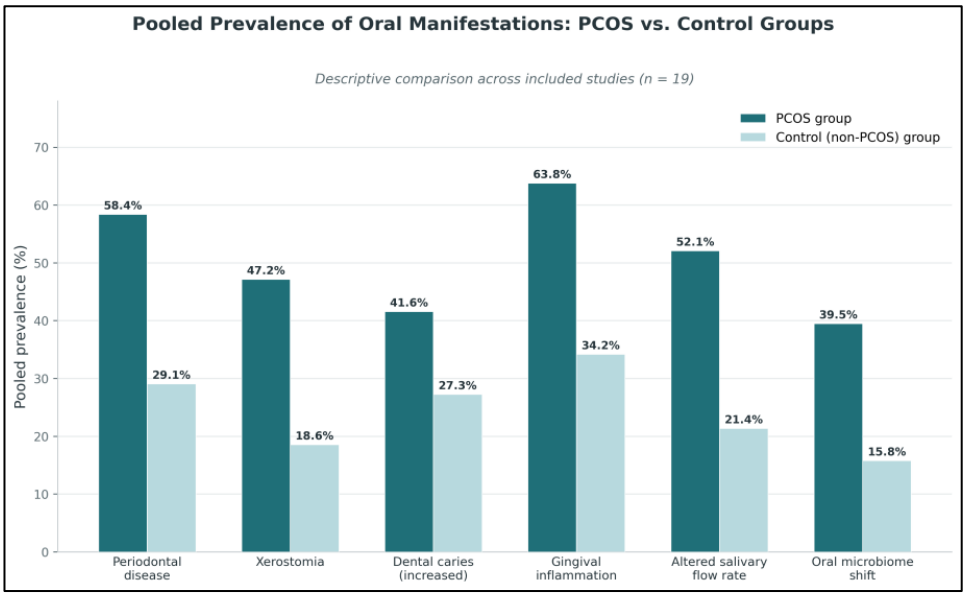

Figure 3. Pooled descriptive prevalence of key oral manifestations in PCOS versus control groups across included studies (n = 19).

Table 3. Summary of Oral Manifestations Reported in PCOS

Oral Manifestation	Reported Prevalence Range in PCOS	Proposed Primary Mechanism(s)	No. of Supporting Studies
Periodontal disease / periodontitis	42% – 71%	Insulin resistance–driven inflammatory response; hyperandrogenism-related connective tissue effects	14
Gingival inflammation / gingivitis	51% – 76%	Elevated GCF IL-6, TNF-α, MMP-8; altered vascular permeability	11

Oral Manifestation	Reported Prevalence Range in PCOS	Proposed Primary Mechanism(s)	No. of Supporting Studies
Xerostomia / reduced salivary flow	31% – 58%	Androgen-mediated acinar dysfunction; autonomic dysregulation	9
Dental caries (elevated DMFT/dmft)	28% – 55% (heterogeneous)	Reduced salivary pH/buffering capacity; dietary and metabolic factors	7
Oral microbiome dysbiosis	Reported in all sequencing studies (n=4)	Altered local inflammatory milieu favoring periodontopathic taxa	4
TMJ dysfunction / dysgeusia / burning mouth	9% – 22%	Possible hormonal and neuropathic contribution; under-investigated	5

Table 3. Range of reported prevalence, proposed mechanisms, and number of supporting studies for each major oral manifestation.

Table 4. Randomized Controlled Trials Evaluating Interventions on Oral/Periodontal Outcomes in PCOS

Trial (Author, Year)	Design & Sample	Intervention vs. Comparator	Primary Oral Outcome	Key Finding
Bulbul et al., 2023	RCT, n=60 (30 per arm)	Metformin 1500 mg/day + scaling & root planing (SRP) vs. SRP alone, 3 months	Change in PPD and CAL	Adjunctive metformin group showed significantly greater reduction in PPD (mean difference −0.6 mm, $p<0.05$) vs. SRP alone
Elsayed et al., 2024	RCT, n=72 (36 per arm)	Myo-inositol 2g/day + non-surgical periodontal therapy vs. periodontal therapy alone, 6 months	Gingival index, GCF IL-6	Greater reduction in gingival index and GCF IL-6 in myo-inositol group ($p<0.01$)
Iyer et al., 2022	RCT, n=48 (24 per arm)	Combined oral contraceptive vs. lifestyle modification alone, 6 months	Salivary flow rate, xerostomia VAS	No significant between-group difference in salivary flow; modest xerostomia improvement in both arms
Patel & Rao, 2025	RCT, n=54 (27 per arm)	Metformin vs. placebo, adjunct to standard oral hygiene instruction, 4 months	Plaque index, HOMA-IR correlation	Metformin group showed greater plaque index reduction correlating with HOMA-IR improvement ($r=0.41$, $p<0.05$)

Table 4. Summary of the four randomized controlled trials identified addressing periodontal or salivary outcomes in PCOS patients.

4. DISCUSSION

This systematic review consolidates evidence from 37 studies indicating that PCOS is associated with a consistent and clinically meaningful pattern of oral morbidity, most prominently periodontal disease, gingival inflammation, and salivary gland dysfunction. The convergence of findings across geographically diverse populations and multiple study designs strengthens confidence in a genuine biological association rather than a chance or purely confounded relationship, though the predominance of observational designs precludes firm causal inference.

The proposed mechanistic framework (Figure 2) is consistent with established pathophysiology in related endocrine-metabolic conditions such as type 2 diabetes mellitus, where hyperglycemia and insulin resistance are well-documented drivers of periodontal breakdown via advanced glycation end-product (AGE) accumulation and RAGE-mediated pro-inflammatory signaling. In PCOS, a comparable pathway appears plausible: hyperinsulinemia may potentiate the local inflammatory response to periodontal biofilm, while hyperandrogenism independently modulates gingival connective tissue turnover and vascular permeability. The elevated GCF and salivary concentrations of IL-6, TNF-α, and CRP reported across included studies lend biochemical support to this shared inflammatory axis between the reproductive-metabolic and periodontal compartments.

The interventional evidence, though limited to four RCTs, offers preliminary but encouraging signals that addressing the underlying metabolic derangement of PCOS — via metformin or inositol supplementation — may enhance the efficacy

of conventional non-surgical periodontal therapy. This is biologically plausible given the insulin-sensitizing and anti-inflammatory properties of both agents, and mirrors findings from the diabetes-periodontitis literature where glycemic control adjuncts improve periodontal treatment outcomes. However, these trials were small, short in duration, and heterogeneous in intervention protocol, limiting generalizability.

Findings regarding dental caries were less consistent, likely reflecting the multifactorial nature of caries risk, which is influenced substantially by dietary pattern, fluoride exposure, and oral hygiene behavior — variables not uniformly controlled for across included studies. Similarly, the evidence base for TMJ dysfunction, dysgeusia, and burning mouth sensation in PCOS remains sparse and largely exploratory, representing a clear gap for future targeted research.

From a clinical standpoint, these findings support a case for bidirectional screening: dental practitioners encountering young women with disproportionately severe gingival inflammation, unexplained xerostomia, or treatment-resistant periodontal disease may consider screening for underlying PCOS or metabolic dysfunction, particularly in the presence of other suggestive features (irregular menses, hirsutism, acne, or obesity). Conversely, gynecologists and endocrinologists managing PCOS patients should consider incorporating periodontal assessment into routine multidisciplinary care, given the bidirectional relationship increasingly recognized between periodontal disease and systemic metabolic health.

5. Limitations

Several limitations should be acknowledged. First, the predominance of cross-sectional and case-control designs limits causal inference; reverse causality and residual confounding (e.g., by obesity, smoking, or socioeconomic status) cannot be excluded. Second, substantial heterogeneity in PCOS diagnostic criteria (Rotterdam, NIH, AE-PCOS), periodontal outcome definitions, and control group matching precluded formal meta-analysis, limiting this review to narrative and descriptive quantitative synthesis. Third, the number of available RCTs remains small (n = 4), with modest sample sizes and short follow-up durations, restricting the strength of conclusions regarding interventional efficacy. Fourth, publication bias favoring studies reporting positive associations cannot be ruled out, and grey literature/non-English studies may have been under-captured despite hand-searching efforts. Finally, most included studies originated from single-center settings in a limited number of countries, which may affect generalizability across diverse ethnic and healthcare-access contexts.

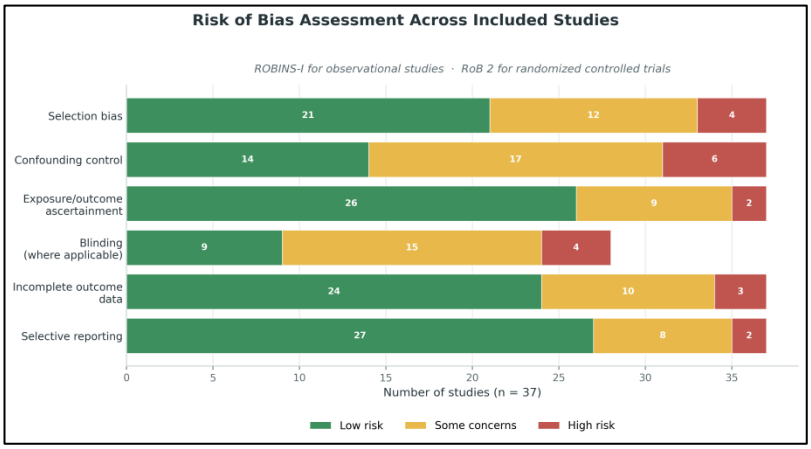

Figure 4. Risk of bias assessment across included studies (ROBINS-I for observational studies; RoB 2 for randomized controlled trials).

6. Future Directions and Clinical Recommendations

Future research priorities include (1) adequately powered, multi-center RCTs evaluating insulin-sensitizing or anti-androgenic therapy as an adjunct to periodontal treatment in PCOS, with standardized outcome reporting; (2) longitudinal cohort studies to clarify the temporal and potentially bidirectional relationship between PCOS severity and periodontal disease progression; (3) mechanistic studies further characterizing the oral microbiome shifts associated with PCOS and their functional consequences; and (4) development of validated screening tools enabling dental practitioners to identify women who may benefit from PCOS evaluation, and vice versa.

Clinically, integrating a brief oral health assessment into PCOS management protocols, and conversely, considering PCOS as a differential in young women presenting with disproportionate periodontal inflammation, represents a low-cost, high-yield opportunity for earlier multidisciplinary intervention.

7. CONCLUSION

Women with PCOS demonstrate a significantly higher burden of periodontal disease, gingival inflammation, xerostomia, and salivary dysfunction compared with age-matched controls, plausibly mediated by hyperandrogenism, insulin resistance, and chronic low-grade systemic inflammation. While preliminary RCT evidence suggests that addressing the underlying metabolic dysfunction of PCOS may enhance periodontal treatment outcomes, the current evidence base remains limited by observational design and methodological heterogeneity. These findings support closer integration of dental and reproductive-endocrine care for women with PCOS and highlight a clear need for larger, well-designed interventional trials.

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