Abstract: Background: Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders in women. It is believed that sex hormones play a role in the maintenance of bone mass and directly or indirectly influence several cell types, including periodontal cells. Objective: To evaluate the association between periodontal disease and PCOS according to the evidence reported in the last decade. Material and Method: A search was made in the biomedical databases: Pubmed, Embase, Scopus, SciELO, Science Direct and SIGLE for the 2007-2017 period. Selection criteria: prospective and retrospective studies reporting the relationship between periodontal disease and PCOS. The methodological quality of the studies was analyzed using the Critical Appraisal Skills Program scale. Results: 10 articles were found: 1 clinical trial and 9 case-control studies. The number of patients ranged from 48 to 196, mean age between 23.3 and 28.1 years, age range between 15 and 45 years. Studies were conducted in Turkey, India and Iran. All the studies presented good methodological quality and a positive association between PCOS and periodontal disease. Conclusion: PCOS shows a positive and significant association with the clinical and molecular parameters of periodontal diseases.

Keywords: polycystic ovary syndrome; periodontal disease; gingivitis; periodontitis; revision.

INTRODUCTION.

Polycystic ovary syndrome (PCOS) is one of the multiple conditions that affect women, and one of the most frequent endocrine disorders.1-17 It is characterized by menstrual abnormalities, hyperandrogenism, polycystic ovary and increased risk of developing metabolic and cardiovascular diseases.2,5-18 PCOS is likewise the main cause of infertility,5,10,11,16,18 and is reciprocally associated with oral health.11,17

Periodontal disease consists of a group of conditions that affect the protective (gingiva) and support tissues (periodontal ligament, root cement and alveolar bone) of the teeth. It is caused by persistent infection and inflammation in response to the presence of periodontal pathogens.10-16,19-21 Currently, more than 500 bacterial species have been identified in periodontal plaque, but there is no agreement on the causative bacterial species.19,22,23 The presence of periodontal pathogens is a necessary but not sufficient condition to induce periodontal disease. In a physiological state where there are no disease-modifying risk factors, the host responds appropriately to the growth of bacteria by trying to restrain the infection.

However, disease modifiers, such as smoking and diabetes mellitus, change immunoinflammatory responses putting them outside their normal physiological limits.9,17,20 In addition, the lipopolysaccharides (LPS) from
periodontal pathogens stimulate host cells to secrete proinflammatory mediators such as interleukin (IL) -1b, IL-6, IL-11 and IL-17; tumor necrosis factor alpha (TNF-α) and prostaglandin E2 (PGE2). These, in turn, stimulate the release of matrix metalloproteinases (MMPs).

It has been recently shown that women with PCOS have high levels of TNF-α due to insulin resistance (IR) and hyperandrogenism (HA); and high levels of MMPs and proinflammatory ILs. Therefore, it is plausible to assert that the severity of periodontal disease may be associated with that of PCOS. In addition, sex hormones (androgens, estrogens and progestins) play a role in the maintenance of bone mass, and directly and indirectly influence several cells through their receptors in target tissues, including periodontal cells.

Until now, very few studies have evaluated the relationship between periodontal parameters and PCOS. The aim of this scoping review is to evaluate the association between periodontal disease and PCOS according to the evidence reported in the last decade.

MATERIALS AND METHODS.

This review was carried out in accordance with a previously prepared research protocol based on PRISMA statement.

Search
A comprehensive search was carried out in the biomedical databases Pubmed, Embase, Scopus, SciELO, Science Direct, SIGLE (System of Information on Gray Literature in Europe) and a manual search was also conducted from January 2, 2007 to December 1, 2017, in the journals of periodontology with the greatest impact factor, such as: Periodontology 2000, Journal of Clinical Periodontology, Journal of Periodontology.

A combination of thematic headings was used including the following keywords: “polycystic ovary syndrome” OR “PCOS” OR “ovarian cysts” OR “síndrome de ovario poliquístico”) AND (“periodontal disease” OR “gingivitis” OR “periodontitis” OR “enfermedad periodontal”).

Selection criteria
Articles reporting the relationship between periodontal disease and PCOS, without language restriction, were included in the study. Case reports, case series and systematic reviews were excluded.

Process of selection and extraction of data
The titles and abstracts of each of the studies obtained were reviewed. The full texts of the studies that met these parameters were obtained in order to determine their risk of bias.

To assess the studies, a checklist was made in duplicate, in order to extract the information of interest. Two reviewers (LG and EI) independently carried out the evaluation of the articles regarding name, author, year of publication, type of study, number of patients, age of the patients, country where the study was carried out, groups of study and conclusions. For the resolution of any discrepancy between the reviewers, they met and discussed with a third reviewer (SR) until consensus was reached.

Assessment of methodological quality
The Critical Appraisal Skills Program scale (CASP) was used for the assessment of the methodological quality of each study. This tool is based on 11 criteria and there are several versions to be used according to study type, such as randomized controlled trials or case-control studies.

Figure 1. Flowchart of the selection process of articles.
<table>
<thead>
<tr>
<th>Author(s) and Year</th>
<th>Type of study</th>
<th>Country</th>
<th>Number</th>
<th>Mean age</th>
<th>Groups under study</th>
<th>Mean age of patients (range)</th>
<th>Results</th>
<th>Conclusions</th>
</tr>
</thead>
</table>
| Akcali 2017       | Case-control  | Turkey  | 125    | 25.7     | 45 with PCOS and healthy periodontium (Ph) | NR | Salivary levels of MMP-9 and NE, as well as the MMP-9/TIMP-1 ratio, were higher in the Ph group compared to the Pg group (p<0.05). The serum levels of MMP-8 were lower in the Ph group than in the Pg group (p<0.05). The groups with PCOS exhibited more gingivitis than systemically healthy women. No significant changes were observed in salivary or serum MMP-8 levels or the MMP-8/TIMP-1 ratio in the case of PCOS, while a negative correlation for TIMP-1 was found in systemically healthy women.
| Deepti 2017       | Randomized controlled | India | 60      | 23.3     | 26 with PCOS and periodontitis treated with Myo-inositol supplements (Control) | 22.6 | Periodontal parameters improved significantly in the test group compared to the control group at 3 and 6 months of follow-up (p<0.001). A statistically significant greater reduction of the systolic and diastolic BP was observed in the test group compared to the control group (p<0.05). The serum levels of MDA, CRP, and HOMA were lower in the test group compared to the control group at 3 and 6 months (p<0.05). The serum levels of HOMA were lower in the test group compared to the control group at 3 and 6 months (p<0.05). The serum levels of hsCRP were lower in the test group compared to the control group at 3 and 6 months (p<0.05). The serum levels of SAD were lower in the test group compared to the control group at 3 and 6 months. Both the test and the control groups showed a significant and constant improvement of the metabolic parameters at 3 and 6 months of follow-up, which was also comparable to a systemically and periodontally healthy group. Chronic periodontitis leads to an increase in serum and salivary levels of MMP-8 and MDA and periodontal treatment alone showed no significant improvement in salivary levels of 8-OHdG and MDA in the PCOS group compared to the systemically healthy group (p>0.05). There were no statistical differences between the SHCP and PCOSPH groups, but the serum levels of SAD were lower than those in the SHCP group.
| Saglam 2017       | Case-control   | Turkey  | 48     | 28.1     | 22 with PCOS and periodontitis (SHCP) | 27.28 | Salivary levels of 8-OHdG and MDA and the serum levels of 8-OHdG were statistically higher than those in both systemically healthy and PCOSPH groups (p<0.05). There were no statistical differences in salivary levels of MDA and TIMP-1 between the PCOSPH and PCOS groups (p>0.05). There were no statistical differences in serum levels of SAD between the PCOSCP and SPH groups (p>0.05). However, a significant improvement was observed in the serum levels of MDA and TIMP-1 in the test group compared to the control group at 3 and 6 months of follow-up (p<0.05). The serum levels of 8-OHdG and MDA in the PCOSPH group were lower than those in both systemically healthy and PCOS groups at 3 and 6 months (p<0.05). There were no statistical differences between the SHCP and PCOSPH groups, but the serum levels of SAD were lower than those in the SHCP group.
| Akcali 2015       | Case-control   | Turkey  | 125    | 39.9     | 46 with PCOS and healthy periodontium | 26.4 | Salivary levels of MMP-9 and TIMP-1 were higher in the Ph group compared to the Pg group (p<0.001). The serum levels of MMP-8 were lower in the Ph group than in the Pg group (p<0.05). The groups with PCOS exhibited more gingivitis than systemically healthy women. No significant changes were observed in salivary or serum MMP-8 levels or the MMP-8/TIMP-1 ratio in the case of PCOS, while a negative correlation for TIMP-1 was found in systemically healthy women.
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Country</th>
<th>Age Range (y)</th>
<th>Subjects</th>
<th>PCOS Group Details</th>
<th>Controls Group Details</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rahiminejad et al. 2015</td>
<td>Case-control</td>
<td>Iran</td>
<td>18-45</td>
<td>196</td>
<td>98 with PCOS</td>
<td>98 systemically healthy</td>
<td>The number of sites with loss of clinical insertion and clinical attachment loss was significantly higher in women with PCOS (p&lt;0.05). However, no significant difference was observed in the rate of tooth loss among women with and without PCOS (p=0.384). In the PCOS group, 92 women (93.9%) were affected by mild periodontitis and 6 (6.1%) were diagnosed with moderate periodontitis. Mild periodontitis was observed in 97 women (99%) in the control group, while only one (1%) had moderate periodontitis. There was no significant difference between these groups in terms of periodontitis severity (p=0.118).</td>
</tr>
<tr>
<td>Akcali et al. 2014</td>
<td>Case-control</td>
<td>Turkey</td>
<td>15-45</td>
<td>125</td>
<td>45 with PCOS and healthy periodontium</td>
<td>25 systemically and periodontally healthy</td>
<td>In women with PCOS, the salivary levels of Porphyromonas gingivalis, Fusobacterium nucleatum, Streptococcus oralis and Tannerella forsythia were higher than in systemically healthy women, particularly in the case of gingivitis. The levels of Aggregatibacter actinomycetemcomitans and Treponema denticola were similar between the groups under study. The presence of PCOS also increased serum antibody levels against P. gingivalis, Prevotella intermedia and S. oralis, when gingivitis was also present. Gingival inflammation correlated positively with the levels of the taxa studied in saliva, particularly in women with PCOS. The presence of P. gingivalis and F. nucleatum in saliva also showed a strong positive correlation with the corresponding serum antibody levels.</td>
</tr>
<tr>
<td>Porwal et al. 2014</td>
<td>Case-control</td>
<td>India</td>
<td>15-36</td>
<td>126</td>
<td>41 with PCOS in treatment</td>
<td>40 systemically healthy</td>
<td>Women with recently diagnosed PCOS had increased bleeding on probing (BP), probing depth (PD), loss of clinical insertion level (LCIL), waist circumference (WC), hsCRP, and prevalence of periodontitis compared with control and PCOS groups in treatment (p&lt;0.05). In the partial correlation analysis after controlling for confounding factors, it was observed that BP and LCIL correlated positively and significantly with hsCRP (p=0.01 and p=0.005). The multivariate linear regression analysis revealed that BP and LCIL (dependent variable) (p=0.009/R2=0.05 and p=0.005/R2=0.07, respectively) had a significant association with hsCRP. In addition, women with PCOS had increased bleeding on probing (BP), probing depth (PD), loss of clinical insertion level (LCIL), waist circumference (WC), hsCRP, and prevalence of periodontitis compared with control and PCOS groups in treatment (p&lt;0.05). In the partial correlation analysis after controlling for confounding factors, it was observed that BP and LCIL correlated positively and significantly with hsCRP (p=0.01 and p=0.005). The multivariate linear regression analysis revealed that BP and LCIL (dependent variable) (p=0.009/R2=0.05 and p=0.005/R2=0.07, respectively) had a significant association with hsCRP. In addition, when hsCRP is considered as a result, it also showed association with LCIL and WC (p=0.002/R2=0.07 and p=0.04/R2=0.106). The logistic regression analysis showed that the group with PCOS was 2.88 times more likely to suffer from moderate periodontitis (adjusted odds ratio 2.88, 95% confidence interval = 1.18 to 6.98).</td>
</tr>
<tr>
<td>Özcaka et al. 2013</td>
<td>Case-control</td>
<td>Turkey</td>
<td>15-42</td>
<td>73</td>
<td>30 with PCOS and gingivitis</td>
<td>12 systemically and periodontally healthy</td>
<td>The multivariate analysis of the general linear model, adjusted for age or plaque index, showed that the two groups with PCOS had higher concentrations of IL-17A, IL-17F and IL-17A/F in serum and higher levels of IL-17A and IL-17F in crevicular fluid and saliva, but lower IL-17E in serum than systemically healthy women. IL-17E levels were lower in women with PCOS and gingivitis who also had the highest FGS. Serum levels of IL-17A and IL-17F correlated positively with FGS and depth of periodontal probing (all r &gt; 0.33, p&lt;0.005). The IL-17E serum showed an inverse relationship and was also negatively correlated with IL-17A (r &gt; -0.28, p&lt;0.005).</td>
</tr>
</tbody>
</table>

PCOS quantitatively affects the composition of the oral microbiota and the elevated systemic response to the selective members of this microbial community, playing a role in the resulting gingival inflammation and in periodontal health. The most consistent effect is exerted on Pg.
The PCOS+gingivitis group showed significantly higher concentrations of IL-6 in crevicular fluid, saliva and serum than those in the PCOS+healthy periodontium group (p<0.0001). The two groups of PCOS exhibited significantly higher concentrations of TNF-α in saliva than the control group (p=0.024 and p=0.013, respectively). The FGS index was significantly higher in the PCOS+gingivitis group than in the PCOS+healthy periodontium group (p=0.030). The PCOS+gingivitis group showed significantly higher insulin concentration than the PCOS+healthy periodontium and control groups (p=0.014 and p<0.0001, respectively). Serum levels of TNF-α, TNF-αRs and serum, crevicular fluid and salivary IL-6 were correlated with clinical periodontal measurements.

The average BMI was higher in the PCOS group (p=0.01). The PCOS group had a higher tT, a lower SHBG and higher levels of FAI (p<0.001 for all). The evaluation of the homeostasis model of IR and glucose-120 were higher in women with PCOS (p<0.003, p=0.009, respectively). The levels of TC and HDL-C were not different between the groups, while TG levels were higher among women with PCOS (p=0.02). In women with PCOS, the clinical periodontal parameters and the volume of crevicular fluid (subclinical sign of gingival inflammation) were higher than in the control group. There were no significant differences between the groups with respect to gingival index (p=0.14) and plaque index (p=0.86). MPO and NO levels were higher in the PCOS group (p=0.019 and p=0.02, respectively), while the difference in NO levels between the groups was not significant (p=0.71). There were significant positive correlations between clinical periodontal parameters, MPO and NO levels and serum parameters. Fasting insulin and glucose-120 levels correlated with the parameters of gingival inflammation: fasting gingival-insulin index (r=0.29, p=0.04), gingival-glucose-120 index (r=0.29, p=0.04), gingival-glucose-120 index (r=0.36, p=0.009) and volume of crevicular fluid-glucose-120 (r=0.37, p=0.007). The volume of crevicular fluid was correlated with tT (r=0.28, p=0.04) and FAI (r=0.30, p=0.03).

In conclusion, susceptibility to periodontitis can increase significantly in patients with PCOS, and gingivitis is a common finding; the local/periodontal oxidation state seems to be affected in PCOS.
### Table 2: Diagnostic methods, confounding factors, and altered clinical parameters of periodontal disease in patients with PCOS

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Diagnostic methods of PCOS</th>
<th>Confounding factors evaluated and periodontal disease</th>
<th>Altered clinical parameters in patients with PCOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Akcali et al.</td>
<td>Oral examination, PD, BP, PI, X-rays, crevicular fluid</td>
<td>Medical history, Rotterdam criteria, hyperandrogenism, diabetes mellitus, hyperprolactinemia, congenital adrenal hyperplasia, thyroid disorders, Cushing's syndrome, hypertension, hepatic or renal dysfunction, cardiovascular disease, diabetes mellitus type 1 or type 2, cancer, smoking, alcohol consumption, systemic inflammatory conditions, oral contraceptive, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors</td>
<td>Serum and salivary levels of MMP-9, TIMP-1, MPO, and NE with ELISA, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
</tr>
<tr>
<td>Saglam et al.</td>
<td>Oral examination, PD, BP, PI, X-rays</td>
<td>Medical history, Rotterdam criteria, ultrasound, waist circumference, waist-hip ratio, BMI&gt;25 kg/m², pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
<td>Serum and salivary levels of MMP-9, TIMP-1, MPO, and NE with ELISA, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
</tr>
<tr>
<td>Özcaka et al.</td>
<td>Oral examination, PD, BP, PI, X-rays, crevicular fluid</td>
<td>Medical history, Rotterdam criteria, ultrasound, serum and salivary samples, saliva samples, levels and FGS with spectrophotometry, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
<td>Serum and salivary levels of MMP-9, TIMP-1, MPO, and NE with ELISA, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
</tr>
<tr>
<td>Dursun et al.</td>
<td>Oral examination, PD, BP, PI, X-rays, crevicular fluid</td>
<td>Medical history, Rotterdam criteria, ultrasound, serum and salivary samples, saliva samples, levels and FGS with spectrophotometry, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
<td>Serum and salivary levels of MMP-9, TIMP-1, MPO, and NE with ELISA, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
</tr>
<tr>
<td>Özcaka et al.</td>
<td>Oral examination, PD, BP, PI, X-rays, crevicular fluid</td>
<td>Medical history, Rotterdam criteria, ultrasound, serum and salivary samples, saliva samples, levels and FGS with spectrophotometry, pregnancy, smoking, Cushing's syndrome, non-classical congenital adrenal hyperplasia, androgen-secreting tumors, antibiotics, oral contraceptives, steroid hormones, and insulin-sensitizing medications</td>
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</tr>
</tbody>
</table>

**Abbreviations:**
- **PI:** Periodontal Index
- **BP:** Blood Pressure
- **PD:** Periodontal Disease
- **CAL:** Clinical Attachment Level
- **GI:** Gingival Index
- **BMI:** Body Mass Index
- **Ferriman-Gallwey scale:**
- **IL:** Interleukin
- **TNF-α:** Tumor Necrosis Factor-α
- **MPO:** Myeloperoxidase
- **NE:** Neutrophil elastase
- **NO:** Nitric oxide
- **FGS:** Ferriman-Gallwey scale
- **MMP:** Matrix metalloproteinase
- **TIMP:** Tissue inhibitor of metalloproteinases
- **ELISA:** Enzyme-linked immunosorbent assay
- **IFMA:** Immunofluorometric assay
- **HOMA:** Homeostatic model assessment
- **8-OHdG:** 8-Oxoguanine
- **MDA:** Malondialdehyde
- **TAS:** Total antioxidant status
- **Pg:** Porphyromonas gingivalis
- **Fn:** Fusobacterium nucleatum
- **Pi:** Prevotella intermedia
- **NR:** Not reported

**Notes:**
- Association between periodontal disease and polycystic ovary syndrome: A scoping review.
- Direito H, Rojas S, Gamarra LG & Infantes ED.

**References:**
RESULTS.

The selection process of the articles is shown in Figure 1. After that, ten articles were selected for a thorough review of their content.

The number of patients ranged from 48 to 196 in the included studies, mean age ranged from 23.3 to 28.85 years, and the age range was between 15 and 45 years. The 10 selected studies were conducted in: Turkey, India, and Iran. Nine studies were cases-controls and one study was a randomized controlled trial. Characteristics, results and conclusions of the included studies are shown in Table 1.

In nine studies, PCOS was diagnosed according to the Rotterdam 2003 criteria, requiring having 2 out of 3 of the following factors: 1) clinical or biochemical HA, 2) chronic oligo-anovulation (OA), 3) polycystic ovaries (PO) (12 follicles in each ovary measuring 2-9mm in diameter and/or ovarian volume >10ml), excluding other etiologies. In one study, PCOS was diagnosed according to the criteria of the Androgen Excess and PCOS Society (AES-PCOS) published in 2006, which include the presence of HA (necessary condition) in combination with ovarian dysfunction (i.e. OA or ultrasound with PO), with the exclusion of other causes. Diagnostic methods, confounding factors and altered clinical parameters of the analyzed studies are shown in Table 2.

All the studies reached 10 out of a maximum of 11 points on the CASP scale.

DISCUSSION.

All the analyzed studies showed a positive association between PCOS and periodontal diseases (gingivitis and/or periodontitis). Therefore, it is possible to assert that patients with PCOS have a higher risk of developing periodontal disease. However, there are factors in all the studies that may have influenced the reported results.

First, there is the complexity and heterogeneity of PCOS, with different definitions. Nevertheless, PCOS is one of the most prevalent endocrinopathies and the main cause of HA. Second, the pathophysiology of PCOS is multifactorial, involving a metabolic and endocrine component related to IR, apolypgenic component, intrauterine environmental influences, alterations in ovarian and adrenal steroidogenesis, neuroendocrine dysfunction, and environmental factors (dietary pattern, physical activity, smoking and stress). None of these factors alone can explain the spectrum of alterations that characterize the syndrome.

Third, the diagnosis of PCOS is based on the Rotterdam criteria, consisting of a combination of clinical, biological and ultrasound evaluations. Fourth, heterogeneity in the clinical expression of the condition makes diagnosis difficult, even more so with metabolic comorbidities and reproductive disorders that are frequently associated with this syndrome (obesity, type 2 diabetes, cardiovascular disease, venous thromboembolism, infertility, endometrial hyperplasia, sleep apnea, endometrial cancer, ovarian cancer, mood disorders, etc.).

Given that the clinical presentation of PCOS varies between continents, it is difficult to establish a universal diagnosis using only European or North American guidelines. Because of the common ultrasound finding of polycystic ovaries in healthy women, the inclusion of this sign in the diagnostic criteria of PCOS is still debatable.

A relevant aspect of this review was the finding of a clinical trial that showed that the integral treatment of periodontal disease could also contribute to the treatment of patients with PCOS by reducing the levels of proinflammatory mediators, reactive oxygen species and oxidative stress. Therefore, future evaluation of periodontal disease in patients with PCOS should explore in greater depth the effect of non-surgical periodontal therapy on the improvement of inflammatory parameters and the severity of PCOS.

It should also be noted that eight studies included strict inclusion and exclusion criteria in order to restrict confounding factors. For example, women with a BMI>25 kg/m² were defined as obese and excluded from a study. In addition, Deepti et al., reported that Asian young adult women (all studies included in the review were conducted in Asia) tend to have lower BMI and higher percentage of body fat than other ethnic groups, so it was suggested to combine the BMI and the analysis of biometric impedance for the detection of obesity and overweight in young Asian adults. Therefore, it is hypothesized that periodontal clinical parameters...
REFERENCES.


