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POLY CYSTIC OVARIAN SYNDROME AND PERIODONTITIS – CURRENT INSIGHTS

Kasukurthi Komali[1], Syed Afzal[2], Suryakanth Malgikar[3*], Raja Babu Palaparthy[4] and Juliet Josephine Johnson[5]

[1,2]PG Student, Department of Periodontology, Kamineni Institute of Dental Sciences, Narketpally, Nalgonda (Dist.), Telangana, India.

[3,5]Professor, Department of Periodontology, Kamineni Institute of Dental Sciences, Narketpally, Nalgonda (Dist.), Telangana, India.

[4]Professor and Head, Department of Periodontology, Kamineni Institute of Dental Sciences, Narketpally, Nalgonda (Dist.), Telangana, India.

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***Corresponding Author**
Suryakanth Malgikar
Professor, Department of Periodontology, Kamineni Institute of Dental Sciences, Narketpally, Nalgonda (Dist.), Telangana, India.

ABSTRACT

Polycystic Ovarian Syndrome (PCOS) is a common endocrine disorder that affects women of reproductive age, characterized by irregular menstrual cycles, hyperandrogenism, and polycystic ovaries. Periodontitis, a chronic inflammatory disease of the gums and supporting structures of the teeth, has been increasingly linked to various systemic conditions, including PCOS. This connection is thought to be mediated by the underlying hormonal and metabolic disturbances associated with PCOS, such as insulin resistance, hyperglycemia, and altered inflammatory pathways. Women with PCOS are at a higher risk of periodontal diseases due to the pro-inflammatory environment and altered immune response seen in this condition. Studies have shown that periodontal disease severity in PCOS patients may be more pronounced compared to women without the condition. Additionally, the presence of periodontitis may exacerbate the metabolic and endocrine dysfunctions of PCOS, leading to a bidirectional relationship between the two. Effective management of both PCOS and periodontitis requires an interdisciplinary approach, emphasizing the importance of early detection, oral hygiene, and metabolic control. This review aims to explore the potential mechanisms linking PCOS

and periodontitis, highlighting the need for greater awareness and targeted interventions in managing these interconnected health concerns.

KEYWORDS: Poly cystic ovarian syndrome, Periodontitis, Cytokines, Inflammation.

INTRODUCTION

One of the most common infectious conditions is periodontal disease. The defining feature of periodontitis, a chronic multifactorial inflammatory condition, is the progressive deterioration of the structures supporting the teeth, which is associated with dysbiosis in plaque biofilms.[1] The key signs include gingival bleeding, the presence of periodontal pockets, loss of alveolar bone as measured radiographically, and clinical attachment loss (CAL), all indicating a reduction in periodontal tissue support; it is considered the 11th most widespread disease globally.[2] Moreover, periodontal diseases are recognized as known or potential risk factors for various systemic conditions, as well as during pregnancy. In industrialized nations, severe forms of periodontitis impact a small segment of the population, roughly 10–15%, although its prevalence rises with age, peaking around 50–60 years.[3]

Polycystic ovary syndrome (PCOS) is a genetically intricate endocrine, reproductive, and metabolic disorder that impacts between 5% and 15% of women of reproductive age globally.[4] It is marked by excessive levels of androgens along with irregular gonadotropin secretion, high insulin levels, dysfunction of the hypothalamic-pituitary-ovarian (HPO) axis, problems with ovulation and menstruation, and the presence of cystic ovaries.[5] Additionally, PCOS seems to be linked to a higher risk of metabolic syndrome (MetS), which includes type 2 diabetes mellitus and insulin resistance.[6] as well as dyslipidemia.[7] cardiovascular issues.[8] and non-alcoholic fatty liver disease.[9]

Etiology and pathophysiology of polycystic ovary syndrome

The causes and physiological mechanisms underlying PCOS are intricate, arising from the interplay of genetic, metabolic, prenatal, and environmental influences. Studies indicate that the condition begins within the intrauterine environment, highlighting the significance of genetic elements.[10] However, Franks and Berga et al. suggested in 2012 that genetic influences contribute only partially to the development of PCOS.[11] According to Abbott et al. in 2005, the observable traits of PCOS might arise as a result of genetically driven excessive androgen production by the ovaries.[12] A concern associated with PCOS is genetic susceptibility. Changes such as alterations, deletions, or inversions in single nucleotide

polymorphisms (SNPs) can contribute to the onset of PCOS. One study identified SNP rs2414096 of the CYP19 gene as a factor responsible for the development of PCOS.[13] The SNP rs4077582 in the CYP11A1 gene can increase androgen levels, which may worsen PCOS. Additionally, polymorphisms in the CYP19A1 gene associated with PCOS have been linked to endometrial and breast cancer.

Poly Cystic Ovarian Disease and Periodontitis

The principle element in the development of polycystic ovarian syndrome (PCOS) and periodontitis is inflammation. Thus, the connection between PCOS and periodontal disease (PD) was originally identified due to their shared association with metabolic syndrome (MetS). Research conducted by Dursun et al. in 2011 found that women with PCOS exhibited increased levels of periodontal inflammation and damage compared to their non-PCOS counterparts, along with significant indicators of oxidative stress and glycemic dysregulation in gingival crevicular fluid. In fact, diabetes mellitus and oxidative stress are recognized risk factors for periodontal diseases and may play a critical role in the relationship between PCOS and PD.[14] Furthermore, metabolic syndrome could be instrumental by promoting systemic inflammation and insulin resistance.[15]

A number of indicators of inflammation, including C-reactive protein (CRP), proinflammatory cytokines and chemokines like interleukin 18 (IL-18), monocyte chemoattractant protein-1 and macrophage inflammatory protein-1, and white blood count, are elevated in PCOS, which is linked to low-grade systemic inflammation. Furthermore, PCOS may be an inflammatory illness based on elevated oxidative stress and associated biomarkers.[16] Periodontitis is known to be a chronic inflammatory illness, and it is this inflammation that connects periodontitis to a number of systemic ailments.[17] Later, it was suggested that PD and PCOS might interact through a series of proinflammatory processes.[18] The severity of PD may also be linked to the exacerbation of PCOS, and both conditions seem to work in concert to raise the expression of proinflammatory cytokines.[19,20]

In general, the chronic subclinical inflammation of both PCOS and PD provides the basis for the potential mechanism connecting the two conditions. Stated differently, a chronic subclinical inflammatory state increases the levels of proinflammatory markers (like matrix metalloproteinase-9, TNF-α, C-Reactive Protein (CRP), interleukin (IL)-6, IL-17, and TNF-α) and intensifies an oxidative stress environment (by increasing local oxidant status, such as myeloperoxidase and nitric oxide).[21] As a result, PCOS-induced subclinical inflammation

may exacerbate periodontal degradation, while PD-induced subclinical inflammation may contribute to PCOS pathogenesis by directly invading the periodontal microbiota. Furthermore, as periodontal therapy reduced elevated glycaemic levels, PD management has been linked to insulin regulation.[22] As a result, unmanaged PD may have an indirect effect on the clinical state of PCOS.

However, high amounts of sex steroid hormones, which negatively affect PD, are a hallmark of PCOS.[23] because the gingival tissues contain androgens, estrogen, and progesterone receptors. Changes in estrogen and progesterone appear to alter the gingival tissues, increasing vascular permeability and decreasing gingival epithelial keratinization.[24] Even though a number of research have clarified the connection between PCOS and PD, it is still unclear how PD and infertility in PCOS-afflicted women are related.[25] A new study on women with both PCOS and PD evaluated the effect of typical Myo-inositol (MI) treatment for PD on systemic diseases, despite the dearth of longer prospective trials. In contrast to a therapy that exclusively used MI, the results of this randomized clinical trial demonstrated that treating PD with MI improved metabolic parameters and decreased systemic inflammation. However, this study was unable to determine if treating PD could have affected the fertility rate because it did not account for the extent of infertility among PCOS women at baseline.[26]

CONCLUSION

The reasonable influence of the PCOS-PD link on female infertility may stem from the fact that PCOS is linked to hyperandrogenism, hyperinsulinemia, and elevated PCOS levels, all of which have a detrimental effect on periodontal inflammation and destruction. Additionally, PD causes proinflammatory reactions and elevated PCOS levels, which dysregulate insulin resistance and impair ovarian function.

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