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Periodontal disease etiology and role of oxidative stress

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Abstract: *The term "periodontal disease" is used to describe an array of inflammatory conditions that can affect the periodontium. In response to the challenge given by the bacteria, the gingival and periodontal tissues undergo a complicated chain of events involving communication. It is between the membranes of a vital nature located under the gums and the person's immune inflammatory reaction. When there is an imbalance between the oxidants and the antioxidants in the body, this is known as oxidative stress. Antioxidants are compounds that may either seek out and destroy oxidants or prevent them from forming in the first place. Some of the oxidants produced by-products of normal cellular metabolism include reactive oxygen species and reactive nitrogen species. These byproducts are known as reactive oxygen and reactive nitrogen species, respectively. The presence of reactive oxygen species (ROS) has been shown to be associated with periodontal disease in research.*

Keywords: *Periodontal disease, Oxidative stress, reactive oxygen species (ROS), antioxidants, oxidants*

1. Introduction

Periodontal Disease

Diseases affecting the periodontium, which includes Some diseases of the gums, their connective tissue, and some diseases of the alveolar bone are referred to together as periodontal disease [1]. Both gingivitis and periodontitis fall under the umbrella term "periodontal disease"[2]. In response to the challenge offered by the bacteria, the gingival and periodontal tissues undergo an inflammatory response, which is the consequence of a complicated two-way dialogue between the subgingival biofilm and

the host immune system. Periodontitis is the clinical term for the immune-inflammatory response that leads to tissue damage. Even while gingivitis comes before periodontitis, it doesn't always lead to the latter. However, periodontitis is one of the things that affect the alveolar bone and periodontal ligaments associated with inflammatory processes[3]. Clinical loss of attachment and alveolar bone resorption are the end results of these inflammatory modifications to the periodontal ligament. [4].

Oxidative Stress

When there is an imbalance between the oxidants and the antioxidants in the body, this

is known as oxidative stress. Antioxidants are compounds that may either seek out and destroy oxidants or prevent them from forming in the first place. Enzymes like catalase, superoxide dismutase, and paraoxonase, along with non-enzymatic macromolecules like ferritin and albumin, make up these systems [5]. Both reactive oxygen species (ROS) and reactive nitrogen species (RNS) are byproducts of the normal cellular metabolism that are classified as oxidants. ROS and RNS stand for reactive oxygen and reactive nitrogen species, respectively. In the organism, ROS and RNS can have both beneficial and harmful effects [6]. Oxidative stress and nitrosative stress are the two main factors that lead to negative consequences and biological damage, the protective effects of ROS against microorganisms are most apparent at low concentrations [7].

Activation by cellular signaling molecules, proteins may be inactivated, DNA is damaged, and the cell membrane sustains partial damage, all of which contribute to tissue damage caused by oxidative stress and as in the soft tissues of the mouth, systemic oxidative stress, which affects the body Entirely possible. Periodontitis, diabetes, atherosclerosis, and rheumatoid arthritis (RA) share a common association with inflammation and oxidative stress as root causes. In addition, oxidative stress has been linked [8]. It's possible that reducing oxidative stress would be a major goal in the treatment of inflammatory disorders. More research is needed to better understand the link between periodontitis and systemic inflammatory disorders [9].

Oxidative stress markers are valuable diagnostic tools for gauging the human body's redox state, the presence and development of disease, and the curative benefits of antioxidants.

Examining the Connection between Oxidative Stress and Periodontal Disease

According to studies, between 10 and 15 percent of people worldwide suffer with

periodontal disease [10]. The primary contributor of this condition is the inflammatory and immunological response triggered by sub gingival plaque. Because they reflect periodontal disease's historical progression rather than its current activity, conventional clinical markers including bleeding on probing, clinical attachment loss, and probing pocket depth have limited utility in periodontal diagnosis [11]. Polymicrobial infections such periodontal disease are frequent [12]. ROS destroy connective tissue [13]. Periodontitis sufferers' gingival crevice phagocytes produce. Oxidative stress occurs when ROS and other oxidants are created faster than the cell's antioxidant response [15]. Reactive oxygen species and periodontal disease are linked [16, 17]. Periodontitis is caused by reactive oxygen species (ROS) and a variety of bacteria in periodontal pockets [18]. PG, Aa, TD, and TF all cause periodontal disease [19].

The disturbance of host immunological responses, with the participation of complement leukocytes and reactive oxygen species (ROS), is the most common factor that leads to injury to periodontal tissue [20]. This is according to a novel model of the etiology of periodontitis, which showed that pathogens alone are essential but inadequate to establish periodontal lesions. Excessive reactive oxygen species (ROS), produced primarily by neutrophils, will use cytotoxic activities, including disruption of cell development and prompting of apoptosis of gingival fibroblasts, leading to severe damage of periodontal tissues [21]. Physiological processes in eukaryotic cells, such as signal transduction, cell differentiation, and apoptosis, all rely on reactive oxygen species (ROS) [22, 23]. In addition, ROS contribute to the oxidative death of pathogens [24].

For instance, *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, and *Prevotella intermedia*-specific immunoglobulin G antibodies were positively linked with serum reactive oxygen metabolites levels in a clinical

investigation [25]. An oxidative stress response, which has been linked to periodontal damage [13, 26, 31], is triggered by a homeostatic imbalance between reactive oxygen species (ROS) and antioxidant defense systems. Total oxidant status (TOS) levels are positively correlated with periodontal parameters in clinical settings [8, 26]. Bone loss was slowed down as a result of decreased reactive oxygen species levels [27]. ROS can also induce immunological responses via redox-sensitive gene transcription factors like NF-B. [28]. Furthermore, ROS can activate c-Jun N-terminal kinase (JNK), leading to cellular death [29, 30].

Conclusion:

It is necessary to conduct additional study in this field since determining the level of oxidative stress and the quantity of periodontal bacteria may be an effective strategy for lowering the overall incidence of periodontal disease.

Ethics

This study was conducted under approval by the medical ethics committee at the University of Babylon. Verbal and written consent was provided by parents and agreement for publication was obtained from both participants and researchers.

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