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CHANGES IN MDA AND SOD LEVELS IN ORAL FLUID OF PATIENTS OF PERIODONTITIS WITH DIFFERENT QUERCETIN DRUGS

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Introduction. Infection is sensed by the host, which often leads to activation of the inflammasome, a cytosolic macromolecular signaling platform that mediates the release of the proinflammatory cytokines interleukin-1 (IL-1) and IL-18 and cleavage of the pore-forming protein gasdermin D, leading to pyroptosis [1]. The role of oxidative stress in periodontitis has been studied for decades. The main source of reactive oxygen species is thought to be neutrophils, which are the first line of defense against bacteria. During the process of respiratory inflammation, a superoxide radical is formed. This may then be released into the phagosomal and extracellular space causing the subsequent formation of other radical and non-radical derivatives [2, 3]. Periodontitis is an inflammatory disease affecting approximately 10% of the population. In essence, it is the destruction of periodontal tissues, and supporting tissues of the tooth. Its progression results in the loss of alveolar bone and premature tooth loss. Subsequently, external factors play a role, as well as the body's response. Thus, the disease is a complex of interactions between pathogenic microorganisms and the host immune response [2, 3, 4]. Modern predominant perception of the nanoscale lipid artefacts - liposomes as drug delivery systems (DDS), based on the ability of universal incorporation of active pharmaceutical ingredients (APl). Liposomes are nano- and micro-structured vesicles with a bilayered phospholipid membrane. In addition to these features, liposomes can be designed to increase the bacterial interaction by modification of superficial vesicle compounds [5, 6].

The aim of this study is to measure in MDA and SOD in patients with CGP of initial-I degrees of severity and assess the influence of periodontal treatment with gel from the Granules of Quercetin (GQ) and Liposomal Quercetin-Lecithin Complex (LQLC) on these parameters.

Material and Methods. Basic group of patients (BG) were treated with the basic treatment of periodontitis and local application of LQLC in the form of a suspension of 1/4 of a bottle with 5 ml of isotonic 0.9% sodium chloride,

heated to 38°C. Patients of the comparison group (CG) underwent basic treatment of periodontitis and local application of QG gel with the exposition of 40 mg a day for 10 days. QG gel and LQLC were placed in the periodontal delivery tray. Complex treatment has been performed for 18 patients of the BG and 17 patients of the comparative group (CG). MDA was $6,15 \pm 0,61 \mu\text{mol/l}$ in BG and $6,02 \pm 0,58 \mu\text{mol/l}$ in CG. SOD was $4,29 \pm 0,18$ c.u. in BG and $4,3 \pm 0,19$ c.u. in CG. In the BG after 1 month, MDA amounted to $4,73 \pm 0,57 \mu\text{mol/l}$ ($P < 0,05$), and with the use of gel with QG amounted to $4,95 \pm 0,51 \mu\text{mol/l}$ ($P < 0,05$). After 6 months, the MDA increased to $4,81 \pm 0,25 \mu\text{mol/l}$ and $4,86 \pm 0,43 \mu\text{mol/l}$ ($P > 0,05$), and after 1 year to $4,78 \pm 0,33 \mu\text{mol/l}$ and $4,91 \pm 0,55 \mu\text{mol/l}$, respectively ($P > 0,05$). The SOD after 1 month increased to $6,35 \pm 0,18$ c.u. ($P < 0,001$) with LQLC and to $5,81 \pm 0,21$ c.u. ($P < 0,001$) with the use of QG ($P > 0,05$). In case of stage initial-I CGP, the SOD decrease up to $5,51 \pm 0,18$ c.u. (LQLC) and $5,27 \pm 0,11$ (QG) was registered after 6 months ($P > 0,05$), and after 1 year - $5,42 \pm 0,13$ and $5,02 \pm 0,13$ c.u. accordingly ($P > 0,05$).

Conclusion. Considerable therapeutic efficacy of the LQLC for treatment patients with CGP, especially that of initial - I degrees of severity is based on its marked anti-inflammatory and periodontoprotecting effects.

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