Oxidative stress in the local and systemic events of apical periodontitis

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Oxidative stress is involved in the pathogenesis of a variety of inflammatory disorders. Apical periodontitis (AP) usually results in the formation of an osteolytic apical lesion (AL) caused by the immune response to endodontic infection. Reactive oxygen species (ROS) produced by phagocytic cells in response to bacterial challenge represent an important host defense mechanism, but disturbed redox balance results in tissue injury. This mini review focuses on the role of oxidative stress in the local and associated systemic events in chronic apical periodontitis. During endodontic infection, ligation of Toll-like receptors (TLRs) on phagocytes' surface triggers activation, phagocytosis, synthesis of ROS, activation of humoral and cellular responses, and production of inflammatory mediators, such as, cytokines and matrix metalloproteinases (MMPs). The increment in ROS perturbs the normal redox balance and shifts cells into a state of oxidative stress. ROS induce molecular damage and disturbed redox signaling, that result in the loss of bone homeostasis, increased pro-inflammatory mediators, and MMP overexpression and activation, leading to apical tissue breakdown. On the other hand, oxidative stress has been strongly involved in the pathogenesis of atherosclerosis, where a chronic inflammatory process develops in the arterial wall. Chronic AP is associated with an increased risk of cardiovascular diseases (CVD) and especially atherogenesis. The potential mechanisms linking these diseases are also discussed.

Apical lesion

Apical periodontitis

Atherosclerosis

Oxidative stress

ROS

gelatinase A

gelatinase B

immunoglobulin G antibody

immunoglobulin M antibody

matrix metalloproteinase

osteoclast differentiation factor

oxidized low density lipoprotein

toll like receptor

Aggregatibacter actinomycetemcomitans

apical periodontitis

atherogenesis

cardiovascular disease

chronic inflammation

coronary artery disease

cytokine production

cytokine release

endothelial dysfunction

human

mouth infection

nonhuman

osteoclastogenesis

oxidative stress

periodontal ligament

periodontitis

phagocytosis

Porphyromonas endodontalis

Porphyromonas gingivalis

Short Survey

vitamin D deficiency