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Tissue Damage and Oxidant/Antioxidant Balance

Doku Hasarı ve Oksidan/Antioksidan Denge

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Abstract

The oxidant/antioxidant balance in healthy tissues is maintained with a predominance of antioxidants. Various factors that can lead to tissue damage disrupt the oxidant/antioxidant balance in favor of oxidants. In this study, disruptions of the oxidant/antioxidant balance in favor of oxidants were found to be a consequence of the over-consumption of antioxidants. For this reason, antioxidants are considered to be of importance in the prevention and treatment of various types of tissue damage that are aggravated by stress.

Key Words: Antioxidant, Ischemia, Oxidant

Özet

Sağlıklı dokularda oksidan/antioksidan denge antioksidanların üstünlüğüyle sürdürülür. Doku hasarına yol açabilecek çeşitli agresif faktörler oksidan/antioksidan dengenin oksidanların leyhine bozulmasını sağlar. Bu derleme çalışmasında, oksidan/antioksidan dengenin oksidanlar leyhine bozulmasının, antioksidan sistemlerin aşırı harcanmasına bağlı olarak geliştiği rapor edilmiştir. Bu nedenle oksidatif strese bağlı çeşitli doku hasarının önlenmesinde ve tedavisinde antioksidanların önemli olduğu anlaşılmaktadır.

Anahtar Kelimeler: Antioksidan, İskemi, Oksidan

The oxidant/antioxidant balance in healthy tissues is maintained with a predominance of antioxidants [1]. A disruption of this balance causes tissue damage. This state is termed oxidative stress [2]. The occurrence of tissue damage is determined by the oxidant/antioxidant balance [3]. The oxidant/antioxidant balance changes in favor of oxidants in various damage models created in living tissues, and decreases in antioxidant levels are observed, whereas the oxidant levels increase [4]. Uzar et al. [5] reported decreases in the total antioxidant level and increases in the oxidant level in the context of brain ischemia-reperfusion damage. The levels of free oxygen radicals increase and the levels of antioxidants decrease depending on the level of consumption of antioxidants in tissues affected by traumatic brain damage. Increasing levels of free oxygen radicals due to the consumption of antioxidants cause lipid, protein and DNA oxidation [6-8]. An oxidant/antioxidant imbalance in favor of oxidants has also been reported in the context of brain damage caused by radiation [9]. Free oxygen radicals are regarded as the major cause of myocardial ischemia-reperfusion injury [10, 11]. Ischemia and post-ischemia reperfusion may lead to the production of free oxygen radicals in the lungs as well [12, 13]. Endothelial cells and type II pneumocytes play important roles in the formation of free radicals in the lungs [14]. The mechanisms of oxidative damage in the lungs caused by ischemia and hypoxia are different. It is argued that an oxidative damage independent of the depletion of ATP stores occurs during oxidative stress related to pulmonary ischemia. The accumulation of hypoxanthine, output product of rapid ATP catabolism, increases during hypoxia [13, 15, 16]. The damage caused by ischemia-reperfusion in many organs, such as the brain, heart, lungs, liver and intestines, has been investigated, and free radicals were found to be one of the major components of ischemia-reperfusion damage [17-22]. Tok et al. [23] reported that the levels of oxidant indicators such as MDA and MPO increased, whereas the levels of antioxidant indicators such as GSH and GST decreased. Yiğiter et al. [24] reported an increase in the MDA level and a decrease in the GSH level in kidney tissues with damage related to ureter ligation. In addition, Yiğiter et al. [24] found an increase in the amount of 8-OH-Gua, a product of DNA oxidation, in damaged kidney tissue.

Evidence of severe histopathological damage in ovary tissue in which the level of MDA was higher has been obtained by experimental studies on the prevention of ischemia-

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reperfusion damage [25]. Kurt et al. [26] histopathologically showed that damage occurs in the ovary tissue of rats when oxidant levels are high. Isaoglu et al. [27] reported a significant increase in the levels of oxidants-compared with the levels in the healthy group-in ovary tissue in which only ischemia occurred. Increases in the levels of oxidant indicators and decreases in the levels of antioxidant indicators have also been observed in ovaries damaged by drugs [28].

Derin et al. [29] showed that damage is related to increases in lipid peroxidation and decreases in PGE2 in gastric tissue affected by ischemia-reperfusion. Moreover, Derin et al. [29] reported that L-carnitine prevents gastric damage by repressing lipid peroxidation and increasing the decreased PGE2 level. It has been reported that carnitine, a powerful antioxidant, prevents damage caused by lipid peroxidation in the spinal cord, retina, kidneys, heart and brain [30-34]. Non-steroidal anti-inflammatory drugs (NSAIDs) used for treatment are also known to cause damage to gastric tissue [35]. NSAIDs not only decrease PG synthesis but also increase the levels of free oxygen radicals in the gastric mucosa. The decrease in PG synthesis leads to increases in neutrophil activation and their production capacity of free radicals and to tissue damage [36]. Free oxygen radicals may react with macromolecules in cells and cause serious cell damage, such as lipid peroxidation, oxidative modifications of proteins and DNA oxidation [37-39].

It is known that antioxidant treatment is useful to prevent tissue damage related to increases in oxidant production. In addition to preventing radical formation prior to damage, antioxidants also repair the existing oxidative damage, neutralize various reactive by-products and degrade oxidized biomolecules [40]. Consequently, it is believed that oxidants play an important role in the development of tissue damage, and antioxidants are important in the prevention and treatment of such damage.

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