
OXIDATIVE STRESS AS A PATHOPHYSIOLOGICAL MECHANISM

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The toxic effect of oxygen molecules on the human organism was first described in 1774 by J. Priestley, who compared it to the effect of a “burning candle”. Years later, superoxide dismutase was discovered (1969) and this gave rise to the theory of the oxidative cell injury, a result of the aggressive impact of oxygen-containing forms.

Contemporary literature contains an increasing amount of data on oxidative tissue and organ injury in various socially significant diseases. It is well known that the brain is vulnerable to oxidation due to its high lipid content, intensive oxygen consumption and, at the same time, relatively low antioxidant levels. Oxidative imbalance is a key mechanism of cell injury in acute (cerebral ischemia-reperfusion, epileptic seizures) as well as in chronic neurological conditions (Parkinson’s, Alzheimer, amyotrophic lateral sclerosis, etc.). Studies of patients with autoimmune diseases, such as rheumatoid arthritis, vitiligo, psoriasis, etc., as well as of patients with neoplastic diseases, demonstrate a decreased potential of the antioxidant defense of the organism. The increased production of superoxide anion radicals in the mitochondria plays a role in the development of micro- and macrovascular complications of diabetes mellitus. The oxidative imbalance is also of great importance in regard to the pathophysiology of many cardiovascular diseases.

At present, the data concerning the role of oxidative imbalance as a mechanism of burn-induced gastric mucosal injury are few in number. Therefore, the article by Hristova, et al. “Apoptosis as a Mechanism for Burn-Induced Gastric Mucosal Injury” is very interesting. The results from the study confirm the high degree of apoptosis in the gastric mucosa due to changes in the Bax/Bcl-2 ratio in favor of the proapoptotic Bax protein. As a whole, the results from the biochemical study of the oxidative stress markers, the established microscopic changes in the gastric mucosa, and the results from the immunohistochemical tests support the hypothesis of the role of oxidative stress as an important pathophysiological mechanism of apoptosis and gastric mucosal injury in the acute period after thermal trauma.