Estrés oxidativo en el paciente crítico

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Abstract

Among critically ill patients, several physio-pathological processes such as global and local hypo-perfusion, hypoxia, endothelial injury and acidosis have been associated with the production and release of large amounts of reactive oxygen species (ROS) in a non regulated fashion. Although in physiologic conditions ROS influence intracellular processes and participate in the defense against infectious organism, in critically ill conditions they are associated with potential oxidative damage over cellular structures and with persistent activation of the inflammatory response. Mechanisms associated with oxidative damage are activation of the macrophage-monocyte system and neutrophils, ischemia-reperfusion events and intracellular ROS production. Endogenous compounds, mainly enzymes, and dietary components act as antioxidant. Several studies show that in critically ill patients increase levels of ROS or reduction of antioxidant levels are related to disease severity. In animal models of critical diseases, antioxidant therapy has shown to reduce mortality. Nevertheless, there are few studies in humans that only show improvements in hemodynamic variables, reduction in inflammatory mediators levels, decreases in oxidized compounds and that suggest a lower incidence of multiple organ failure.