A Short Perspective: Relationship between Oxidative Stress and Cardiovascular Diseases

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Received: October 25, 2018; Published: January 08, 2019

Keywords: Antioxidants; Cardiovascular Diseases; Myocardial Infarcts; Oxidant Stress

Cardiovascular diseases (CVD) are one of the prime causes of mortality worldwide. 2344 research is detected when oxidative stress myocardial infarction keywords are screened in pubmed [1]. All these studies focused on oxidative stress in myocardial infarction, what can be done as a preventive and treatment options.

High levels of reactive oxygen species (ROS) have been associated with the progression of CVD such as ischemic heart disease (IHD), myocardial infarction, hypertension, atherosclerosis, aortic aneurism, aortic dissection and others. Moreover, low levels of antioxidants were associated with exacerbated cardiovascular events. The researchers gave special attention to the role of oxidant/antioxidant enzymes in vascular cells, including vascular endothelium. The major oxidant enzymes related with CVD include NADPH Oxidase, nitric oxide synthase, monoamine oxidase, and xanthine oxidoreductase. The major antioxidant enzymes that have been attributed to normalizing the levels of oxidative stress include superoxide dismutases, catalase and glutathione peroxidases (GPx), and thioredoxin [2]. Increased levels of reactive oxygen species (ROS) have been associated with the development and progression of CVD. Several groups of researchers hypothesized that halting ROS production and augmenting antioxidant levels can have a promising outcome in the treatment of CVD [3].

Oxidative stress may have potential role in cardiac hypertrophy development. Oxidative stress is observed primary stimulant for the signal transduction in cardiac cells pathological conditions, via inflammatory cytokines, and MAP kinase. The understanding of the pathophysiological mechanisms which are involved in cardiac hypertrophy and remodeling process is crucial for the development of new therapeutic agents and therapy modalities such as antioxidant remedies, supplements, exercises therapy [4].

Another research topic was the role of heme oxygenase 1 (HO1) in cardiac diseases. HO-1 provide an adaptive cellular response against chronic inflammation and oxidative injury. Indeed, the immunomodulatory and anti-inflammatory properties of HO-1 were demonstrated in several experimental studies, as well as in human cases of genetic HO-1 deficiency. HO-1 was shown to suppress the production, myocardial infiltration and inflammatory properties of monocytes and macrophages what resulted in limitation of post-MI cardiac damage. The role of HO-1, heme and its degradation products in macrophage biology and post-ischemic cardiac repair are essential important mechanisms to develop new therapeutic approaches [5].

Xanthine oxidase inhibitors (XOI), classified as purine-like (allopurinol and oxypurinol) and non-purine (febuxostat and topiroxostat) XOI, present antioxidant properties by reducing the production of reactive oxygen species derived from purine metabolism. Purine-like XOI may reduce the incidence of adverse cardiovascular outcomes [6].

Importance of antioxidant nutrients

Mediterranean diet (Improves oxidative defense and reduces oxidative stress) F-2 isoprostanes and 8 hydroxy guanosine; Milk and milk products, Omega 3 fatty acids (prevent oxidation in the cell membranes) are reduce oxidative stress [7]. Zinc is another essential nutrient for human health and has anti-oxidative stress and anti-inflammatory functions [8].

Citation: Hacı Kemal Erdemli. “A Short Perspective: Relationship between Oxidative Stress and Cardiovascular Diseases”. EC Cardiology 6.2 (2019): 100-101.
Increasing evidence also indicates that nut consumption may confer protection against CVD via lowering of oxidative stress, inflammation, and improvement in endothelial function. Nut components, such as unsaturated fatty acids, l-arginine, beneficial minerals, phenolic compounds and phytosterols, appear to be of paramount importance for their health effects [9].

Due to the increased oxidative stress, antioxidant capacity was affected. It is believed that such parameters can contribute to protection and early diagnosis of AMI and understanding the mechanism of development of the disease [10].

Useful results are obtained from the studies. Different treatment modalities should be investigated to reduce oxidative stress. Clinical use of antioxidant agents should be given importance and patients should be encouraged to consume antioxidant nutrients.

The fact that the researchers will be study of the predictive value of oxidative stress markers in heart disease will eliminate the deficiency in this area. For instance the role of ischemia modified albumin [10], thiol/disulphide [11] in acute myocardial infarction.

Bibliography