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Oxidative Stress Parameters as Cardiovascular Disease Biomarkers

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About the Study

Cardiovascular disease (CVD) imposes a massive burden on global health services. Every year, CVD contributes nearly 44 percent of mortality when compared to other noncommunicable diseases, with ischemic heart disease being the leading contributor, with a prevalence of 126.5 million people. According to health statistics, 92.1 million people in the United States have at least one form of CVD. Comorbidities such as hyperlipidaemia, hypertension, and hyperglycemia are common in CVD patients, hastening disease progression. The need for novel biomarkers to provide early diagnosis and improve cardiovascular risk stratification remains a growing concern. It is assumed that oxidative stress plays a critical role in cardiac remodelling and is responsible for the promotion and spread of CVD. Recent studies on its pathophysiology and development have focused on the breakdown of normal homeostatic systems, which results in oxidative stress. The imbalance between reactive oxygen or nitrogen species (ROS/RNS) and the body's antioxidant defence systems causes it. Overproduction of ROS may exceed the cellular antioxidant capacity, disrupting the equilibrium. These can then cause protein and lipid peroxidation, as well as DNA mutagenesis, which can have negative consequences. In addition to causing direct cellular damage, oxidative stress [1-3] causes mitochondrial dysfunction and promotes the formation of free radicals, which only adds to the disease burden.

The precise mechanism by which pro- and anti-oxidative factors influence CVD manifestation and contribute to its complications remains unknown. However, in addition to causing direct cellular damage, oxidative stress has been shown to stimulate mitochondrial dysfunction, dyslipidaemia, and genetic predispositions, as well as promote the formation of free radicals, which only exacerbates the disease burden. Many clinical and experimental studies provide a comprehensive understanding of antioxidant approaches, such as vitamins C and E to be used concurrently with guideline-recommended drug treatment, but the majority of these antioxidants show discouraging results in cardiovascular prognosis. There is emerging evidence that oxidized targets are associated with or even cause CVDs. Throughout the onset and progression of heart failure (HF), both animal models and patients showed increased oxidative stress [4,5].

Future Perspective

In the context of cardiac hypertrophy, ROS-sensitive pro-hypertrophic and remodelling signalling cascades provide additional support. Despite the recognition of oxidant biomarkers for their ease of use and low cost, available information on their prognostic relevance remains limited. Nonetheless, the nature of the ROS/RNS that are present in the body and specifically target CVD meet the essential requirement of a biomarker. The use of oxidant targets with prognostic values may be used to reflect disease risk on individuals and may soon be used in precision medicine. The goal of this review is to summarise current knowledge about oxidant-related parameters in order to estimate their effectiveness as a biomarker for the development and progression of CVD.

Low-density lipoprotein (LDL) is routinely used in clinical settings to assess cardiovascular risk, with higher levels indicating a higher risk of CVD. MPO can induce oxidative modification of LDL and release oxidized LDL (ox-LDL) in the presence of hydrogen peroxidase. While normal LDL only specified one type of receptor, ox-LDL had a higher affinity for several receptors, which greatly aided its uptake by macrophages and endothelial cells. Together with other underlying metabolic syndromes, ox-LDL accelerates its deposition in arteries and causes plaque formation. Large prospective cohort studies have found a link between elevated plasma ox-LDL and adverse cardiovascular events. Ox-LDL levels were found to be positively related to the severity of CAD. Similarly, Zhao used six different LDL-related parameters to summarize the predictive values of CAD severity. Although all of them increased with increasing CAD severity, ox-LDL had the highest predictive value and was independently associated with CAD severity.

Under physiological conditions, nitric oxide contributes to oxidative stress by regulating lipid peroxidation and producing MDA as an end product. Romuk discovered an association between MDA elevation and mortality prediction in chronic heart failure patients. In the presence of high MDA, the risk of mortality and the combined endpoint of death in the research subjects were increased twofold. Derivatives of reactive oxidative metabolites (d-ROMs) are a newly discovered ROS marker that accurately reflects a biological target's ROS production. Xuan and colleagues (2019) discovered a higher level of d-ROMs in the incidence of MI and stroke, but the findings were only significant in male subjects. d-ROMs were also linked to an increased risk of fatal MI.

Conflict of Interest

None.

Acknowledgement

None.

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