Plasma Oxidative stress parameters as early diagnostic markers of cardiovascular disorders

[1]Girish Chandran, [2]Jyoti Bala Chauhan
[1]Assistant Professor, [2]Professor and Head
[1]Department of Biotechnology, Microbiology, Biochemistry and Botany
[1]Pooja Bhagavat Mahajana Memorial Education Centre
PG Wing of SBRRMFGC, Metagalli, Mysuru-570016, India

Abstract : Cardiovascular disease (CVD) is one of the major causes of death worldwide. Generally total cholesterol and low density lipoproteins are considered the major markers for the CVD are. However, these markers do not address the role of oxidative stress in the pathophysiology of atherosclerosis, which is the main phase of CVD. Hence, we enlist the possible redox markers in the plasma which can be used as diagnostic tools for the early detection of the oxidative stress during the initiation of CVD.

IndexTerms **- Cardiovascular disease, atherosclerosis, redox markers, oxidative stress, plasma, early diagnostic markers.**

I. INTRODUCTION

Oxidative stress is associated with many chronic degenerative disorders. Here we present the discussions to support the pivotal role of oxidative stress in cardiovascular disease (CVD) and how oxidative damage may be measured in the plasma. Elevated generation of reactive oxygen species (ROS) has been implicated in the initiation and progression of both of these conditions and it may be that oxidative stress accounts for the unexplained increase in cardiovascular risks. Because of the highly reactive nature of the markers, the plasma measurements are difficult (Gracia et al., 2015). Numerous reports have focused on measuring the total antioxidant buffering capacity of plasma or alternatively specific measures of free radical-mediated damage in terms of lipid peroxides (malondialdehyde/ 4-hydroxynonenol), hydroperoxides, and activity levels of antioxidant molecules and enzymes.

Oxidative stress results from a disproportion between the formation and neutralization of oxidants. Numerous studies have demonstrated that plasma markers of oxidative stress are increased in CHD. Oxidation of lipid in cell membranes modulates diverse signal transduction pathways, leading to the pathophysiology of atherosclerosis. The prominent markers increased expression of cell adhesion molecules, induction of pro-inflammatory pathways, activation of matrix metalloproteinase, vascular smooth muscle cell proliferation and death, endothelial dysfunction, and lipid peroxidation (LDL oxidation) (Lee et al., 2012).

Oxidative stress can be either a cause or a consequence of metabolic disorders, leading to cardiovascular disease pathophysiology. The risk factors depend on numerous environmental situations such as pollution, stress, food quality, lifestyle, and many psychosocial factors. However, normal ageing also results in compromised antioxidant status leading to elevated levels of oxidant markers. The important role of oxidative stress in the etiology of atherosclerosis is marked by lipid alterations and easily oxidizable nature of lipoproteins. Formation of oxidized LDL is a pivotal event in the initiation of which promotes inflammation leading to endothelial injury and progresses the early atherosclerotic lesions (Ho et al., 2013; Moselhy and Demerdash, 2003).

Apart from the global effects associated with increased oxidative stress described above, more specific effects also occur. Low density lipoproteins are the prime target of plasma oxidative pathways and formation of ox-LDL is considered as the initial marker for atherosclerosis. However, symptomatic manifestation is required for such conclusions (Yilmaz et al., 2005). In vivo observations from the previous animal studies, have demonstrated elevated oxidative stress following hypoxia among cardiac ischemia models. Among humans, unmitigated oxidative stress is demonstrated during coronary bypass grafting, post-myocardial infarction, and in congestive cardiac failure. Therefore, numerous data are available in support of the role of oxidative stress in the etiology of chronic heart disease (CHD). Meanwhile, it is constantly debated whether increased oxidative stress is a “cause” or “effect” of CHD.

Various researchers have shown protective effects of standard antioxidant molecules as well as phytochemicals against oxidative markers in vitro and in vivo for various degenerative disorders (Ho et al., 2013). However, studies about potentiating the antioxidant power of the blood or plasma are limited. Antioxidant vitamins comprise one of the defense mechanisms available to living systems to counteract the damaging effects of excess free radical production (He and Zuo, 2015). However, despite early encouraging findings from clinical trials about antioxidant supplementation for improving antioxidant status of the plasma have yielded null results (Lee et al., 2012).

Recent studies suggest the consideration of isoprostanes as the gold standard biomarker of oxidative stress and lipid peroxidation in vivo (De Rosa et al., 2010). Thus, we aimed to revisit the OxS hypothesis to evaluate whether the degree to which an individual with CVD is potentially able to respond to OxS (e.g., plasma levels of antioxidant vitamins) is associated with cardiovascular risk and whether this varies with levels of such stress. Hence, artificial induced oxidative stress *in vitro* are monitored for protection by supplementation of plasma with antioxidants in vitro.

Conflict of Interest

Authors declare that there is no conflict of interest.

Acknowledgement

Authors thank the constant support from The Director, PBMMEC, Mysuru and Ms. Chaithra R, for kind support in our clinical studies.

References

De Rosa S, Cirillo P, Paglia A, Sasso L, Di Palma V, Chiariello M. 2010. Reactive oxygen species and antioxidants in the pathophysiology of cardiovascular disease: does the actual knowledge justify a clinical approach? Curr Vasc Pharmacol, 8: 259-75.

Gracia C.K., Llanas-Cornejo, D., & Husi, H. (2017). CVD and Oxidative Stress. Journal of clinical medicine, 6(2), 22.

He, F., & Zuo, L. (2015). Redox Roles of Reactive Oxygen Species in Cardiovascular Diseases. International journal of molecular sciences, 16(11), 27770–27780.

Ho, E., Karimi Galougahi, K., Liu, C. C., Bhindi, R., & Figtree, G. A. (2013). Biological markers of oxidative stress: Applications to cardiovascular research and practice. Redox biology, 1(1), 483–491.

Lee, R., Margaritis, M., Channon, K. M., & Antoniades, C. (2012). Evaluating oxidative stress in human cardiovascular disease: methodological aspects and considerations. Current medicinal chemistry, 19(16), 2504–2520.

Moselhy, S. S., & Demerdash, S. H. (2003). Plasma homocysteine and oxidative stress in cardiovascular disease. Disease markers, 19(1), 27–31.

Yilmaz M, Biri A, Bukan N, Karakoç A, Sancak B, Törüner F, Paşaoğlu H. 2005. Levels of lipoprotein and homocysteine in non-obese and obese patients with polycystic ovary syndrome. Gynecol Endocrinol, 20: 258-63.