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Editorial Note

Editorial Note for Oxidative stress and Diabetic Complications

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Oxidative stress is a case, where the variation occurs among the production and accumulation of oxygen reactive species (ROS) in the cells and tissues such that detoxifying capacity of a biological system is overwhelmed.

Oxidative stress is responsible for the development of diabetes complications. Due to the diabetes there is a defect of metabolism which causes the excessive production of mitochondrial superoxide in endothelial cells of both large and small vessels, and along with the myocardium. By the increase of superoxide production which leads to the activation of pathways: increased expression of the receptor for AGEs, activation of protein kinase C (PKC) isoforms, polyol pathway flux, increased formation of advanced glycation end-products (AGEs), and over activity of the hexosamine pathway. Along these pathways, intracellularly there is an increase of ROS which produces defective angiogenesis concerning to ischemia, there is an activation of pro-inflammatory pathways and gives rise to long-lasting epigenetic changes where by it manages the constant expression of pro-inflammatory genes after glycemia isundercontrol.

In type 2 diabetes, Atherosclerosis and cardiomyopathy are generated partially by pathway-selective insulin resistance, and-that raises ROS production in mitochondria from free fatty acids and ROS directly inactivates anti-atherosclerosis enzymes i.e., eNOS and Prostacyclin synthase.

The therapeutic strategy for preventing the level of oxidative stress by the inhibition or by searching of intracellular free radical creation. Through the incorporation of antioxidant formulations into conventional therapeutic treatments, either by consumption of natural antioxidants or through dietary supplementation should be supported for a whole approach for the management and anticipation of DM and complications related to the pathology.