

Coronary artery disease

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Chronic Endothelial injury Hypothesis- Endothelium protection and its repair are performed by glycosaminoglycan or GAGs when GAGs become weak, atherosclerosis is instigated. The process is promoted due to exposure of endothelial cells and free-radical damage. As the damage of endothelial lining is initiated the site promotes the permeability of plasma constituents especially the lipoproteins. Thus oxidized LDLs are capable of damaging the endothelial lining of the cells which results in the formation of plaque (Stephen, 2011).

Other contributing factors are immunological, inflammation, virus-related, or exposure to some chemical or mechanical resources prevalent in the environment.

When the endothelial cells are injured, the macrophages present in the blood move around from the blood circulation. The inner side of the artery forms a layer called a layer of the intima. Consequently, smooth muscles join intima along with connective tissue as well as lipids present inside as well as outside the cells to generate a plaque. This brings about union as well as accumulation of platelets, as well as release various growth-regulating factors to amplify the procedure ensuing obstruction of the lumen of the artery, leading to thrombus establishment (Mottillo, 2010).

Jeopardy Factors encompass

Modifiable- Obesity, Hyperlipidemias, Smoking, Alcohol consumption, Diet, Sedentary lifestyle.

Non-modifiable- Age, for female 55 and above, for males between 40- 55 Genetic predisposition or family history, Hypertension, Diabetes etc (Mottillo, 2010).

Reference

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