ATHEROSCLEROTIC HEART DISEASE AND FREE RADICALS – A HAND IN GLOVE PHENOMENON

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Pathogenesis of atherosclerotic heart disease is an outcome of oxidative modification of low density lipoprotein. It is a free radical driven lipid peroxidation process. The aldehyde products of this process, are responsible for the modification of the LDL apoprotein. Aldehyde modified apo-B protein has altered affinity, causing it to be scavenged by macrophages in an uncontrolled manner with the development of foam cells and the initiation of the atherosclerotic lesion. Endothelial dysfunction causes reduction in nitric oxide concentration, increased release of endothelin 1/angiotensin II, increased endothelial cell permeability and adhesion. Damaged endothelium is leaky, sticky and unable to relax. This leads to atherosclerosis, formation of thrombus causing occlusion of coronary arteries. A coronary plaque is clearly an inflammatory granuloma and does not result only from the accumulation of lipids.