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Commentary

Risk Factors of Atherosclerosis

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DESCRIPTION

Atherosclerosis is a condition characterized by plaque build-up within the arteries and thickening of the arterial walls. It is widely regarded as the leading cause of cardiovascular disease (henceforth referred to as atherosclerotic cardiovascular disease), which is currently the primary cause of death worldwide. Atherosclerosis is a chronic inflammatory disease initiated by the subendothelial accumulation of lipids that trigger maladaptive, non-resolving immune response. Atherosclerosis is a widespread disease of the arterial system that is generated by injury to the vasculature due to hypercholesterolemia, hypertension and inflammatory diseases. It is caused by a combination of dysfunction of the endothelium of the vessels, oxidative stress and inflammation. Endothelial dysfunction is exacerbated in the presence of high blood pressure, high blood cholesterol levels and smoking. Atherosclerosis can cause various complications, the most severe of which are heart attacks and ischemic strokes. In both cases blood flow is cut off from the heart or the brain, this lack of blood then leads to ischemia and the subsequent infarct. Both types of infarcts can lead to necrosis of tissue and may be fatal depending on the site of the infarct. The risk factors for cardiovascular disease can be either non-modifiable; such as age, gender, ethnicity, and genetics; or modifiable, such as elevated serum lipids, high blood pressure, high fasting plasma glucose, high low density lipoprotein-cholesterol, low physical activity, obesity, ambient and household air pollution, and tobacco. Interest in triglycerides as a cardiovascular risk factor is growing, since elevated postprandial lipidemia was recently shown to be an independent predictor of the risk of arteriosclerosis in the general population. This triglyceride intolerance syndrome results from difficulty processing triglyceride-rich lipoproteins, which in turn increase the risk of atherosclerosis. The main risk factors leading to the occurrence of atherosclerosis are obesity, hypertension and hypercholesterolemia, resulting in inflammation. Well-established risk factors (example, hypertension, dyslipidemia, and smoking), some other factors such as choice of lifestyle, food habits, occupation, and physical inactivity complicate body homeostasis that greatly enhances the risk of atherosclerotic cardiovascular disease and other cardio metabolic disorders that may lead to life-threatening situations. Obesity itself jeopardizes health with diminished quality of life. Higher total cholesterol levels and low density lipoprotein cholesterol help immensely foster an atherosclerotic plaque in the coronary artery. Cholesterol deposition, inflammation, extracellular matrix formation, and the gut microbiota elevate the risk of atherosclerotic cardiovascular disease development. Gathering of lipids within arterial walls is the hallmark feature in atherosclerosis that leads to fatty streak and formation of lipid-foam cells in the intima of an artery, which ultimately gets hardened and forms plaque, thereby causing artery constriction and hardening resulting in full blockage in later stages. Plaque buildup in the blood vessels of the heart is responsible for the coronary artery disease, which further leads to a heart attack. Key atherosclerotic mechanisms include the immune reaction to lipid accumulation in the arterial wall. As the disease progresses, macrophage function deteriorates, their capacity to metabolize and clear lipids from the arterial wall becomes inefficient, and they undergo apoptosis contributing to the formation of a necrotic core. Defective efferocytosis are the clearing of apoptotic cells by macrophages, which promotes inflammation resolution of further promotes a chronic non-resolving inflammatory state that results in plaque progression. Dysregulated innate immune responses within the plaque microenvironment are tied to equally complex alterations of the adaptive immune system. T cell subsets in progressing and regressing atherosclerotic plaques are highly heterogeneous and have a dynamic spectrum of pro-atherogenic and anti-atherogenic functions. Lipoprotein can be internalized and accumulated in the intima of arteries and the aortic valve leaflets. Lipoprotein enters the intima at similar rates to low density lipoprotein cholesterol, although this does not occur *via* the lipoprotein receptor for low density lipoprotein cholesterol, but is dependent on lipoprotein plasma concentrations, lipoprotein particle size, blood pressure, and arterial wall permeability.

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CONCLUSION

Atherosclerosis is a chronic inflammatory disease initiated by the sub endothelial accumulation of lipids that trigger maladaptive, non-resolving immune response. The risk factors for cardiovascular disease can be either non-modifiable; such as age, gender, ethnicity, and genetics; or modifiable, such as elevated serum lipids, high blood pressure, high fasting plasma glucose, high low density lipoprotein-cholesterol, low physical activity, obesity, ambient and household air pollution, and tobacco. Lipoprotein enters the intima at similar rates to low density lipoprotein cholesterol, although this does not occur via the lipoprotein receptor for low density lipoprotein cholesterol, but is dependent on lipoprotein plasma concentrations, lipoprotein particle size, blood pressure, and arterial wall permeability. Inflammation promotes endothelial cell activation, dysfunction and loss of endothelial integrity, failure of endothelial repair, intima lipid deposition, and plaque formation and instability.