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The Development of Atherosclerosis by Viral Infections

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Description

Atherosclerosis is a chronic disease developed by the accumulation of plaque inside arteries, which slowly thickens artery walls, narrows blood vessels, obstructs blood flow, and ultimately develops various severe cardiovascular diseases. Atherosclerosis becomes one of the leading causes of all deaths in many developed countries. It is well known that atherosclerosis can be developed by the undesirable lifestyle such as excessive cholesterol diets, high sugar diets, smoking, and stress. However, it can be also developed by the individual genetic factors or infections by microorganisms.

Not only are bacteria such as *Chlamydia pneumoniae* and *Helicobacter pylori* known for their involvement in the development of atherosclerosis, but also many viruses such as influenz a viruses, severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), human immunodeficiency virus (HIV), herpes viruses are highly linked to the pathology of atherosclerosis. It has been reported that atherosclerosis can be developed through systematic inflammatory syndromes after infection by influenza viruses; many patients with coronavirus disease 2019 (COVID-19) have claimed symptoms of cardiovascular diseases; HIV-positive patients show a higher prevalence of atherosclerosis than the HIV-negative population [1].

Because these viruses are significantly prevalent worldwide, and are not easily controlled, the development of atherosclerosis by these viruses is very notable for study. In this study, the development of atherosclerosis by influenza viruses, SARS-CoV-2, HIV, and herpes viruses are described.

For the development of atherosclerosis, viral infections can induce overexpression of different growth factors and various inflammatory molecules such as cytokines, adhesion molecules, and chemoattractant molecules, provoke oxidation and uptake of low-density lipoprotein (LDL), and increase resistance against apoptosis [1].

- For examples, influenza viruses activate and upregulate various inflammatory signals such as tumor necrosis factor (TNF) proteins, interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and neutrophil extracellular traps (NET), apoptosis of epithelial cells, the change of endothelial permeability and vascular leak, and the platelet activation [2-4].
- It is understood that COVID-19 can develop atherosclerosis due to the extremely high levels of proinflammatory cytokine produced after SARS-CoV-2,infection [1]. SARS-CoV-2,infection stimulates secretions of IL-1β, interleukin-4 (IL-4), IL-6, and interleukin-10 (IL-10), interferon-γ (IFN-γ), IFN-γ-induced protein 10 (IP-10), monocytic chemoattractant protein-1 (MCP-1), some of which can trigger a serious cytokine storm in a host and develop atherosclerosis [5].
- HIV infection upregulates and elevates various inflammatory signal components such as IL-1β, IL-6, interleukin-12 (IL-12), Interleukin-18 (IL-18), IFN-γ, and MCP-1, TNF-α, TNF-β, and the nuclear factor kappa-light-chain-enhancer of activated B cells (NFκB), some of which can mediate recruitment of monocytes and differentiation of macrophages to induce lipid engulfment and formation of atherosclerotic foam cells and plaque [6].
- Herpes simplex virus (HSV)-1 and -2 upregulate lectin-like oxLDL receptor-1 (LOX-1), a major receptor protein of oxLDL in a host, and stimulate the uptake of oxLDL in endothelial cells [7]. This mechanism can lead the acquisition of saturated cholesteryl esters and triacylglycerol, and provoke lipid accumulation and its metabolism to increase incidence of atherosclerosis.

Conclusion

Virus infections and the development of atherosclerosis are highly linked. The data from autopsy, biopsy, metadata analysis, animal study, and molecular biology research data confirm viral infections can promote and aggravate atherosclerosis [1]. The viral infections can activate the innate immune responses, and elevate various pro-inflammatory cytokines, mediators, growth factors, and receptors. Increased secretion of various chemokines and interleukins, overexpression of immunogenic receptors, and hyperactivation of immune cells provoke atherosclerosis; for example, macrophages are activated by various cytokines such as IL-1β, IL-6, INF-γ, and TNF-α after virus infections, and the activated macrophages induce phagocytosis of oxLDL, which will lead the formation of the foam cells and plaque. Also, increased oxLDL, MCP-1 production, calcium levels, and ER stress in aortic endothelial cells can cause apoptosis of the foam cells and plaques in the arteries, which will ultimately lead the development of atherosclerosis [1].

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Conflict of Interest

The author declares that there is no conflict of interest associated with this manuscript

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