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CMV and Atherosclerosis

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Although our understanding of the cellular and molecular mechanisms of atherogenesis has greatly increased over the last decade, surprisingly modern concepts of its basic pathophysiology have existed for over a century. In the mid-19th century, Virchow [1] first postulated that atherosclerosis was a chronic arterial disease secondary to vascular injury. Nowadays, it is clear that the most significant basic feature of the disease is the presence of a chronic but active inflammation of the arterial wall which, over many years, results in intimal fibroproliferation and vascular occlusion. The current prevailing hypothesis of atherogenesis is that it is an inflammatory response to acute or chronic endothelial injury [2]. Possible causes for this endothelial injury include free radicals, modified LDL, hypertension, diabetes mellitus and various other risk factors. Recently, evidence suggests that infections should be included in this already long list of potential risk factors for atherosclerosis.

Historical Perspective

As early as the late 1970s, reports demonstrating a role for herpes viruses in the development of atherosclerosis were published. Based on earlier experiments by Paterson and Cottral [3], Fabricant and Fabricant [4] hypothesized that an avian herpes virus, Marek's disease virus (MDV), might contribute to the atherosclerotic process in chickens. When chickens were infected with this virus, fatty proliferative arterial lesions were observed in coronary, gastric, mesenteric and celiac arteries and aortas of infected birds while these lesions were absent in uninfected birds irrespective of adherence to a high cholesterol diet or not. Furthermore, MDV-specific antigens as well as the MDV genome