OPEN ACCESS Freely available online

ISSN: 2329-6925
Journal of Vascular Medicine & Surgery

Commentary

Atherogenesis in Rabbits by Using Dietary Design

Jayesh Patel*

Department of Vascular Surgery, Shree Krishna Hospital, Gujarat, India

DESCRIPTION

Atherosclerosis is the leading cause of cardiovascular disease and accounts for the majority of deaths worldwide. The lack of reproducible and controlled induction of atherosclerosis in preclinical animal models limits the study of atherosclerosis. The development of the first automatic vessel wall injury system in a rabbit model of atherosclerosis that results in more uniform injury and more pronounced atherosclerotic plaque formation at low balloon pressure, and risk factors using this system. By studying the interaction of elevated plasma homo cysteine is an independent risk factor for atherosclerosis and is closely associated with cardiovascular mortality. In the absence of hypercholesterolemia, a deficiency of vitamins and choline required for homo cysteine metabolism is atherosclerosis of the aorta, i.e. impaired biodynamic properties, changes in collagen tissue, accumulation of macrophages and lipids and exacerbation of atherosclerosis, including significantly altered amounts of water diffusion and impaired vascular reactivity in its presence.

Cardiovascular disease, the world's leading cause of death, is primarily due to the development of atherosclerosis. However, today only 50 % of the incidence of atherosclerosis can be explained by known risk factors such as hypercholesterolemia. Rabbits and mice are the most commonly used animal models for studying atherosclerosis. Rabbits fed a high-fat diet against genetically engineered mice, such as apolipoprotein E and Low-Density Lipoprotein Receptor (Ldlr) mice, have foam cells that resemble human atheroma. Spontaneously develops abundant plaques (fatty streak).Damage to the arterial wall reduces the time required to form advanced lesions in response to hyperlipidemia. In most cases, vascular injury in animal models of atherosclerosis is achieved by repeatedly withdrawing a balloon-equipped catheter that is inflated in the artery. The developement of an automatic balloon-mediated injury system and compared this technique to the classic manual injury of the aorta in a rabbit atherosclerosis model. Automatic balloon-mediated injury system controls both balloon pressure and catheter withdrawal speed, allowing you to standardize and optimize vessel wall damage.

To standardize the rabbit model of atherosclerosis, automatic injury technology (JURY) has been developed that enhances the reproducibility and uniformity of lesion formation in atherosclerosis. The JURY device compensates for anatomical differences in vessel diameter and keeps the friction effect more constant with an automatic contraction system. JURY is connected to a computer that allows you to adjust injury parameters and record and display balloon pressure data on a real-time monitor. This technique already induces mild damage at 1.2 bars, single contraction limits damage to the endothelial layer, and an aortic aneurysm that can be caused by high pressure and /or repeated exposure steps. It reduces adverse effects such as. Importantly, automation of vessel wall injury results in complete exposure of the endothelium and more homogeneous and reproducible atherosclerotic plaque formation compared to manual injury. In fact, balloon pressure is an important parameter that affects not only the severity of vascular injury, but also subsequent intimal hyperplasia and vasomotor responses. The damage generated by our method corresponds to type II damage and can be induced over the intended range, providing a more comprehensive analysis and extended applicability of the model. Make it possible. Comparing automated JURY-induced injuries with traditional blind and manually pressure-adjusted exposures, the automated injuries are superior to other techniques.

CONCLUSION

Significant thickening of the aortic wall, massive impairment of vascular responsiveness, changes in FA, and increased accumulation of lipids and macrophages in the neointima are stronger for HCD for atherosclerotic transformation of the aorta in combination with VCDD. VCDD also has an independent effect on atherosclerotic changes in the aortic wall by inducing lipid and macrophage accumulation, endoplasmic reticulum dilation, and loss of vascular elasticity. Therefore, the experimental rabbit model of atherosclerosis described herein

Correspondence to: Jayesh Patel, Department of Vascular Surgery, Shree Krishna Hospital, Gujarat, India, Email: JayeshPatel@gmail.com

Received: 04-Feb -2022, Manuscript No. JVMS-22-15857; **Editor assigned:** 06-Feb-2022, Pre QC No. JVMS-22-15857 (PQ); **Reviewed:** 20-Feb-2022, QC No. JVMS-22-15857; **Revised:** 24-Feb-2022, Manuscript No. JVMS-22-15857 (R); **Published:** 03-Mar-2022, DOI: 10.35248/2329-6325.22.10.445.

Citation: Patel J (2022) Atherogenesis in Rabbits by Using Dietary Design. J Vasc Surg. 10:445.

can not only exacerbate high cholesterol-induced atherosclerosis, but also induce independent atherosclerosis changes in the aortic wall. JURY technology combined with this model provides new tools for advancing research on atherosclerosis and broadening the horizons of new therapeutic strategies.