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Editorial

Spontaneous Aortic Rupture Plaque: Refocus on the Atherosclerosis as the Systemic Disease

Atsushi Hirayama*

Cardiovascular Division, Osaka Police Hospital, Osaka, Japan

***Corresponding Author**: Atsushi Hirayama, Cardiovascular Division, Osaka Police Hospital, Osaka, Japan.

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Atherosclerotic plaques initially developed by endothelial damage and accumulation of oxidized-low density lipoprotein cholesterol (LDL-C). Circulating macrophages invaded into vessel wall to scavenge the accumulated LDL-C. LDL-C was degraded and cholesterol crystal was generated in the macrophages. Further accumulation of LDL-C turned macrophages to formed cell and cholesterol crystal was released into the plaque after the death of formed cell. Cholesterol crystal was scavenged by the macrophage again and stimulated the invertasome and enhanced the inflammation inside the plaques, then the plaques became vulnerable and finally ruptured [1]. Ruptured plaques in the coronary artery cause the thrombus and occlude the coronary sometime, then resulted in acute coronary syndrome; however, plaque rupture without coronary occlusion were frequently observed in the patients with coronary artery disease [2]. These spontaneous ruptures have not been thought to affect the human body. Recently, these spontaneous atherosclerotic plaque ruptures have been shown to be observed in the aorta by the non-obstructive general angioscopy (NOGA) by Komatsu., *et al* [3]. He also aspirated the blood just above the ruptured plaques observed by NOGA and found the various kinds of plaque materials including fibrin, thin-cap and cholesterol crystals with various sizes. Furthermore, he identified the cholesterol crystal itself raged from 20 - 100 micrometer. We do not know how these materials goes on. Plaque materials with large size may cause the arteriole obstruction and organ damage with symptom, such as aortogenic stroke or peripheral artery disease as described by Narula., *et al* [4]. She pathologically demonstrated that the most of critical limb ischemia or peripheral artery disease was caused by the thromboembolism. On the other hand, the effect of cholesterol crystal is not known. Those may affect the microcirculation without symptom as well as obvious organ damage; however, multiple unlimited repeated plaque rupture in the aorta during the life time produce the infinite cholesterol crystals in the blood and may induce the insufficient microcirculation resulting in the cellular as well as the organ damage. Recent pathological findings reveled cholesterol crystal itself was observed in the myocardium and caused the micro infarction in patients with acute coronary syndrome [5]. These phenomena should occur in the entire organs. Cholesterol crystal embolization might be the cause of dementia, renal insufficiency, sarcopenia and so on.

This hypothesis is still hypothesis and there are no evidences yet. Two results of large clinical trials support this hypothesis. One is the Fourie trial, which showed that the further LDL-C reduction by the addition to the routine statin therapy, showed the significant reduction of stroke event [6]. LDL-C reduction of statin has been shown to stabilize the vulnerable plaque [7]. Further LDL-C reduction may reduce the number of vulnerable plaques in the aorta. Another is COMPASS, which rivaroxaban plus aspirin reduce the significant reduction of the events of stroke as well as peripheral artery disease compared to aspirin only [8]. The mechanism of rivaroxaban has not been clarified; however, rivaroxaban may degrade the plaque materials from the ruptured plaque in to the small size with asymptomatic state, or stabilize the vulnerable plaques with anti-inflammatory effects. From this point of view, we have to refocus the atherosclerotic disease as the systemic disease.

Spontaneous aortic rupture plaque (SARP) observed by NOGA opens the new window to understand various kinds of unknown cause of disease with might be related to aging. In the next step, we should focus on the aortic plaque and find out to stabilize the plaque in the aorta and prevent its rupture.

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