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Preprint · May 2022

DOI: 10.13140/RG.2.2.18381.31209

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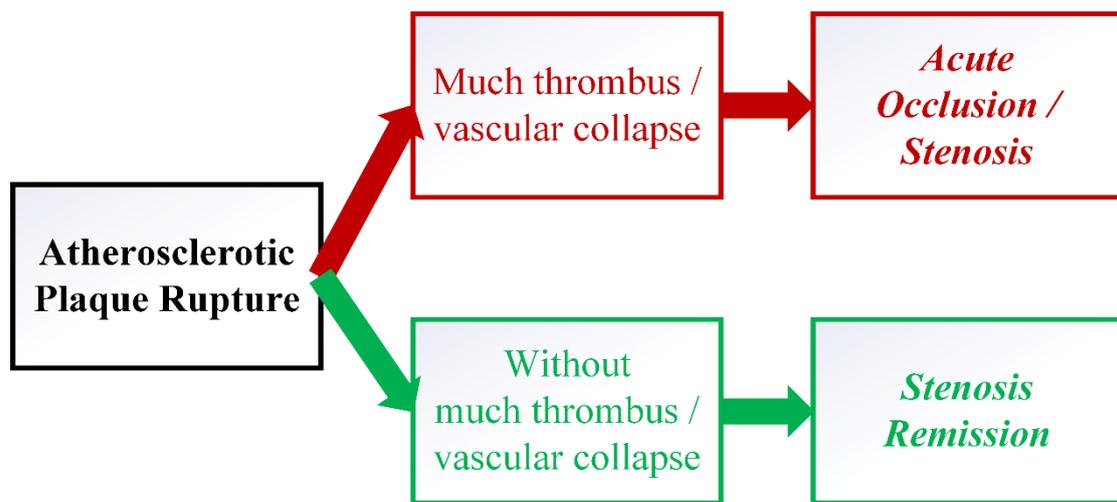
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## Is Atherosclerotic Plaque Rupture Necessarily Detrimental? -Not Always

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**Figure 1. Different outcomes of atherosclerotic plaque rupture**

### Main text

Atherosclerosis is the result of vascular repair or remodeling<sup>1-28</sup>. Various risk factors may increase the risk of dysfunction / injury of smooth muscle cells (SMC)<sup>12,23-27</sup>. The dysfunction / injury of SMC could lead to the tunica media dysfunction. Tunica media dysfunction could lead to pressure redistribution on vascular wall, resulting in fibrous connective tissue remodeling and vascular changes such as atherosclerosis, stiffening or aneurysm<sup>2,16</sup>. With the development of fibrous connective tissue remodeling, fibrous connective tissue would undergo degeneration, necrosis, rupture or

calcification, etc<sup>1,2</sup>. The rupture of atherosclerotic plaque may lead to intravascular thrombosis or vascular collapse, which may endanger the life and health of patients. **Is atherosclerotic plaque rupture necessarily harmful? It may not always. This paper will elaborate on this view (Figure 1).**

Clinically, acute coronary syndrome (ACS) is mainly due to the rupture of atherosclerotic plaque and vascular thrombosis / collapse, which may lead to complete occlusion or severe stenosis of blood vessels. It is certainly harmful (**Figure 1**). However, some people may have atherosclerotic plaque rupture, but it does not lead to vascular occlusion or severe stenosis. These people should not have any clinical symptoms and would not be hospitalized for coronary angiography or intracoronary ultrasound. Plaque rupture without significant vascular thrombosis / collapse may be beneficial. Because plaque rupture could effectively release the denatured / necrotic fibrous connective tissue in the plaque, which would enlarge the severely narrow lumen without obvious vascular thrombosis / collapse, while the denatured or necrotic fibrous connective tissue would be phagocytosed by macrophages without causing obvious damage to the body. This is a valuable compensatory mechanism for severe vascular stenosis (**Figure 1**). Therefore, we hope that more people with atherosclerotic plaque rupture would not form obvious vascular thrombosis / collapse, meanwhile it would relieve severe vascular stenosis (**Figure 1**). In order to achieve this effect as much as possible, it may be beneficial to strengthen anticoagulant therapy in patients with unstable atherosclerotic plaque<sup>28</sup>.

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