The Current Understanding of Atherosclerosis Pathogenesis: From Bench to Bedside

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ABSTRACT

Understanding of atherosclerosis pathogenesis is constantly developing. The causes of atherosclerotic plaque formation process include lipid retention, oxidation, and modification, which provoke chronic inflammation at susceptible sites in the walls of all major arteries. Improvement of clinical imaging modalities such as IVUS (intravascular ultrasound) or OCT (optical coherent tomography) also attributed the deeper understanding of the pathogenesis and natural history of coronary atherosclerosis. Recent advantages of molecular imaging techniques shed light into the molecular basis of atherosclerosis formation and development. Our research using NIRF(near-infared fluorescence)-OCT catheter system enabled the visualization of both anatomical information from clinical grade OCT and plaque characteristics such as inflammation simultaneously. In this presentation, we will mainly present our recent data using such molecular imaging technology to visualize pathogenesis of atherosclerosis.

Keywords: atherosclerosis; pathogenesis; current understanding

Atherosclerosis and Its Journey Within Cardiovascular Disease Continuum

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ABSTRACT

Atherosclerosis is the underlying cause of nearly all cause mortality and morbidity of coronary, peripheral artery disease and many cases of stroke. Atherosclerosis is a systemic inflammatory process characterized by the accumulation of macrophages and lipids within the intima of arteries. These plaques significantly compromise the residual lumen, leading to ischaemic event distal to the arterial stenosis. However, these initial fatty streak lesions may also evolve into fibrous plaques vulnerable to rupture or erosion. Plaque disruption initiates both platelets adhesion and aggregation on the exposed vascular surface and the activation of the cascade leading to the so called atherothrombotic process. In fact, many local and systemic "thrombogenic risk factor" at the time of plaque disruption influence the duration of thrombus deposition and hence the different pathological and clinical syndromes.

Keywords: plaqeu; endothel dysfunction; thrombogenic risk factor

The State-of-the Art Diagnostic Modalities for Atherosclerosis: Recognizing Stable and Unstable Plaque

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ABSTRACT

Coronary artery disease (CAD) is the leading cause of death in many developed and developing countries. Although the pathogenesis of atherosclerotic plaque in CAD is not fully understood, CAD typically cycles in and out of defined clinical phases from asymptomatic to stable angina, unstable angina to acute myocardial infarction.

Gradual but stable progression of atherosclerotic plaquein CAD patient may affect quality of life by causing exertion-limiting angina, butit seldom kills. The development of coronary collaterals as well as myocytes switching to hibernating state protects the myocardium. It is the sudden change of clinical phase from stable angina to acute coronary syndrome (ACS) that leads to major adverse cardiac events (MACE). We now understand that rupture ofa previously stable plaque and subsequent thrombus formation is the mechanism for this sudden transformation. Unstable or vulnerable plaques are plaques that are prone to rupture, irrespective of the degree of stenosis. If we could identify these vulnerable plaques and predict the possibility of their rupture, we should be able to prevent ACS and reduce MACE. With recent advances in imaging technologies (IVUS, OCT, NIRS, CT etc.), certain characteristics of vulnerable plaque have been identified, which include thin-cap fibro-atheroma, high lipid core content and plaque burden. However, clinical studies (PROSPECT, VIVA) have not demonstratedthat identifying these “vulnerable” plaque wouldimprove risk prediction as compared to traditional approaches as the positive predictive value was low and there were complications associated with invasive imaging to identify these plaques. The results of these studies have raised scepticism on the effectiveness of identification of vulnerable plaque in clinical practice. Other factors, e.g. hemodynamic of coronary blood flow, coagulation state of the patients and other systemic factors may play important roles rather than just plaque characteristic and composition alone. Unless there are newer imaging technologies that could identify plaque characteristics withhigher positive predictive value for rupture, the concept of vulnerable plaque detection may not be recommended in clinical practice. It may be better to consider vulnerable patients (diabetes, CKD, hyperlipidaemia), rather than plaque, who are at increased risks of MACE.

Interventional Therapy for Stable Coronary Artery Disease: What to Offer and How to Select

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ABSTRACT

Ischemic Heart Disease (IHD) including Acute Coronary Syndrome (ACS) and Stable Coronary Artery Disease (SCAD) is the leading cause of death in the United States. SCAD is generally used to define patients with coronary atherosclerotic disease that have clinical appearance as asymptomatic or non accelerating symptomp. Lifestyle modification and optimal medical therapy are advised for all patients with SCAD to reduce morbidity and mortality. The other option for patients with IHD is Percutaneus Coronary Intervention (PCI) as a revascularization. It was established that PCI reduced mortality in ACS patients. In contrast, the role of PCI in management of patients with SCAD has been a matter of continued controversy. Many studies have failed to show benefit of PCI in reducing hearte cardiac event including death and Myocardial Infarction (MI) in SCAD. Some studies showed a subgroup of patients with SCAD have benefit of PCI. Selection of patients with SCAD undergone PCI had an important role.
Coronary angiography coronary angiography be considered early in patients who are identified to have high risk non-invasive test features. Patients develop medically refractory symptoms or inadequate CV quality of life on medical therapy should undergo elective coronary angiography in anticipation of possible revascularization procedures.

Keywords: stable coronary artery disease; coronary angiography; revascularization