OMICS Group Conferences
Accelerating Scientific Discovery

3rd International Conference on
Clinical & Experimental Cardiology

April 15-17, 2013 Hilton Chicago/Northbrook, USA

Parallel cholesterol crystals: A sign of impending plaque rupture

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Background: Currently, there are no reliable methods that can reliably predict when or if an atheroma will rupture. Recent reports suggest cholesterol crystals (CC's), present within the necrotic core, are sharp and can penetrate and disrupt the fibrous cap, contributing to plaque rupture (PR).

Aim: is to show CC, normally distributed at random within the necrotic core, often develop a parallel configuration at the site of plaque rupture which may be a sign of impending rupture.

Materials and Results: The coronary arteries of 83 patients who died of acute coronary disease (ACD) were injected with a colored barium gelatin mass. The arteries were dissected, decalcified, cut at 2-3 mm intervals, and all segments mounted for microscopic study. All segments were reviewed to identify PR'sand to determine the frequency of parallel cholesterol crystals (P-CCs) at the site of these PRs. There were 215 separate PRs in 83 patients with 64 (77%) patients showing more than one PR. P-CCs were present in 126 (59%) of all PRs regardless of plaque size or the severity of luminal stenosis and were present with or without luminal thrombosis. The parallel configuration appears to reflect increased intra-plaque pressure.

Conclusion: P-CCs are commonly present at the site of PR and may contribute to PR by penetrating and disrupting the fibrous cap. The ability to recognize this parallel configuration may help to identify plaques with impending PR.

Biography

Frink is the Principal Investigator of the Heart Research Foundation of Sacramento. He received his training at the University of Iowa, the Mayo Clinic and the University of Alabama in Birmingham. He practiced invasive cardiology in Sacramento, California for 35 years and established a laboratory to study the post-mortem heart. He has published approximately 25 research papers and a book, Inflammatory Atherosclerosis: Characteristics of the Injurious Agent, detailing the pathologic findings in patients who died of acute coronary disease. The primary focus of his work has been the pathogenesis of atherosclerosis and the mechanism responsible for sudden cardiac death.

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