Plaque Burden vs. The Vulnerable Plaque and How to assess CAD Risk

Arthur Agatston
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Disclosures

• None
The Vulnerable Plaque

Motoyama JACC 2009;54:49-59

Summary

- CAD is diffuse before events occur
- Rupture of “vulnerable plaques” are common and almost always subclinical
- Subclinical CAD quantification reflects the dose and duration of integrated risk factors
- Rheology explains the “feed forward” mechanism of atherosclerosis progression
- Increase in calcium score does not necessarily indicate disease progression
- Serial calcium scans showing new lesion progression is the best way to monitor prevention measures

The Origin of the Conventional View of Coronary Stenosis-Canine Model

K. Lance Gould Am J Cardiol 1974;33:87-94
The Conventional View Proved Wrong: The Courage Trial-Stents vs. Medical Therapy

Why?

Coronary Atherosclerosis is a Diffuse, Not a Focal Disease

Roberts WC. Am J Cardiol 1988:62; 142-143
Coronaries in Angina

Roberts WC Circulation 1976: 54(3) 388.

The Myth of Coronary Percent Stenosis

Glagov S, NEJM 1987; 316:1371-5
Coronary Remodeling

Glagov S, NEJM 1987;316:1371-5

Abnormal epicardial coronary resistance in patients with diffuse atherosclerosis but "Normal" coronary angiography

Bernard De Bruyne Circulation. 2001;104:2401-2406

Fractional Flow Reserve vs. Angiographic Stenosis (FAME)

J Am Coll Cardiol 2010;55:2816–21
Coronary Flow Reserve (CFR) vs. Anatomy With Diffuse Coronary Artery Disease and Remodeling

Risk of CV Death: Diffuse Non-obstructive Disease vs. Limited Obstructive Disease

CAD Risk vs. Extent of Atherosclerosis

Roberts WC. Am J Cardiol 1988;62 (1): 144-147

Traditional 10-Year Risk Model


“Lifetime” Risk Model
The Concept of Dose/Duration of Risk Factor Exposure

Hyperlipidemia in Early Adulthood Increases Long-Term Risk of Coronary Heart Disease (Framingham)
Why Do Coronary Events Occur Almost Exclusively After Extensive Atherosclerosis is Present?

The Role of Rheology And the Feed Forward Mechanism of CAD

The role of shear stress in the pathogenesis of atherosclerosis

Cunningham, C. Laboratory Investigation (2005) 85, 9–23
Effects of Disturbed Flow on Vascular Endothelium

Plaque Rupture: But no Occlusion

Physiol Rev 91: 327-387, 2011;

Rheology and Thrombosis

Shear gradient-dependent platelet aggregation mechanism drives thrombus formation

Nesbitt WS, NATURE MEDICINE VOLUME 2009;15 (6) : 665
Shear gradient–dependent platelet aggregation mechanism drives thrombus formation

The Feed Forward Concept of Coronary Progression
A vicious cycle of vascular injury

Hypertension. 2013;61:615-621
69 yo hypercholesterolemia
The Future of Coronary Imaging

There is a lot more clinical information in CT and CTA Images than just the calcium score

Steady Progression

50 yo Presenting to ER Without Prior RX
JA 71 M +FH PCAD, CAC=450 age 54


SG 69 yo WM -6 yr F/U

Conclusion-1

• Clinical CAD is diffuse
• Rupture of “vulnerable plaques” are common and almost always subclinical
• Rheology explains the “feed forward” mechanism which in turn explains why subclinical CAD is both atherogenic and thrombogenic and begins proximally and proceeds distally
• Subclinical CAD reflects the dose and duration of integrated risk factors

Conclusion-2

• The “feed forward” physiology explains why “plaque burden” trumps the “vulnerable plaque”
• Increase in calcium score does not necessarily indicate disease progression
• The appearance of new “young plaques” (low density) indicates disease activity

Thank You
Plaque Calcium Density Predicts Events at a given Volume of CAC

In Diffuse Disease, Young Lesions = Higher Risk