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SPECIAL FOCUS ISSUE: CARDIOVASCULAR HEALTH PROMOTION

EDITORIAL COMMENT

Coronary CT Angiography

Identification of Patients and Plaques "At Risk"*

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Despite advances in medical and revascularization therapies, complications of coronary artery disease (CAD) are still the number 1 cause of mortality in the Western World. Catastrophic acute ST-segment elevation myocardial infarction and sudden coronary death constitute persistent societal scourges that beg for novel approaches for detection and prevention. The concept of the "vulnerable plaque" (VP) is based on the hypothesis that a prospectively identifiable precursor lesion evolves into the culprit responsible for acute coronary syndromes (ACS). The implicit hope is that pacification of such lesions by novel medical therapies and/or pre-emptive focal stenting can reduce adverse events.

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The Chang et al. (1) study of computed tomographic angiography (CTA) in this issue of the *Journal* should be congratulated for important observations that support and further the promise of noninvasive identification of patients and plaques "at risk." This nested case-control study of 234 pairs of ACS and control patients (derived from a cohort of 25,251 cases undergoing CTA) assessed the association between baseline plaque characteristics and subsequent ACS events over 3 years. Lesion parameters analyzed focused on "high-risk plaque" (HRP) (≥2 features, including positive remodeling, low-attenuation plaque [LAP], and spotty calcification), together with plaque burden and composition. Key findings on a *per-patient* basis showed: 1) at baseline, one-half of ACS patients had HRPs (more frequent than control subjects), and the presence of such lesions was associated with subsequent ACS; and 2) lesion characteristics, including HRP, plaque burden, and burden composition, identified high-risk patients above and beyond stenosis severity and aggregate plaque burden. Analysis on a *per-lesion* basis showed that 75% of ACS culprit lesion precursors at baseline exhibited <50% stenosis. However, only 31% of culprit precursors exhibited HRP features. These findings have potential implications for characterizing at-risk patients who may benefit from systemic therapeutics designed to stabilize the atherosclerotic coronary vasculature, but they provide lesser evidence to support prospective identification of HRPs that might benefit from pre-emptive focal stenting.

PROVING THE VP HYPOTHESIS: SATISFYING KOCH'S POSTULATES

Robert Koch (1843 to 1910) propounded a series of 4 generalized principles linking specific microorganisms to specific diseases, a paradigm for proof of disease causation. Conversely, to validate the VP hypothesis, the following must be established: 1) definable VP characteristics (morphology and composition) delineated by an imaging "snapshot in time" are associated with a prevalence of focally linked ACS events sufficient to warrant interventions beyond aspirin and statins; and 2) novel systemic medical therapies and/or preemptive stenting can reduce adverse outcomes.

PORTRAIT OF THE VP

The proximate cause of most ACS cases is rupture of a lipid-rich necrotic core plaque with inflamed disrupted fibrous cap and superimposed thrombus. Direct coronary imaging and histopathological studies characterize the putative precursor VP as a

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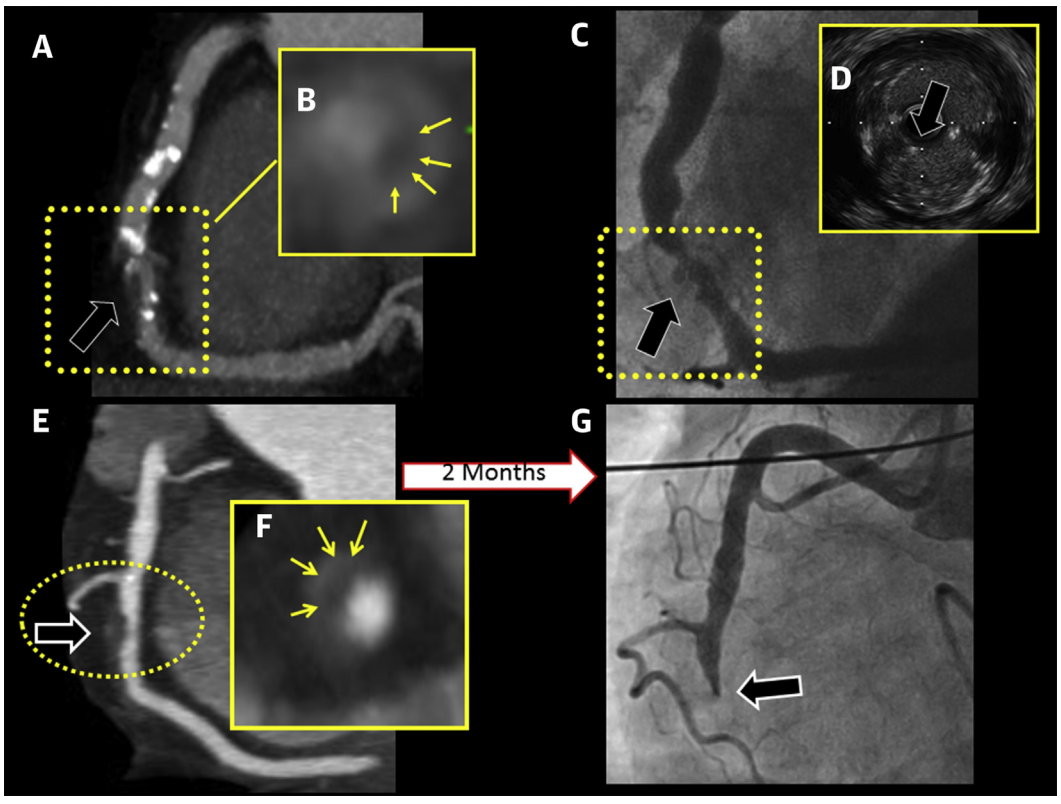
FIGURE 1 Plaque Disruption by CTA



(A) A patient with acute chest pain undergoing emergency computed tomography angiography (CTA) demonstrating gross plaque ulceration **(dotted yellow rectangle, black arrow)**. **(B)** Lesion cross section illustrates "ring-like enhancement" **(yellow arrows). (C)** Invasive imaging the following day shows concordant plaque ulceration **(yellow dotted rectangle, black arrow)**, confirmed by intravascular ultrasound **(D, black arrow)** (images modified from Madder et al. [6]). **(E)** A stable patient with atypical chest pain, with CTA showing mild stenosis but bulky positively remodeled low-attenuation plaque **(yellow dotted circle)** with discrete intraplaque dye penetration **(black arrow)**, suggesting plaque hemorrhage. **(F)** Lesion cross section illustrates "ring-like enhancement" **(yellow arrows)**; **(G)** 2 months later, the patient experienced inferior ST-segment elevation myocardial infarction due to total occlusion at the same site **(black arrow)** (images modified from Bilolikar et al. [7]).

thin-capped fibroatheroma, with larger lipid-necrotic core plaque volume and reduced lumen imparting a greater risk of future instability (2). However, a paucity of natural history data has relegated such VPs to a status of "indicted but not yet convicted" (James Muller, personal communication, June 2013).

CTA: IDENTIFICATION OF AT-RISK PATIENTS

The value of CTA can be considered from 3 basic perspectives: detection of patients with CAD, identification of at-risk patients, and delineation of VP. Extensive studies validate computed tomographic coronary calcium scoring alone as a marker of atherosclerosis and establish the relationship of elevated scores with increased risk of adverse outcomes. Prior studies have shown that CTA provides incremental benefit in risk stratification and prediction of events in those with diabetes. Focal CTA plaque characterization adds further prognostic value. The present findings are consonant with prior CTA observations (3) demonstrating that patients harboring "2-feature positive" plaque (LAP and positive remodeling) are at greater risk of adverse events over time (3). CTA delineates additional HRP morphological features, including "napkin ring" enhancement, a lesion with a discrete pocket of very LAP (presumably reflecting lipid-necrotic core), which may add further pathophysiological insight and prognostic value (4).

CAN CTA IDENTIFY VPs?

The interventionist's quest for VP is based on the concept that a precursor lesion can be prospectively

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