

EDITORIAL COMMENT

The Stress of Plaque Prognostication*

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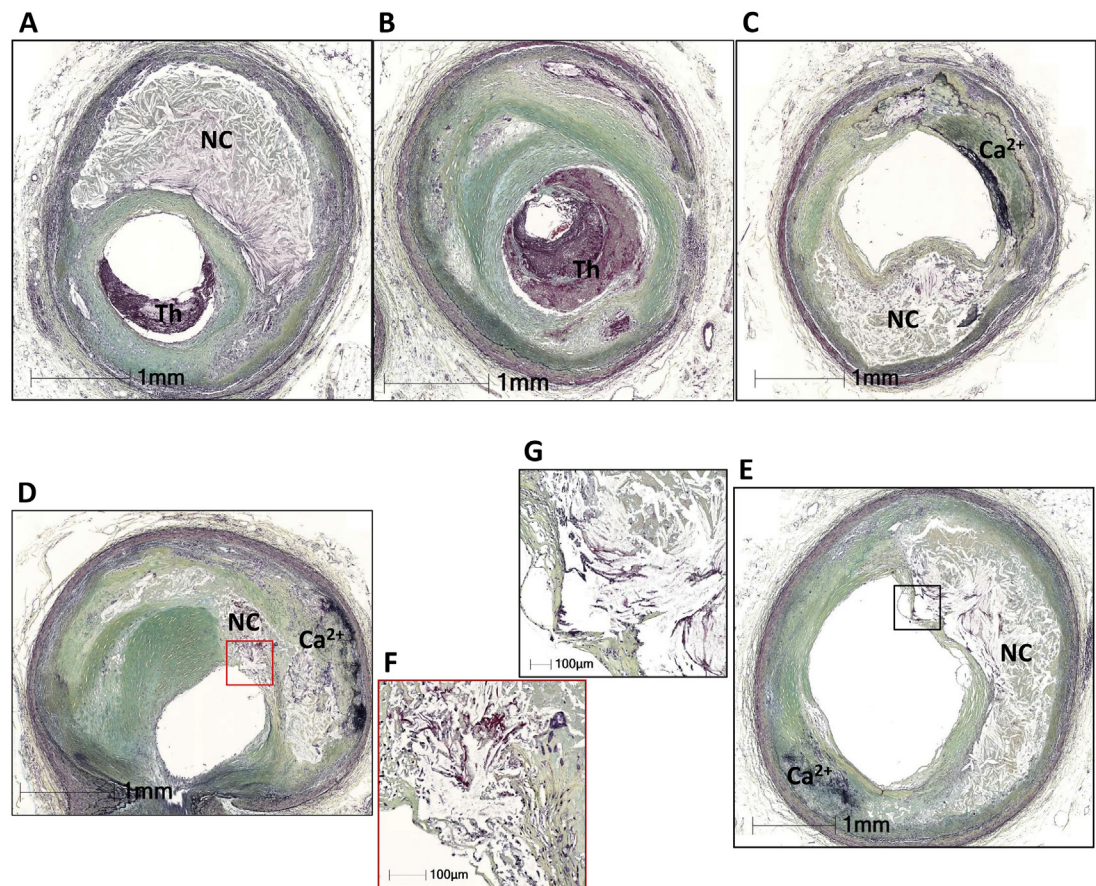
Plaque rupture is a predominant cause of acute coronary syndromes and sudden coronary death (1). However, not all plaques progress to cause clinical events (2). Many remain dormant for the life of the individual and cause neither symptoms nor clinical events, whereas others progressively narrow but do not rupture, thus resulting in stable angina. However, only a few plaques become progressively unstable in morphology and eventually are the cause of serious clinical events (Figures 1A to 1G). Because we currently lack the understanding and technology to identify correctly which so-called vulnerable plaques (thin-cap fibroatheromas) in the short term will go on to cause symptoms, and especially acute myocardial infarctions, our focus therefore has been on the development of techniques that restore blood flow in arteries possessing hemodynamically significant lesions that cause ischemia or infarction. This reactive strategy has meant that although death rates for heart disease have continued to fall in recent decades, the number of people with cardiovascular disease is rising because many more people are living with the crippling aftereffects of

heart attacks (3). In addition to finding better therapies for those patients with heart disease, preventing future events remains a major priority for the cardiology community.

Because most acute coronary syndromes result from plaques that are modest in severity (4,5), coronary angiography is of limited utility in distinguishing lesions with a high short-term risk of causing clinical events. Detailed pathological examination of plaque ruptures has allowed us to define morphological criteria that we believe characterize plaques at high risk for rupture (1). These vulnerable plaques have a large lipid core, a thin fibrous cap, and inflammatory cell infiltration, with calcification resembling plaque rupture lesions but with an intact fibrous cap (1). However, perhaps the greatest problem with this paradigm is that we lack high predictive values for in vivo evidence that these so-called vulnerable plaques actually do go on to rupture. In what was arguably the most thorough attempt to examine the relationship between plaque morphology as identified by intravascular ultrasound (IVUS) and clinical events in living patients, Stone et al. (2) conducted the landmark PROSPECT (Providing Regional Observations to Study Predictors of Events in the Coronary Tree [NCT00180466]) trial, a study of 697 patients presenting with acute coronary syndromes who underwent 3-vessel coronary angiography and grayscale and radiofrequency IVUS-virtual histology (IVUS-VH) imaging after percutaneous coronary intervention (2). Subsequent major cardiac events (MACE) over a median of 3.4 years were recorded and adjudicated to be caused either by the original culprit lesion or by untreated nonculprit lesions (2). Although this group was able to define criteria that were associated with new nonculprit MACE (large plaque burden $\geq 70\%$, minimum luminal area 4.0 mm^2 or less, and the presence of what appeared to be vulnerable plaque characteristics as defined by composition using IVUS-VH), the positive predictive value of these criteria was very low (2). Most patients (88.2%) with lesions consistent with these so-called high-risk characteristics did not have MACE (2).

*Editorials published in *JACC: Cardiovascular Imaging* reflect the views of the authors and do not necessarily represent the views of *JACC: Cardiovascular Imaging* or the American College of Cardiology.

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FIGURE 1 Why Plaque Rupture Occurs at One Site and Not at Other Sites With Similar Plaques Must Remain a Mystery, at Least for Now

A 77-year-old white man with no known medical history was found unresponsive in his house. (A and B) Histological sections showed healing plaque rupture with a large, underlying necrotic core (NC) in the mid-left circumflex artery. Thin-cap fibroatheromas were seen in the distal consecutive sections of the mid-left circumflex artery (C and D) and in the proximal left anterior descending artery (E). (F and G) High-power images showing the thin cap at the 2 different sites. Ca^{2+} = calcification; Th = thrombus.

These findings suggested that although such lesion characteristics are conducive to the occurrence of a subsequent event, they are not sufficient to predict which atheromas will undergo plaque progression on a per-patient basis.

In this issue of *iJACC*, Stone *et al.* (6) attempt to refine these criteria by adding low endothelial shear stress (ESS) as a potential predictor of nonculprit events in the PROSPECT trial. Using the same dataset but adding ESS values for nonculprit lesions using computational fluid dynamics obtained from angiography and IVUS imaging, the investigators explored whether adding such data would provide additional prognostic information about nonculprit lesions.

Maintenance of physiological laminar shear stress is essential for normal vascular function, which includes the regulation of vascular caliber

and the inhibition of proliferation, thrombosis, and inflammation of the vessel wall. Low flow and oscillatory flow are often seen opposite arterial flow dividers that have a predisposition to atherosclerosis. Endothelial cells have different behavioral responses to altered flow patterns that promote atherosclerosis in combination with other well-defined systemic risk factors. Earlier work conducted by Stone *et al.* (7) in the PREDICTION (Prediction of Progression of Coronary Artery Disease and Clinical Outcome Using Vascular Profiling of Shear Stress and Wall Morphology [NCT01316159]) study demonstrated that the positive predictive value for coronary artery disease progression requiring percutaneous coronary intervention during 1-year follow-up was 22% on the basis of plaque anatomy alone (large plaque burden and small minimal lumen area), but it increased to

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