Clinical Significance of Coronary Calcification

To the Editor:

We read with interest the review by Abedin et al, who provide valuable insights into the mechanisms of vascular calcification and teleological concepts. However, regarding the clinical ramifications, they make diverse statements. We agree that, in patients selected by knowledgeable physicians, coronary artery calcification can be used “...for identifying patients at risk for adverse cardiac events.” This position has been adopted in American and European guidelines. However, only 2 paragraphs below, the authors state that “a consensus has developed that coronary calcification is associated with chronic symptomatic coronary artery disease rather than with acute coronary events...” and that these findings have been interpreted as evidence that vascular calcification is protective against acute events. We believe these statements are misleading. They are based on perceptions from cross-sectional data comparing patients who present with acute coronary syndromes and chronic stable angina pectoris. In many patients, the acute coronary syndrome is the first manifestation of coronary artery disease, whereas many patients with chronic disease have a long-standing history. When comparing findings in unstable and stable patients, the different medical history and time course of the disease needs to be taken into account. Intravascular ultrasound studies have produced contradictory results and have in part observed that calcification appeared neutral or even associated with acute coronary events.

A series of histopathologic reports have been published by the Armed Forces Institute of Pathology group that characterize the relationship between plaque rupture and calcification in some detail. These reports demonstrate that calcification is a frequent feature of plaque rupture in victims of sudden coronary death, even in young adults. Among all types of histologically defined types of plaques, acute ruptures were calcified most frequently (80%), whereas healed ruptures were calcified most extensively. Plaque erosions, on the other hand, were associated with little calcium. Calcification was found preferentially in plaques with expansive (“positive”) arterial remodeling, known to be associated with an increased risk of rupture.

Most importantly, a number of prospective studies have consistently reported that the degree of coronary calcification is predictive of hard coronary events (for review, see Rumberger). Abedin et al quote the only study that has ever reported a negative finding in that coronary calcification was not better than risk factors and ECG in predicting hard coronary events. However, follow-up reports from the same study found superior predictive ability of the coronary calcium score, in line with 4 large independent studies (see Rumberger for review) and 2 preliminary reports from the truly unselected general population. Measured in the clinical setting, coronary calcification clearly does not indicate protection but rather a relevant increase in coronary risk. It is our task as physicians to reduce that risk.

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Clinical Significance of Vascular Calcification

In response:

We were pleased to read the letter from Drs Schmermund and Erbel concerning our review on vascular calcification. In their letter, they point out the strong evidence that vascular calcification increases the risk of acute coronary events. We agree entirely with their view. Our reference to the concept that calcification is protective was merely intended to preface our analysis dispelling the notion. Our theoretical analysis showed that, as total calcification burden increases, the soft-hard interface area (hence rupture risk) peaks at intermediate levels of calcification due to coalescence. With progression, the interface area may decline but remains higher than in noncalcified plaque. Thus, both clinical and theoretical evidence support the concept that vascular calcification increases risk. We regret that our article was not clear on this point, and we greatly appreciate the important clarification provided by Drs Schmermund and Erbel.

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