Coronary Calcium Scoring: Calcium Location Needs to Be Integrated!

Coronary calcium scoring (CCS) has been a topic of great interest lately. In a large population-based study comprising 6,722 patients, Detrano et al. (1) have effectively shown that CCS can be a strong predictor of incident coronary heart disease among different racial groups. Henneman et al. (2) have, however, reported that CCS does not reliably exclude the presence of (significant) atherosclerosis. This topic is quite controversial as there is significant evidence from Detrano’s work that higher CCS is associated with an increased risk of acute coronary events.

We think that the location of calcium within the coronary arteries should also be considered. Li et al. (3,4) have shown that the position of the calcium in the plaque is a better determinant of plaque vulnerability than the total calcium load. Using a biomechanical model, predicted maximum stress was found to increase by 47.5% when calcium deposits were located in the thin fibrous cap. The presence of calcium deposits in the lipid core or remote from the fibrous cap resulted in no increase in maximum stress. It was also noted that the presence of calcification within the lipid core may even stabilize the plaque.

Integration of calcium location in CCS will, therefore, enable better assessment of severity of atherosclerosis and prediction of future cardiovascular events.

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We thank Drs. Li, Sadat, and Gillard for their interest in our article on plaque characteristics on multislice computed tomography coronary angiography (MSCTA) versus coronary calcium score (CCS) in patients presenting with suspected acute coronary syndrome (1). The authors raise the possibility that not the total extent of calcium but rather its location within the atherosclerotic plaque is an important determinant of vulnerability. Indeed, the authors have previously demonstrated that while calcifications within the lipid core may stabilize the lesion, the presence of calcifications in the thin fibrous cap may in fact result in high stress and increased likelihood of rupture (2). Accordingly the authors suggest that integration of calcium location in the CCS may improve prediction of future cardiovascular events. Unfortunately, only calcified tissue is recognized on CCS. In contrast, other plaque components are not visualized, and as a result, it remains impossible to differentiate between calcium located in the thin fibrous cap or elsewhere in the lesion. Moreover, it is likely that precisely these small calcified deposits in the thin fibrous cap cannot even be identified during CCS or MSCTA because of the limited resolution of the technique. Indeed, previous comparison of plaque observations between MSCTA and virtual histology intravascular ultrasound showed that plaques deemed to be completely noncalcified on MSCTA still contained some small amount of calcium, albeit only very limited (3). Accordingly, small calcium deposits that potentially predispose the lesion to increased vulnerability may not always be detectable during CCS. Although in the general population with stable coronary artery disease, the presence of noncalcified tissue and small calcium deposits will coincide with larger calcifications and thus evidence of calcium on CCS, this phenomenon may not be the case in younger patients or in patients presenting with suspected acute coronary syndromes (1). Accordingly, for these patients caution remains warranted, as the observation of only minor or even no calcifications during CCS may not reliably exclude the presence of substantial atherosclerosis or potentially vulnerable lesions.
REFERENCES

