

## Letters

### TO THE EDITOR

## Plaque Rupture in Stable Coronary Artery Disease



We read with interest the report by Guagliumi et al. (1) of a prospective diagnostic study investigating sex differences in pathophysiology of ST-segment elevation myocardial infarction and vascular healing after primary percutaneous coronary intervention. The authors did not address nonculprit vessels, and we wonder what the prevalence of multiple plaque ruptures may have been in this cohort. In the context of ST-segment elevation myocardial infarction, it seems likely that observed plaque ruptures and erosions occurred in the acute setting. However, it has also been shown that plaque rupture may be present on follow-up intracoronary imaging in stable patients over a period of up to 6 months (2). Furthermore, plaque rupture has been observed in patients undergoing angiography and intracoronary imaging for the diagnosis of stable angina (3,4), with reported rates of up to 31%. We believe that these previous reports overestimated the prevalence of plaque rupture in stable patients because it remains possible that a change in clinical status prompted clinicians to refer these patients for angiography; that is, they may truly have had unstable angina. Therefore, the prevalence of plaque rupture and erosion on follow-up OCT in the OCTAVIA (Optical Coherence Tomography Assessment of Gender Diversity In Primary Angioplasty) trial is of great interest because the only indication for testing in these patients was research. We write to request these data.

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<http://dx.doi.org/10.1016/j.jcin.2015.01.023>

### REFERENCES

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### REPLY: Plaque Rupture in Stable Coronary Artery Disease



We thank Dr. Mahajan and colleagues for their interest in our study (1), a prospective investigation by optical coherence tomography (OCT) of sex-related differences in the mechanisms of atherothrombosis and vascular response to everolimus-eluting stents in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI). Indeed, they raise an interesting question regarding the putative coexistence of multiple ruptured or eroded plaques in serial OCT assessments of nonculprit coronary segments, including the 9-month follow-up presentation of our study, approximating that of patients undergoing catheterization in the context of stable coronary artery disease.

The concept of destabilized (i.e., ruptured or eroded) plaques not responsible for acute coronary syndromes or anticipating the risk of upcoming cardiovascular events is intriguing in view of a previously published prospective study of grayscale and radiofrequency intravascular ultrasonographic imaging of patients with acute coronary syndromes undergoing PCI, where unanticipated cardiovascular events at follow-up were equally attributable to recurrence at the site of culprit lesions and to nonculprit lesions, the latter particularly if presenting with thin-cap fibroatheromas, large plaque burdens, and/or a small luminal areas (2). The idea of such nonculprit plaques not playing the part of "innocent bystanders" in STEMI is also gaining some degree of consensus, given the results of recent trials of complete versus staged revascularization in patients with multivessel disease (3,4).