Letter by Ward and Figtree Regarding Article, “Mechanisms of Myocardial Infarction in Women Without Angiographically Obstructive Coronary Artery Disease”

To the Editor:

We read with interest the article by Reynolds et al1 but have some reservations regarding the conclusions drawn by the authors based on the presented data. We note that 16 of 42 patients who underwent intravascular ultrasonography examination had plaque rupture as adjudicated by an independent core laboratory, but this had no relationship to ECG changes or left ventricular wall motion abnormality (page 1421, column 1, line 2) or ischemic pattern late gadolinium enhancement on cardiac magnetic resonance (only 1 of 16 with plaque rupture had ischemic late gadolinium enhancement versus 11 of 22 with no plaque rupture; page 1421, column 2, line 8). Based on these data, it is reasonable to conclude that, indeed, the intravascular ultrasonography–detected ruptured plaques were not responsible for the troponin elevation and that plaque rupture was more common in those without definite infarction defined by late gadolinium enhancement. Indeed, given the low incidence of ischemic pattern late gadolinium enhancement in this study, it seems likely that the majority of patients had nonischemic causes for their troponin elevation. Assuming plaque rupture was not operator/wire induced, we wonder whether the high incidence of incidental plaque rupture in patients without myocardial infarction could be explained by high levels of systemic inflammation augmenting the inflammatory response to atherosclerosis or whether these patients simply had high levels of inflammation in their plaques due to poor risk factor control.

Disclosures

None.

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Reference

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