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

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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).
Although the usefulness of targeting nonculprit lesions anticipated to be responsible for future cardiovascular events is part of an ongoing investigation (PROSPECT II & PROSPECT ABSORB—an Integrated Natural History Study and Randomized Trial; NCT02171065), we agree with Mahajan et al. that the true prevalence of ruptured or eroded plaques according to detailed imaging modalities such as OCT would be of paramount interest in patients with stable coronary artery disease. However, they should note that in the OCTAVIA (Optical Coherence Tomography Assessment of Gender Diversity In Primary Angioplasty) study, OCT images were obtained at baseline presentation for STEMI, post-stent implantation, and at 9-month follow-up in the culprit vessel only, as part of the pre-specified study protocol. Therefore, no information can be extrapolated from this study on the prevalence of destabilized plaques in nonculprit vessels. Differently, the OCT technology available at the time of the study enabled a pullback assessment of the culprit vessel starting from approximately 2 cm distal to the target segment for a total length of approximately 5.4 cm upstream. The prevalence and fate of ruptured or eroded plaques in the culprit vessel beyond the treated lesion along the studied segment will be the objects of an upcoming separate OCTAVIA sub-study, which is beyond the scope of the present reply.

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http://dx.doi.org/10.1016/j.jcin.2015.01.022

Please note: The study was promoted and supported by the Italian Society of Invasive Cardiology with unrestricted grant support provided by Abbott Vascular. OCT catheters for the study were donated by St. Jude Medical. Dr. Guagliumi has received consulting fees from Boston Scientific, St. Jude Medical, and AstraZeneca; and grant support from St. Jude Medical, Medtronic Vascular, Boston Scientific, and Abbott Vascular. Dr. Capodanno has received speaker honoraria/consulting from Eli Lilly and Company, The Medicines Company, and AstraZeneca. Dr. Saia has received consulting fees from Abbott Vascular, Eli Lilly and Company, St. Jude Medical; and is on the speakers bureaus of Abbott Vascular, Eli Lilly and Company, St. Jude Medical, Terumo, Biosensors, Edwards Lifesciences, and Boston Scientific.

REFERENCES


Left Atrial Decompression Using Unidirectional Left-to-Right Interatrial Shunt

Initial Experience in Treating Symptomatic Heart Failure With Preserved Ejection Fraction With the W-Wave Device

Approximately one-half of all heart failure (HF) patients have HF with preserved ejection fraction (HFpEF); the incidence of which is expected to rise in parallel with increasing life expectancy (1). Importantly, and unlike HF with reduced ejection fraction (EF), no specific therapy has thus far proven efficacious in pivotal clinical trials enrolling patients with HFpEF. Most patients with decompensated HF present with elevated left atrial (LA) pressure, and the strict control of LA pressure via invasive monitoring in these patients is associated with significant improvements in New York Heart Association (NYHA) functional class, and quality of life, and significant reductions in re-hospitalizations and mortality (2,3). Reported experience with an open (non-valved) interatrial shunt device (Interatrial Septal Device; DC Devices Inc., Tewksbury, Massachusetts) have demonstrated improvement in NYHA functional class in HFpEF patients (4).

The V-Wave implantable shunt (V-Wave Ltd, Or Akiva, Israel) consists of an ePTFE-encapsulated hourglass shaped nitinol frame which is implanted at the interatrial septum and contains 3 porcine pericardial leaflets sutured within to ensure unidirectional flow from the left to the right atrium (Figure 1A). We recently reported the initial successful clinical experience with the V-Wave device in a patient with systolic HF (5). The present case discusses the first experience with the V-Wave device in a patient with HFpEF. A 64-year-old man with a history of coronary artery disease, chronic atrial fibrillation, and ischemic cardiomyopathy with a left ventricular EF of 50% was admitted to our center. Clinically, the patient had HF symptoms of NYHA class III and orthopnea despite high doses of diuretics (furosemide 240 mg/day in divided doses). Right heart catheterization revealed a mean pulmonary wedge pressure of 22 mm Hg, pulmonary artery pressures of 68/21/(39) mm Hg, and a mean right atrial pressure of 12 mm Hg. At baseline, the 6-min hall walk (6MHW) distance was 281 m, the