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Letters

TO THE EDITOR

Early Angiography Use in Patients With Non–ST-Segment Elevation Myocardial Infarction in the United States

Focus on Elderly Patients

In their recent paper, Malta Hansen et al. (1) reported patterns in hospital practices and predictors of early angiography (defined as <24 h) among patients presenting with non-ST-segment elevation myocardial infarction (NSTEMI). One of the findings was a strong association of older age with delayed angiography (>24 h).

This observation deserves particular attention. Although the optimal timing of coronary angiography for NSTEMI has been debated, current guidelines reflect the published reports supporting evidence for early angiography in higher-risk patients (2). These guideline recommendations were also supported in a recent collaborative meta-analysis of 8 randomized controlled trials (RCTs) that suggested lower risk of mortality with early angiography in high-risk patients (3). One of the patient-related factors associated with "high risk" is older age. Multivariate analyses of prior studies have demonstrated age as an independent predictor of adverse cardiovascular outcomes after NSTEMI (4). Furthermore, in a recent meta-analysis of RCTs, we demonstrated that older patients >75 years of age with NSTEMI derive significant benefits from a routine invasive strategy compared with initial conservative strategy (5). However, despite evidence for benefits of a routine (or early) invasive strategy, studies have demonstrated significantly lower rates of invasive (early or delayed) management in older patients (particularly >75 years of age). Similarly, as examined in the study by Malta Hansen et al. (1), older patients are less likely to undergo early angiography.

Two important questions arise from the above findings. First, it highlights physician reluctance regarding evidence-based early angiography in older "high-risk" patients admitted with NSTEMI. Second, the optimal timing of angiography in older patients remains less defined. Several factors such as chronic kidney disease, a higher bleeding risk, and so on might play a role in the decision regarding early or delayed angiography in older patients. Therefore, future studies should specifically investigate patterns and predictors of timing of angiography in older population.

*Aakash Garg, MD
Sahil Agrawal, MD
Marc Cohen, MD

*Division of Cardiology
Newark Beth Israel Medical Center
194 Bloomfield Avenue, Apt 411
Bloomfield, New Jersey 07003
E-mail: drgarg.aakash@gmail.com

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TO THE EDITOR

Does Late Lumen Loss Represent a Measure of the Efficacy of Bioresorbable Scaffolds?

In this journal, Han et al. (1) recently reported the 1-year angiographic follow-up of a randomized

clinical trial that assessed the performance of NeoVas (Lepu Medical, Beijing, China), a novel poly-L-lactic acid bioresorbable scaffold, compared with the well-known XIENCE everolimus-eluting stent (Abbott Vascular, Santa Clara, California). I want to congratulate the investigators for pursuing research on bioresorbable scaffolds after the premature "disappearance" of the Absorb platform. Many physicians and several companies are at the window to see if the increased thrombotic risk observed during the first 3 years of the Absorb follow-up is related to a young and imperfect technology or if current-generation drug-eluting stents are unapproachable, in terms of safety and efficacy, by any bioresorbable technology.

The results of the study by Han et al. (1) show a comparable effect in terms of angiographic outcomes, with a similar primary endpoint of late lumen loss. In contrast, the superiority of this bioresorbable scaffold in terms of strut coverage (an absolute difference of 2.5%) and malapposed struts (an absolute difference of 0.6%) on optical coherence tomography seems of limited importance, given that the hypothesized mechanisms of scaffold thrombosis with Absorb were related to the high strut thickness, to an inadequate technique of implantation, or to premature scaffold dismantling (2–4).

I would also like to make the following observations: 1) Despite dilation with the same size or larger balloons, at similar pressure and with the same starting reference vessel diameter, patients treated with the NeoVas scaffold had smaller post-procedural in-device minimal luminal diameter (2.58 ± 0.40 mm vs. 2.78 ± 0.42 mm; $p < 0.001$), indicating more acute recoil than in patients treated with everolimus-eluting stents. 2) At 1 year, patients treated with the NeoVas scaffold also had greater in-device late loss (0.22 ± 0.33 mm vs. 0.16 ± 0.28 mm; $p = 0.05$), indicating either more chronic recoil or restenotic tissue formation than in patients treated with everolimus-eluting stents, with at least a contribution of the latter witnessed in the optical coherence tomographic substudy by greater device volume obstruction in the NeoVas cohort (24.5 ± 5.9 vs. 10.4 ± 7.0; $p < .001$).

Taken together, these findings could be of concern when the NeoVas scaffold is studied in larger numbers of patients with more complex anatomy.

*Bernardo Cortese, MD
*Cardiac Department
San Carlo Clinic Milano
Via Leonardo da Vinci, Paderno Dugnano
20100 Milan
Italy
E-mail: bcortese@gmail.com

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TO THE EDITOR

Don't Leave the Back Door Open

I read with great interest the article "Neurocognition and Cerebral Lesion Burden in High-Risk Patients Before Undergoing Transcatheter Aortic Valve Replacement: Insights From the Sentinel Trial" (1). I commend the goal of the investigators to try to minimize or eliminate zones of brain infarction (as seen by diffusion-weighted imaging lesions on magnetic resonance imaging) during transcatheter aortic valve replacement. It is also very useful that they attempted to determine the relationship of these new lesions with cognitive decline. However, they did not find a relationship with worsening cognitive deficit in patients with increased volume of these diffusion-weighted imaging lesions, because of what the investigators postulate may be a "floor" effect of the patient population (i.e., these patients were already compromised, such that any further decrease would be difficult to assess). Although this is almost certainly one of the factors, it is also important to view the data acquired.

The discrepancy in the device arm between protected territories of infarction and total volume of infarction, including unprotected territories (2) speaks volumes, literally and figuratively. If I subtract the median volume of infarction in the protected territories of infarction (102.8 mm^3) from the median total volume of infarcted tissue in all territories (294 mm^3), the difference is 191.2 mm^3. Therefore, 65% of lesions were outside the