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Controls EF was 66% (62% to 66%). In R-DCM, baseline EF was 27% (25% to 38%) and current EF was 64% (60% to 66%). For the imaged slice midwall, late gadolinium enhancement was present in 9 of 12 DCM patients and 5 of 12 R-DCM patients. All patients were in New York Heart Association functional class 1.

Myocyte orientation HAs were negative epicardially, near circumferential mesocardially, and positive HAs endocardially and similar across groups. Median (interquartile range) diastolic E2A in R-DCM was 25° (22 to 30°), which was similar to DCM (19° [16 to 27°]; $p = 0.11$) and controls (20° [15 to 29°]; $p = 0.24$). However, systolic E2A in R-DCM was 59° (52 to 66°), significantly greater than DCM (35° [22 to 38°]; $p < 0.0001$), but lower than controls (65° [63 to 71°]; $p = 0.01$). E2A mobility was 35° [29 to 39°] in R-DCM, greater than DCM (9.6° [3.4 to 19°]; $p < 0.0001$), but less than controls (46° [41 to 51°]; $p = 0.001$) (**Figure 1**).

Myocardial contractile recovery is complex and multifactorial, with increasing focus on different patients groups with reverse remodeling, remission and full recovery (3). Clinically, R-DCM is based upon structural, biochemical, and symptomatic improvement with improved LV size and LVEF >40% to 50%. However, recovered patients can still demonstrate abnormal biomarker levels, subclinical dysfunction, and substantial cardiac hospitalizations and heart failure symptoms (4,5). In this study, all R-DCM patients described symptomatic and structural improvement with entirely recovered LV size and EF within reference ranges. However, systolic E2A and sheetlet mobility remained significantly reduced in R-DCM compared with controls indicating persistent microstructural abnormalities in R-DCM, despite normalization of LV size and EF. E2A mobility in R-DCM was greater than in DCM, however, implying a variable microstructural measure that alters along the disease trajectory. This suggests a potential role of DT-CMR in the assessment of DCM recovery/remission and predicting patients at risk of relapse, and reinforces the ability of DT-CMR to provide insight into LV dynamics beyond the limitations of EF.

Limitations of DT-CMR (1) include low spatial resolution, affecting measurements in thin walls. In this cross-sectional study with retrospective analysis of DCM diagnosis, patient heterogeneity remains. This includes disparity between echo and CMR derived EF. Further work in DCM of extrinsic origin such as myocarditis may yield different microstructural findings.

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Mismatch Between Diameter Stenosis and Plaque Atheroma Volume

Challenging Glagov Phenomenon?

We eagerly read the report by Lee et al. (1) that found slower rate of total and noncalcified percentage of atheroma volume (PAV) progression and a faster progression of calcified PAV in statin-taking patients without history of coronary artery disease who underwent coronary computed tomography angiography at an interscan interval ≥2 years when compared with statin-naïve patients. To gain an understanding of the results, some questions remain unanswered.

This report conflicts with other progression/regression trials for the following reasons: 1) the

percentage of stenosis severity does not correlate with the baseline percentage of PAV because, per the Glagov phenomenon, PAV >40% starts encroaching the lumen. Therefore, 13% diameter stenosis should correspond to a PAV much >40%. Indeed, most progression/regression clinical trials have baseline PAV of ~40%. 2) The observed annual change in PAV in patients taking statins is not only going in the direction of progression (instead of regression), but it is also much larger than any previous report.

In the interscan interval, 8.1% of patients underwent revascularization (surgical and percutaneous) with a significantly higher number of patients belonging to statin-taking group (n = 96) than the statin-naïve group (n = 6) (p = 0.001), which contradicts the slower high-risk plaque progression in the statin-taking group. Also, the number of lesions in the statin-taking patients with diameter stenosis ≥50% is reported as 76 at follow-up, which is lower than the number of revascularizations in the group during the interscan period.

Inclusion of lesions that were revascularized in the interscan period may have further confounded the results if these segments have been excluded in the analysis.

Intensity, duration, and type of statin therapy were not indicated in the study although they are important factors as prior studies have shown data that change in atheroma size is not constant with different statins.

The number of patients starting therapy during the interscan period is significantly high (38.8%), and the exclusion of data for their time of initiation of therapy can have an impact on annualized change in PAV.

Lastly, we feel the inclusion of absolute data for external elastic membrane, lumen, and plaque volumes should be included. Stenosis severity percentage in the population is clinically insignificant, so the results of this study therefore cannot be generalized to a population with clinically significant disease.

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THE AUTHORS REPLY:

We thank Drs. Gill and Garcia-Garcia for their interest in our paper (1). Drs. Gill and Garcia-Garcia are concerned about 1) the lack of correlation between the percentage of diameter stenosis severity (13.6%) and the percentage of atheroma volume (PAV) (13.3%) at baseline in our data based on the Glagov phenomenon and 2) that previous clinical trials have reported baseline PAV as close to 40% and lesser annual change in PAV. However, these prior trials mostly represented a high-risk population indicated for invasive assessment, and consequently, both the PAV and stenosis severity were much greater than lesions included in our study (2). The PARADIGM (Progression of Atherosclerotic Plaque Determined by Computed Tomographic Angiography Imaging) registry was designed to represent the patients at low risk or at an earlier stage of coronary artery disease, who would possess much lower PAV than patients in previous studies would. This difference in study population makes the direct comparison of PAV between the present study and previous clinical trials inappropriate. Furthermore, what the Glagov phenomenon acknowledges is that "functionally" important luminal stenosis may be delayed until the lesion occupies 40% of the internal elastic lamina area and the development of positive remodeling during the course (3). It is difficult to agree that the lesions with mean diameter stenosis of 13% are functionally important and therefore should correspond with PAV much >40%. In our study, only 1.6% of lesions had ≥50% diameter stenosis, and positive remodeling was observed in 53.5% of lesions at baseline. The result of our study supports the findings of previous studies rather than conflicting with them.

We certainly acknowledge Drs. Gill and Garcia-Garcia's concern about the revascularized lesions in the interscan interval and agree that inclusion of revascularized lesions in the interscan interval may have further confounded the results. We excluded "patients" who revascularized during the interscan interval, and hence all of their lesions, from the analysis because the revascularization occurred prior to the follow-up coronary computed tomography angiography scan due to the observational nature of the study. This design makes inadequate the direct