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LETTERS TO THE EDITOR

CMR-Verified Lower LA Strain in the Presence of Regional Atrial Fibrosis in Atrial Fibrillation

The regional relationship between left atrial (LA) strain and fibrosis is not known. Late gadolinium enhancement (LGE) cardiac magnetic resonance (CMR) identifies regions of possible atrial fibrosis (1). LA strain measurement, extracted by feature tracking using echocardiography or cine CMR, is an emerging tool for assessment of atrial function, which correlates globally with atrial LGE (2-5). Here, we report data comparing *regional* strain with the *regional* presence of fibrosis by LGE enhancement.

Eighteen patients with atrial fibrillation (AF) and 12 healthier controls (51 ± 13 years of age; 30% female) were imaged using 3-dimensional LGE and 2-dimensional cine CMR with 2- and 4-chamber views on a Siemens 1.5-T scanner (Erlangen, Germany). Controls had no AF, no cardiomyopathies, and no more than mild cardiac dysfunction or chamber enlargement. The 3-dimensional LGE sequence (1) ($0.7 \times 0.7 \times 2$ mm^3 after zero-filling) used 0.2 mmol/kg gadobutrol (Gadavist, Bayer Healthcare, Leverkusen, Germany). LGE burden was measured by segmenting LGE-enhanced pixels on the atrial wall, normalized by atrial myocardial volume. LGE-enhanced pixels were defined as atrial wall pixels with a contrast-to-noise ratio (vs. blood pool) >3.5. Noise was measured as the SD of signal within a blood pool region of interest (ROI). The atrial myocardial volume was estimated using the surface area of a scalene ellipsoid (measuring 3 radii) and a myocardial thickness of 2.1 mm. LA volumes and LA ejection fraction (EF) were measured using the biplane method.

Tangential strain, largely representative of long-axis longitudinal strain, was estimated using a customized point-matching method for the LA contours at phases of maximum and minimum area on the cine MRIs. The contours were discretized into points, with points matched between phases using an iterative process that included closest point matching followed by piece-wise affine transformation. Strain was calculated as the change in distance between neighboring points, normalized by the initial distance. Regional 2- and 4-chamber strain maps were generated, and "global" strain was calculated. Continuous variables were compared using Pearson correlation. Two-tailed Student *t* tests were used to compare between groups.

Using 4-chamber LGE views aligned visually with corresponding cines, ROIs were drawn on the cines indicating regions with and without LGE enhancement. These ROIs were automatically propagated to the strain maps, so that all 4-chamber strains were labeled as either positive or negative for LGE enhancement in a blinded fashion. Receiver-operating characteristic curve analysis identified the optimal strain threshold for predicting LGE enhancement.

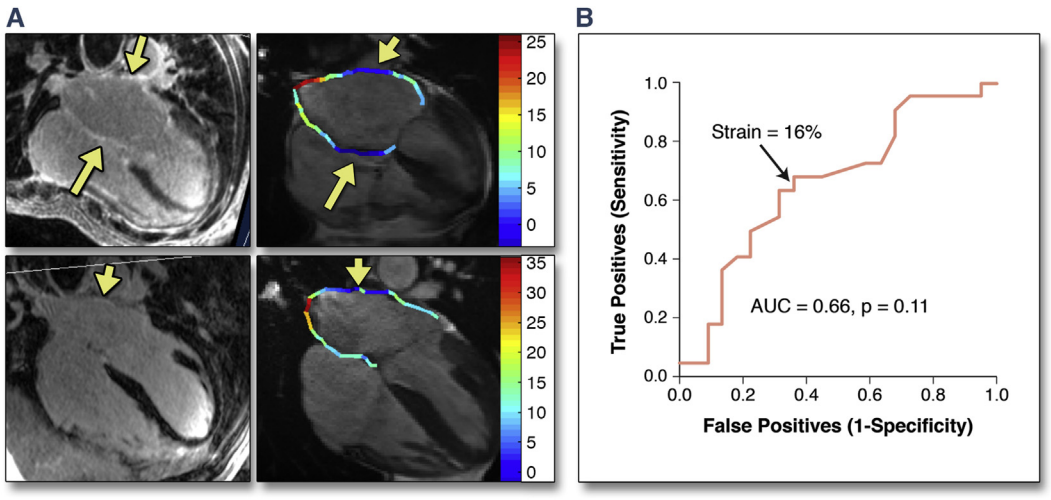
AF patients (1 with nonparoxysmal AF) had lower global strain values versus controls (17 ± 11% vs. 27 ± 10%; $p = 0.02$), greater LA volumes (60 ± 18 ml/m^2 vs. 35 ± 10 ml/m^2; $p < 0.001$), lower LA EF (34 ± 18% vs. 52 ± 10%; $p = 0.002$), but similar LGE burden (6 ± 7% with AF vs. 3 ± 3%; $p = 0.17$). Between groups, left ventricular EF, prevalence of hypertension, and sex were similar, but age and body mass index were greater in AF patients ($p = 0.04$).

Global strain correlated well with decreasing maximum LA volume and increasing LA EF (both $r = \pm 0.57$; $p \leq 0.001$), but modestly with LGE burden ($r = -0.36$; $p = 0.049$). By multivariate regression (LGE burden, AF, LA volume), LA volume had a marginal association with strain ($p = 0.056$). Importantly (**Figure 1**), in atrial regions of LGE enhancement, regional strain was lower compared with regions without LGE (14 ± 11% vs. 20 ± 12%; $p = 0.007$). By receiver-operating characteristic analysis, strain <16% best predicted LGE enhancement (area under the curve = 0.66; $p = 0.11$, ns).

This study confirms (3-5) strong correlations between global strain and LA volumes and EF, and a weak correlation with LGE burden in paroxysmal AF patients (2) that have less remodeling. Additionally, there is a regional relationship between locations of atrial LGE (potentially fibrosis) and reduced strain.

The cohort size was small, and the control group included subjects with minor cardiovascular findings. Our strain methodology requires further refinement and extension to 3 dimensions. Strain mapping might be an alternative approach for detection of atrial fibrosis, serving in risk stratification of AF therapies such as ablation, but this remains undemonstrated. In conclusion, we report the new finding that lower atrial strain collocates with regional LGE enhancement.

FIGURE 1 LGE Enhancement Is Associated With Low Regional Strain



(A) Reformatted 3-dimensional late gadolinium enhancement (LGE) volumes show LGE enhancement **(left)** located in sites of low strain **(right)** in 2 atrial fibrillation subjects. Strain (in %) is color-coded. **(B)** Using receiver-operating characteristic analysis, strain <16% best predicts LGE enhancement (area under the curve [AUC] = 0.66, p = 0.11, not significant).

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Statin Use Is Associated With Fewer High-Risk Plaques on Coronary CT Angiography

Although there is an ongoing debate whether the detection of individual high-risk plaques (HRP) may help to improve patient outcomes, emerging data from coronary computed tomography angiography (CTA) studies suggest that the presence of HRP features (i.e., positive remodeling and low CT plaque attenuation) is independently associated with a 10- to 20-fold increased risk for future acute coronary syndrome (1). Statin therapy, perhaps via anti-inflammatory properties, may have favorable effects on coronary atherosclerotic plaque composition and lead to plaque stabilization by accelerating plaque calcification and regression of HRP features such as positive remodeling and low CT plaque attenuation (2,3). We performed an observational cross-sectional subanalysis of the ROMICAT (Rule Out Myocardial Ischemia/Infarction Using Computer Assisted Tomography) II trial to determine whether patients on statin therapy had lower prevalence of HRP as compared to those not on statins.