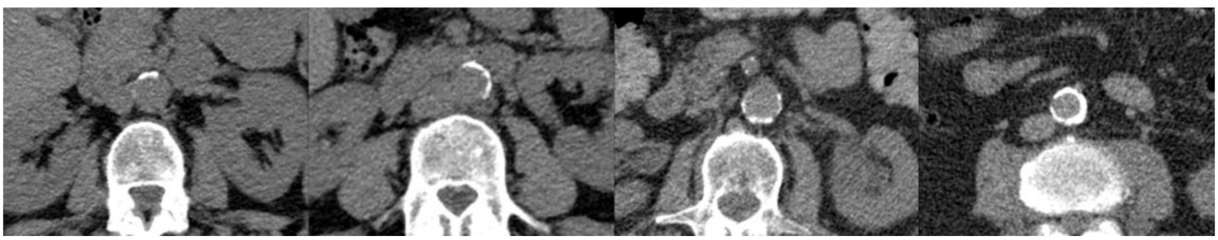
TABLE 1 Associations of Annularity of Calcification in the AA With Total Mortality

	Model 1		Model 2		Model 3		Model 4	
	HR (95% CI)	p Value	HR (95% CI)	p Value	HR (95% CI)	p Value	HR (95% CI)	p Value
None	1.00		1.00		1.00			
1°–90°	1.09 (0.60–2.01)	0.771	0.86 (0.45–1.65)	0.658	1.08 (0.46–2.56)	0.859		
91°–180°	1.68 (0.84–3.38)	0.143	1.21 (0.57–2.58)	0.614	1.76 (0.51–6.07)	0.374		
181°–270°	2.31 (1.08–4.91)	0.03	1.39 (0.62–3.13)	0.422	2.12 (0.53–8.55)	0.291		
271°–360°	3.43 (1.65–7.13)	0.001	2.31 (1.08–4.95)	0.032	3.66 (0.86–15.65)	0.080		
p Value for trend		<0.001		0.002		0.006		
z Score log (1 + mAgatston)					0.81 (0.47–1.41)	0.461	1.35 (1.01–1.80)	0.045

Model 1 is adjusted for age and sex. Model 2 is adjusted for age, sex, smoking status, dyslipidemia, body mass index, diabetes, and hypertension. Model 3 is adjusted as model 2 + adjustment for the log(1+ mAgatston score). Model 4 is adjusted as model 2 but instead of the circularity scoring, the log(1+ mAgatston score) is modeled as a predictor variable.



Illustrated are, left to right: 1° to 90°, 91° to 180°, 181° to 270°, 271° to 360°. Examples are of the infrarenal AA.
AA = abdominal aorta; CI = confidence interval; HR = hazard ratio; mAgatston = modified Agatston.

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REFERENCES

1. Prentice RL. A case-cohort design for epidemiologic cohort studies and disease prevention trials. Biometrika 1986;73:1–11.

2. Allison MA, Hsi S, Wassel CL, et al. Calcified atherosclerosis in different vascular beds and the risk of mortality. Arterioscler Thromb Vasc Biol 2012;32:140–6.

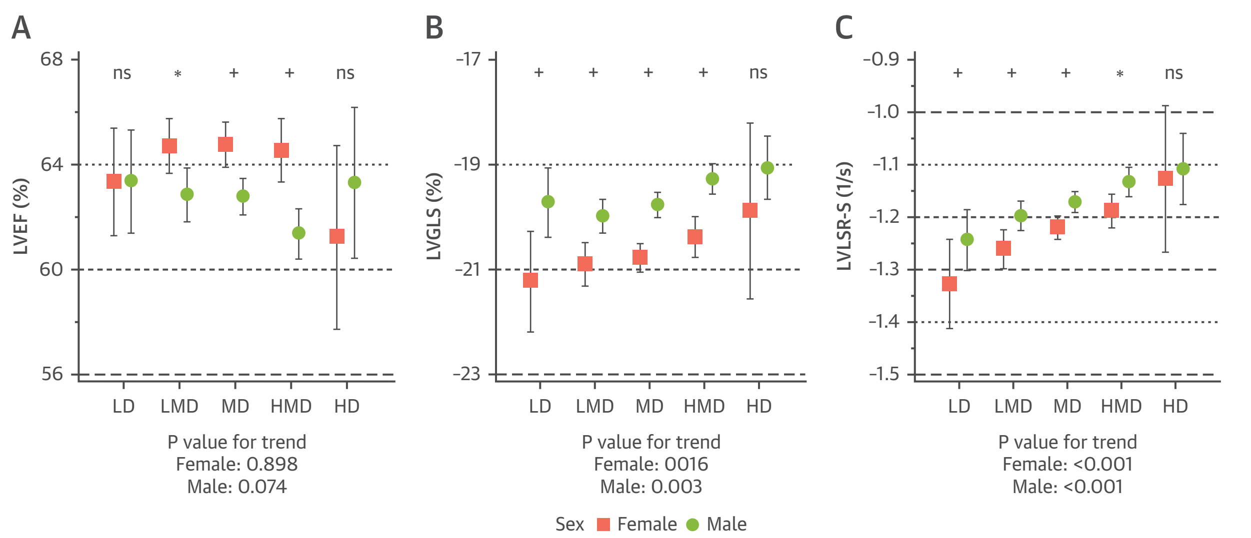
3. Agatston A, Janowitz W, Hildner F, Zusmer N, Viamonte M, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. J Am Coll Cardiol 1990;15:827–32.

4. London GM, Guérin AP, Marchais SJ, Métivier F, Pannier B, Adda H. Arterial media calcification in end-stage renal disease: impact on all-cause and cardiovascular mortality. Nephrol Dial Transplant 2003;18:1731–40.

Left Ventricular Longitudinal Strain and Strain Rate Values According to Sex and Classifications of Sports in the Young University Athletes Who Participated in the 2015 Gwangju Summer Universiade

Left ventricular (LV) strain values measured by 2-dimensional speckle tracking echocardiography (2DSTE) represent global and regional myocardial functions. These values can give prognostic information, detect subclinical LV changes, and distinguish physiologic adaptation from pathologic hypertrophy. Because highly trained athletes can have altered cardiac structures, it is difficult to differentiate normal adaptation from pathologic changes. In this regard, 2DSTE can be a useful tool for the screening of athletes (1). Because the differences in myocardial deformation according to sex and classifications of sports have been poorly studied in university athletes, we investigated the impact of sex and classifications of sports on LV deformation in 1,120 (22 ± 2 years of age; 649 men) young athletes during the 2015 Gwangju Summer

FIGURE 1 LVEF, LVGLS, and LVGLSR-S According to Sex and Classification of Sports



(A) LVEF is similar among groups by classification of sports. **(B, C)** LVGLS and LVGLSR-S show linear correlations with the increased cardiovascular demand of sports disciplines. *p $<$ 0.005 between women and men; +p $<$ 0.001. HD = highest demand; HMD = high moderate demand; LD = lowest demand; LMD = low moderate demand; LVEF = left ventricular ejection fraction; LVGLS = global peak longitudinal systolic strain; LVGLSR-S = LVGLS strain rate; MD = moderate demand; ns = not significant.

Universiade. Sports were divided according to cardiovascular demand into lowest demand (LD) (including riflery and golf), low moderate demand (LMD) (including baseball and table tennis), moderate demand (MD) (including running and soccer), high moderate demand (HMD) (including basketball and swimming), and highest demand (HD) (including rowing and triathlon) (2).

Left ventricular ejection fraction (LVEF) was 63.4 ± 6.3%, and left ventricular mass index (LVMi) was 77.3 ± 21.8 g/m^2. Left ventricular global longitudinal peak systolic strain (LVGLS) and left ventricular global longitudinal strain rates (LVGLSR-S) were −20.1 ± 2.1% and −1.19 ± 0.17 s^{-1}, respectively. Women had lower LV volumes and LVMi (67.6 ± 15.9 g/m^2 vs. 83.6 ± 21.7 g/m^2, respectively; $p < 0.001$) and higher LVEF (64.5 ± 6.2% vs. 62.5 ± 6.3%, respectively; $p < 0.001$) than men, although classifications of sports and training times were similar. Along with the increase in cardiovascular demand, body surface area, body mass index, LV volumes, and LVM were increased, whereas LVGLS and LVGLSR-S were decreased ($p < 0.001$) (Figure 1).

The main findings of this study are significant sex differences in strain values, even though these values were within normal limits from the previous meta-analysis (3). Women had significantly higher LVGLS (−20.7 ± 2.1% vs. −19.7 ± 2.0%, respectively; $p < 0.001$) and LVGLSR-S (−1.23 ± 0.17 s^{-1} vs. −1.17 ± 0.17 s^{-1}, respectively; $p < 0.001$) than men. The effect of sex hormones, especially estrogen, on cardiac function would be a possible explanation for these sex differences (4).

This study also demonstrated that LVGLS and LVGLSR-S values significantly decreased along with the increase in cardiovascular demand of sports disciplines. Athletes with HD had the lowest values of LVGLS and LVGLSR-S. Cardiac geometric changes from repeated exercise would be a possible explanation. Because LVMi showed significant negative correlations with LVGLS ($r = -0.211$; $p < 0.001$) and LVGLSR-S ($r = 0.298$; $p < 0.001$), LVMi may be a confounder of strain values in different types of sports. However, classification of sports showed significant correlations with LVGLS and LVGLSR-S, even after adjusting for LVMi. Unlike the study by D'Ascenzi et al. (5), we reported results showing that exercise training leads to lower (more positive) strain values. Further studies will be needed to clarify this issue. Systolic blood pressure and heart rate were significantly different according to the type of sport in this study, and these may also affect the strain values. After adjustment for systolic blood pressure and heart rate, classification of sports was as significantly associated with LVGLS and LVGLSR-S as ever ($p < 0.001$ and $p < 0.001$, respectively). Because there were significant ethnic differences among groups according to their sport types, these differences might have affected the correlations in this study. However, the statistical significance remained evident after the adjustment for ethnicity ($p < 0.001$ for LVGLS and $p < 0.001$ for LVGLSR-S).

In conclusion, female university athletes had lower LV volumes, mass, and higher LVEF and strain values than men. LVGLS and LVGLSR-S were significantly decreased along with the increase of cardiovascular demands. The present study demonstrated that there are sex or sports discipline differences in LV mechanics as measured by 2DSTE in highly trained university athletes. Because this was a cross-sectional study, further long-term follow-up studies will be needed to determine whether the reduced strain values reflect subtle myocardial damage and its clinical significance.

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REFERENCES

1. Caselli S, Montesanti D, Autore C, et al. Patterns of left ventricular longitudinal strain and strain rate in Olympic athletes. J Am Soc Echocardiogr 2015;28:245–53.

2. Mitchell JH, Haskell W, Snell P, Van Camp SP. Task Force 8: classification of sports. J Am Coll Cardiol 2005;45:1364–7.

3. Yingchoncharoen T, Agarwal S, Popović ZB, Marwick TH. Normal ranges of left ventricular strain: a meta-analysis. J Am Soc Echocardiogr 2013;26:185–91.

4. Deroo BJ, Korach KS. Estrogen receptors and human disease. J Clin Invest 2006;116:561–70.

5. D'Ascenzi F, Pelliccia A, Alvino F, et al. Effects of training on LV strain in competitive athletes. Heart 2015;101:1834–9.

Triglyceride-to-High-Density Lipoprotein Cholesterol Ratio and Vulnerable Plaque Features With Statin Therapy in Diabetic Patients With Coronary Artery Disease

Frequency-Domain Optical Coherence Tomography Analysis

Ongoing cardiovascular risks in patients with type 2 diabetes mellitus (T2DM), despite the use of statins, need additional therapeutic targets. We investigated whether diabetic dyslipidemia, characterized by hypertriglyceridemia and low high-density lipoprotein cholesterol (HDL-C), was associated with plaque features in 267 statin-treated patients with T2DM and coronary artery disease who underwent frequency-domain optical coherence tomography (FD-OCT) within the target vessel for percutaneous coronary intervention.

FD-OCT measures and fasting blood biochemical data were compared in subjects stratified by triglyceride-to-HDL-C ratio tertiles. A generalized estimating equations approach was used to consider the intraclass correlation because of the multiple plaques analyzed within a single patient's data. Multivariate linear regression analyses were performed to identify independent determinants for lipid arc (°) and cholesterol crystal (CC) presence. Receiver-operating characteristic analysis was performed to determine the optimal cutoff value of the triglyceride-to-HDL-C ratio for lipid-rich plaque containing CC. The Institutional Review Board of the Cleveland Clinic (Cleveland, Ohio) approved this retrospective study. All statistical analyses were performed using SPSS version 17.0 software (SPSS, Inc., Chicago, Illinois).

A total of 482 nonculprit and 325 culprit plaques were analyzed within 423 imaged target vessels. Nonculprit and culprit plaques in patients with a higher triglyceride-to-HDL-C ratio exhibited a larger lipid burden and a higher frequency of CC (**Figures 1A and 1B**). Patients with a higher triglyceride-to-HDL-C ratio had a larger total lipid index (1,309.6 mm° vs. 2,933.4 mm° vs. 4,105.6 mm°; $p = 0.005$), by calculating the sum of the lipid index within the entire imaged vessel. After adjusting for age, sex, risk factors, medications, glycosylated hemoglobin (HbA_{1c}), and lipids, the triglyceride-to-HDL-C ratio was an independent determinant of lipid arc