

on the arterial side of the stent. The appearance of flare spots in IVOCT images is consistent with light reflecting from surfaces formed at these gap boundaries before returning to the catheter.

Sahar Elahi, PhD
Thomas E. Milner, PhD
Richard J. Rapoza, PhD
Jouke Dijkstra, PhD
Marc D. Feldman, MD*

*University of Texas Health Science Center
Cardiac Catheterization Laboratory
Room 5.642 U
7703 Floyd Curl Drive
San Antonio, Texas 78229
E-mail: feldmanm@uthscsa.edu
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REFERENCE

1. Gutiérrez-Chico JL, Radu MD, Diletti R, et al. Spatial distribution and temporal evolution of scattering centers by optical coherence tomography in the poly(L-lactide) backbone of a bioresorbable vascular scaffold. *Circ J* 2012;76:342-50.

Cardiac Steatosis and Left Ventricular Dysfunction in HIV-Infected Patients Treated With Highly Active Antiretroviral Therapy



Heart disease is a major contributor to morbidity and mortality in persons infected with human immunodeficiency virus (HIV), and both HIV and highly active antiretroviral therapy (HAART) may be associated with abnormalities in cardiac function and metabolism (1). Ectopic fat deposition in cardiomyocytes constitutes one possible mechanism, especially considering the influence of HIV itself and HAART on the human metabolic milieu. To test this hypothesis, we studied 27 HIV-seropositive (+) cases on stable HAART (≥ 3 years) and 22 HIV-negative control subjects; neither group had history of personal or family history of cardiovascular disease.

Myocardial triglyceride content was measured by magnetic resonance spectroscopy (3T MAGNETOM Verio, Siemens, Germany) (2) and revealed a 3-fold elevation in cases compared with control subjects (Figure 1A). To assess left ventricular (LV) function, magnetic resonance tissue tagging was performed on a mid-ventricular short-axis image (3). Both peak

circumferential strain and the peak rate of circumferential strain in diastole were impaired in cases compared with control subjects (Figures 1B and 1C, respectively). Clinical evidence of lipodystrophy was also assessed by physical exam of the dorsal back, neck, extremities, and abdomen.

Myocardial triglyceride content was significantly associated with circumferential strain ($r = 0.41$, $p = 0.009$) and diastolic strain rate ($r = -0.47$, $p = 0.004$). Moreover, HAART exposure was also significantly associated with myocardial triglyceride

content ($r = 0.42$, $p = 0.043$) and diastolic strain rate ($r = -0.43$, $p = 0.029$), both of which were significantly higher with clinical presence of lipodystrophy ($p = 0.02$ and 0.001 , respectively).

The data herein support the hypothesis that ectopic fat deposition in the myocardium may contribute to cardiac disease in patients with HIV infection treated with HAART. In this study, clinically visible lipodystrophy predicted myocardial triglyceride content and LV dysfunction in HIV(+) patients; this may provide an important mechanistic insight

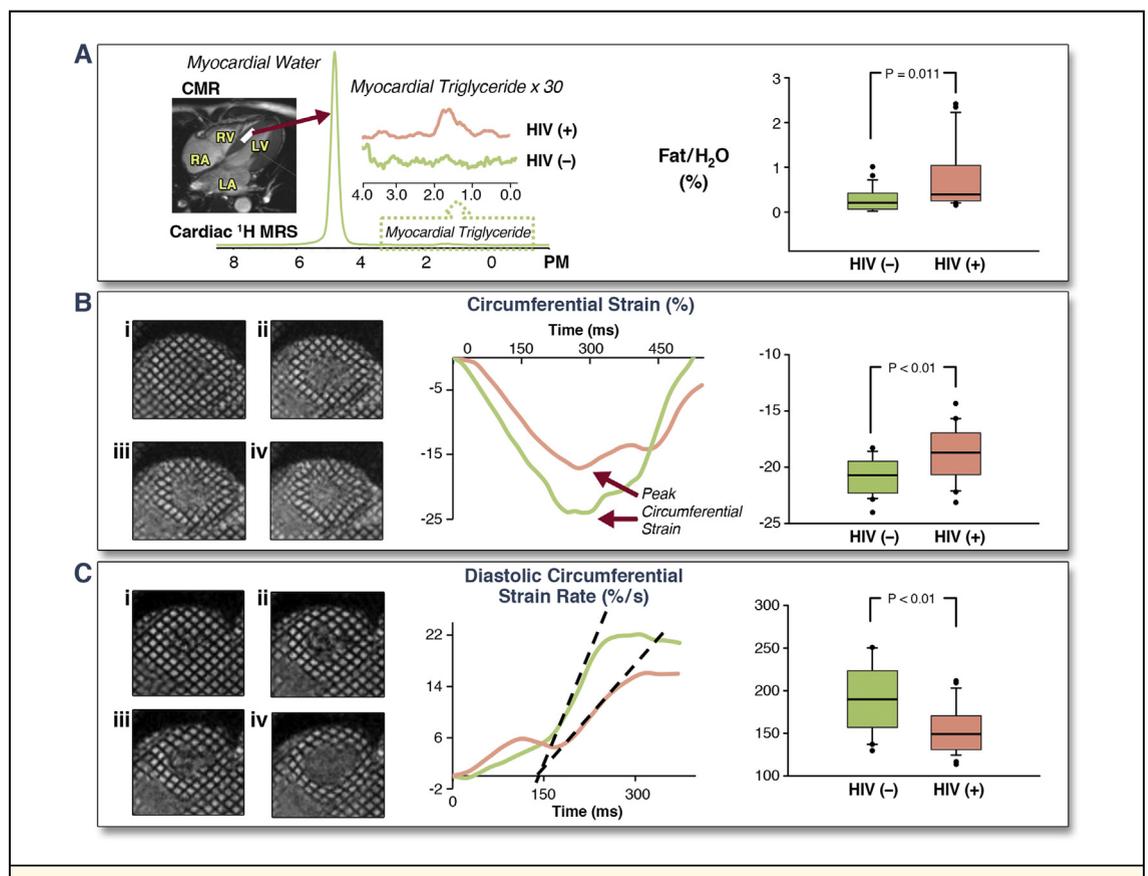


FIGURE 1 Ectopic Fat Deposition and LV Dysfunction in HIV-Infected Patients Receiving HAART

(A) Representative illustration of a typical cardiac magnetic resonance (CMR) image, showing all 4 chambers of the heart. The white box over the intraventricular septum illustrates the voxel position during spectroscopy. The green line illustrates a representative cardiac spectra, showing a large area of resonance for myocardial water and a small area of resonance for myocardial triglyceride content; magnified directly above for a representative human immunodeficiency virus (HIV)-positive (+) subject (pink line) and a representative HIV-negative (-) subject (green line). The summary data demonstrate a 3-fold elevation in myocardial triglyceride content in HIV-positive subjects (pink bar) versus well-matched HIV-negative control subjects (green bar). (B, i to iv) Representative illustration of a mid-short-axis image of the LV, with tissue tagging applied at end diastole (i) and progressing to end systole (iv). The graph shows the circumferential strain from a representative HIV-positive subject (pink line) and control subject (green line). The summary data demonstrate impaired circumferential strain in cases versus control subjects. (C, i to iv) Representative illustration of a mid-short-axis image of the left ventricle, with tissue tagging applied at end systole (i), progressing to diastasis (iv). The graph represents the diastolic circumferential strain rate from a representative HIV-positive subject (pink line) and a control subject (green line). Time 0 represents end systole. The dashed line indicates the rate of ventricular relaxation. The summary data show impaired diastolic strain rate in cases versus control subjects. Data are presented as mean \pm SE. HAART = highly active antiretroviral therapy; ^1H MRS = hydrogen ion magnetic resonance spectroscopy.

into the pathogenesis of heart disease in patients treated for HIV infection. Lipodystrophy can indeed lead to ectopic spillover of triglyceride into non-adipose tissues—including the myocardium—and has previously been associated with adverse LV remodeling (2). Furthermore, that HAART exposure also predicted myocardial triglyceride content and LV dysfunction provides further insight into the pathogenesis of heart disease in HIV(+) patients. For example, HAART has been associated with lipodystrophy, hyperlipidemia, and hyperglycemia (4) and may be directly cardiotoxic (5). Thus, whereas HAART has significantly reduced HIV-related morbidity and mortality, we speculate that HAART may also contribute to the cardiovascular decline in HIV(+) patients. Future studies are warranted to further investigate these findings.

Michael D. Nelson, PhD
Lidia S. Szczepaniak, PhD
Troy M. LaBounty, MD
Edward Szczepaniak, PhD
Debiao Li, PhD
Mourad Tighiouart, PhD
Quanlin Li, MS
Rohan Dharmakumar, PhD
Gregg Sannes, RN
Zhaoyang Fan, PhD
Roya Yumul, PhD, MD
W. David Hardy, MD
Antonio Hernandez Conte, MD, MBA*

*Cedars-Sinai Medical Center
Department of Anesthesiology
8700 Beverly Blvd., Suite 8211
Los Angeles, California 90048
E-mail: antonio.conte@cshs.org
<http://dx.doi.org/10.1016/j.jcmg.2014.04.024>

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REFERENCES

1. Varriale P, Saravi G, Hernandez E, Carbon F. Acute myocardial infarction in patients infected with human immunodeficiency virus. *Am Heart J* 2004;147: 55-9.
2. Nelson MD, Victor RG, Szczepaniak EW, Simha V, Garg A, Szczepaniak LS. Cardiac steatosis and left ventricular hypertrophy in patients with generalized lipodystrophy as determined by magnetic resonance spectroscopy and imaging. *Am J Cardiol* 2013;112:1019-24.
3. Nelson MD, Szczepaniak LS, Wei J, et al. Diastolic dysfunction in women with signs and symptoms of ischemia in the absence of obstructive coronary

artery disease: a hypothesis-generating study. *Circ Cardiovasc Imaging* 2014; 7:510-6.

4. Wanke CA. Epidemiological and clinical aspects of the metabolic complications of HIV infection: the fat redistribution syndrome. *AIDS* 1999;13: 1287-93.

5. Fiala M, Murphy T, MacDougall J, et al. HAART drugs induce mitochondrial damage and intercellular gaps and gp120 causes apoptosis. *Cardiovasc Toxicol* 2004;4:327-37.

A Comprehensive Evaluation of Left Atrial Performance Using Volumetric Analysis, Strain, and Strain Rate Imaging



We read with great interest the article by Habibi et al. (1), which demonstrates the association of left atrial (LA) function and the development of heart failure using cardiac magnetic resonance (CMR) feature-tracking. We fully agree that LA function assessment has the potential to gain an incremental role in the early diagnosis and risk stratification already at pre-clinical stages of heart failure (2).

As correctly cited by the investigators, LA echocardiographic speckle tracking studies (3) have demonstrated that a comprehensive assessment of LA deformation should include strain and strain rate parameters to describe the 3 functional components of atrial physiology: 1) reservoir function (collection of pulmonary venous return during ventricular systole—assessed with global peak longitudinal strain and global peak positive strain rate); 2) conduit function (passage of blood to the left ventricle during early ventricular diastole—assessed with global passive longitudinal strain and global peak early negative strain rate); and 3) contractile booster pump function (augmentation of ventricular filling during late ventricular diastole—assessed with global longitudinal strain during atrial contraction and global peak late negative strain rate). Furthermore, when using CMR, an atrial volumetric analysis is also well established to assess these 3 functional components (4).

Habibi et al. (1) have now applied CMR feature-tracking strain—but not strain rate—to assess LA reservoir and contractile booster pump function without studying conduit function. Conversely, they used volumetric indices to study LA reservoir and conduit function but not contractile booster pump function. It is important to note that there is evidence to suggest that impaired LA contractile booster pump function assessed with volumetric analysis has strong prognostic implications for adverse cardiac events (including development of heart failure) in asymptomatic patients at risk for left ventricular diastolic dysfunction (5).

A more comprehensive analysis including all atrial deformation parameters (strain and strain rate) and