

Myocardial Steatosis is Associated with Regional Ventricular Dysfunction

C-Y. Liu¹, A. Redheuil¹, E. Chamara¹, J. Lima¹, D. Bluemke², and S. Lai³

¹Department of Radiology, Johns Hopkins Hospital, Baltimore, MD, United States, ²Radiology and Imaging Sciences, National Institutes of Health, Bethesda, MD, United States, ³Department of Epidemiology, Johns Hopkins School of Hygiene and Public Health, Baltimore, MD, United States

Introduction: The increased myocardial triglyceride pool is associated with impaired myocardial function in animal experiments (1). Human studies also indicate that myocardial steatosis is associated with impaired left ventricular filling and diastolic dysfunction using echocardiography (2,3). Proton Magnetic Resonance Spectroscopy (¹H-MRS) has proven to be reliable and reproducible in measuring myocardial triglyceride content in humans (2-4). The primary goal of the present study was to evaluate myocardial fat content in an at risk cohort of subjects with HIV infection using ¹H-MRS, and to correlate the septal triglyceride content with the regional ventricular function measured by myocardial strain using tagged MRI.

Materials and Methods: All MRI/MRS studies were performed using a 3T MR scanner (TIM Trio, Siemens) in 43 HIV infected and non-infected individuals who had no clinical cardiovascular disease. To measure left ventricular function, the entire heart was imaged in short-axis orientation using a retrospectively gated TrueFisp sequence. The short-axis along with the two and four chamber views were used to position the spectroscopic volume (6-8ml voxel) within the interventricular septum. Myocardial ¹H-MRS spectra were obtained with ECG and navigator gated with water suppressed (32 averages) and unsuppressed (4 averages) PRESS, TR/TE =1-RR/30ms. Fat content was quantified with Amares/MRUI at resonance frequency of lipids at 0.9 and 1.3 ppm and related to water in unsuppressed spectra and expressed as fat/water percent ratios. To assess the regional heart function, myocardial MR-tagged images were obtained in the short-axis scan plane (10mm thickness; 256x125, 25ms temporal resolution) at the midlevel of the LV myocardium. The average midwall circumferential strain (Ecc, in percentage) in the systolic phase and peak early diastolic strain rate (SR_E, in 1/second) was determined in all myocardial segments, except the apical segment 17, in the 3 short-axis slices by HARP (5). By convention, the value of Ecc is normally negative during the contraction of the ventricle. Less negative Ecc (or positive Ecc) indicates regional LVD dysfunction.

Results: Using 1% as the threshold for normal vs. abnormal lipid content, the overall prevalence of cardiac steatosis was 46% (20/43 patients) by ¹H-MRS. Table 1 summarizes the global heart function parameters and the regional LV functional measurements by tagged MRI stratified by high (≥1%) and low (<1%) myocardial fat fraction groups. Mid-wall systolic strain (Ecc) was significantly reduced (i.e., less negative) (p<0.01) in the septum and left anterior descending (LAD) coronary territory in the high fat fraction group. The diastolic strain rate (SR_E) was also significantly lower (p<0.05) in the right coronary artery (RCA) territory in this group, although this area was not directly measured by ¹H-MRS. Global function of the LV was normal.

Discussion: The presence of myocardial steatosis was significantly associated with regional ventricular dysfunction detected by tagged MRI in the presence of normal global function. To our knowledge, this is the first study showing increased myocardial lipid content leads to regional dysfunction of the myocardium.

Table 1, N=43	Low fat fraction (<1% lipid)	High fat fraction (≥1% lipid)	p value
Sex (F/M)	N=23, 12F/11M	N=20, 7F/13M	
End diastolic volume	152±33	154±34	0.42
End systolic volume	59±19	62±17	0.31
Stroke volume	91±3	92±19	0.44
Ejection fraction (%)	62±7	60±4.5	0.2
Cardiac output (l/min)	6.3±1.2	6.2±1.2	0.4
LV Mass (gm)	114±33	118±22	0.3
Systolic Ecc (%) (LAD)	-23.51±2.57	-20.98±2.63	0.005
Diastolic SR_E (1/sec) (RCA)	0.17±0.05	0.13±0.03	0.013

References: 1. Christoffersen C, et al. *Endocrinology* 2003;144(8):3483-3490. 2. Rijzewijk LJ, et al. *J Am Coll Cardiol* 2008;52(22):1793-1799. 3. van der Meer RW, et al. *Eur Heart J* 2008;29(12):1516-1522. 4. Szczepaniak LS, et al. *Circ. Res.* 2007;101:759-767. 5. Osman NF, et al. *Phys Med Biol* 2000; 45:1665-1682.