Association between epicardial adipose tissue volume and follow-up echocardiographic outcomes in patients with acute coronary syndrome

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Background: The epicardial adipose tissue (EAT) plays important physiological and pathological roles in the regulation of myocardial function. Among obese patients, the development of inflammatory EAT precipitates myocardial dysfunction and heart failure. Recent studies highlighted the possible divergent role of EAT in the pathophysiology of heart failure with reduced ejection fraction (HFrEF) and preserved ejection fraction (HFpEF). However, the effect of EAT on left ventricular remodelling among patients with acute coronary syndrome (ACS) remains unclear.

Purpose: According to the previous our study, patients with ACS had a higher EAT volume. This study evaluated the association of the EAT volume with left ventricular ejection fraction (LVEF) changes over time among patients with ACS.

Method: This prospective single-centre study selected 197 consecutive Japanese patients, who were hospitalised for ACS between June 2011 and November 2014. Among them, 143 (120 males, 23 females, mean age 64±12 years) patients underwent follow-up with serial echocardiograms. The EAT volume was measured using a multi-slice computed tomography in the hospital. Echocardiography was performed during their hospitalization and follow-up examinations for the next 3 (mean 3.2±1.8) years. They were divided into three groups according to the LVEF: HFrEF, heart failure with mildly reduced ejection fraction (HFmrEF), and HFpEF.

Results: No correlation existed between the EAT volume at the onset of ACS and LVEF during the follow-up (r=0.033, p=0.695). The peak creatine phosphokinase at ACS onset exhibited the strongest correlation with chronic ejection fraction (r=-0.512, p<0.01). In the chronic phase, the patients with HFrEF had the highest EAT volume (HFrEF, 134±38 ml; HFmrEF, 102±35 ml; and HFpEF, 120±51 ml; p=0.042). However, there was no correlation between the EAT volume and LVEF changes during the follow-up (r=-0.058, p=0.518) (Figure 1). The change in LVEF was not statistically significant among the three groups (HFrEF -4.8±11 %, HFmrEF -1.2±10 % and HFpEF 0.1±9 %, p=0.156).

Among patients with chronic HFmrEF and HFpEF, the EAT volume was positively correlated with the body mass index (r=0.373, p=0.029 and r=0.453, p<0.01, respectively). This correlation was not found in patients with chronic HFrEF (Figure 2).

Conclusion: There was no association between the EAT volume and improvements in LVEF after a follow-up period of 3 years among patients with ACS. Patients with chronic HFrEF had a significantly higher EAT volume despite having a non-obese body mass index. Non obese ACS patients with increased EAT may be prone to HFrEF in the chronic phase.

Figure 1

No correlation between ∆LVEF and EAT volume

Figure 2
EAT was increased in patients with HFrEF independent of BMI

Figure 2