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Echocardiography determined epicardial fat correlates with subclinical atherogenesis and serum nitric oxide

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Background: Intense metabolic activity occurring in the visceral adipose tissue results in the production and release of pro-inflammatory mediators such as nitric oxide (NO), which plays an important role in the early stages of subclinical atherogenesis. Epicardial Adipose Tissue (EAT) is a type of visceral fat, accessible by echocardiographic measurement with potential for the non-invasive identification of high atherogenic risk population. However, characterization of EAT as a marker of subclinical inflammation and atherogenic progression has been explored in a limited way.

Purpose: To evaluate the relation between echocardiography determined EAT thickness with serum NO concentration and atherogenic progression.

Methods: The study population was constituted by subjects more than 18 years old who attended at the Department of Echocardiography from a reference Medical Center. Classical cardiovascular risk factors were balanced within the study population and subjects with heart surgery, pericardial effusion or peripheral artery disease were excluded. Serum samples were collected and NO serum levels were measured immediately by colorimetric tests for nitrite (Greiss reaction). EAT thickness was determined by echocardiography with a long axis of the sternum. Flow Mediated Dilation (FMD) was determined by following standard procedure and Carotid Intima-Media Thickness (CIMT) ultrasound, according to the consensus of Mannheim.

Results: 60 patients of mean age 59 years old (33-86) were included. Hypertension was the cardiovascular risk most frequently found, followed by dyslipidemia and type 2 Diabetes Mellitus. Echocardiography determined EAT thickness was 6.6±2.42 mm. NO serum concentration was 35.6±8.13 uM/mL, FMD was 22.9±21.2% and CIMT was 0.87±0.24 mm (right carotid) and 0.91±0.252 mm (left carotid). EAT thickness significantly correlated with CIMT (rho=0.51; $p<0.01$). EAT thickness did not significantly correlate with serum NO (rho=-0.26; p=0.11) for the whole study population; nevertheless, the sub-population presenting classical cardiovascular risk factors showed a significant negative correlation between EAT thickness and serum NO (rho=-0.56; p=0.04).

Conclusions: Echocardiography determined EAT thickness positively correlated with subclinical atherogenesis markers such as CIMT and negatively correlated with serum NO concentration, selectively in subjects at higher cardiovascular risk. Echocardiography determined EAT thickness may be clinically useful to estimate advanced atherosclerosis, eliciting further stratification of higher cardio-ischemic risk based on NO availability.

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