

Association of Epicardial Adipose Tissue Thickness with Left Ventricular Systolic and Diastolic Dysfunction among Indian Phenotype

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Introduction: The metabolic effects of obesity are a result of increased adiposity in the ectopic sites. Ectopic fat deposition in the liver, skeletal muscle and visceral adipose tissue promotes insulin resistance and increases cardiovascular risk. Epicardial adipose tissue (EAT) is the visceral fat of the heart. Increased EAT is a measure of ectopic obesity. Increased epicardial ectopic fat is proposed to increase the incidence of cardiac dysfunction by release of inflammatory adipokines which act in a paracrine and endocrine fashion. Indian phenotype is different in many sense, despite of low body mass Index (BMI), they have high abdominal and visceral fat, high insulin resistance, low level of adiponectin, high CRP, low HDL and high small dense lipoprotein and triglyceride. It has been seen that metabolic complications are more pronounce in same BMI Indian phenotype compare to western counterpart. The purpose of this study was to detect the presence and extent of left ventricular dysfunction in relation with EAT among patients with Indian phenotype and its correlation with already known metabolic parameters i.e. fasting insulin, LDL cholesterol, triglyceride level, BMI, waist circumference, inflammatory markers like hS CRP and uric acid.

Method: Overweight and obesity was diagnosed based on BMI (Body MassIndex) which is weight in kg divided by height in m². All overweight and obese individuals were screened for obesity related complications and classified according to stage. Visceral obesity was diagnosed based on waist circumference (WC). The measurement of EAT thickness and left ventricular dysfunction was performed by transthoracic echocardiography. Biochemical and inflammatory parameters were assessed. Data thus obtained analysed by standard statistical software.

Results: Among 146 obese and overweight patients were assessed, the mean EAT thickness was 5.607 (SD 1.59). Patients having left ventricular diastolic dysfunction (LVDD) had a mean EAT thickness of 5.60 (SD 1.66) compare to 4.80 (SD 2.2) among normal persons (two-tailed P= 0.011, <0.05), which is statistically significant. Left ventricular Systolic dysfunction (LVSD) patients had mean EAT thickness of 6.33 (SD 0.94) compare to 5.35 (SD 1.58) in normal patients (two-tailed P=0.0001, <0.05), that is statistically significant. Moderate insulin resistance patients had a low mean EAT compare to sever insulin resistance patients. Mean EAT was poorly correlated with waist circumference and BMI in this study. EAT thickness was significantly correlated with LVDD and LVSD, even after adjusting for other cardiometabolic risk factors such as age, systolic blood pressure, BMI, blood glucose and LDL cholesterol and triglyceride.

Conclusion: Greater EAT is found in subjects with higher insulin resistance. EAT is significantly associated with LVDD and LVSD even after adjusting for other risk factors. Waist circumference, although a marker of visceral adiposity was poorly correlated with EAT as patients even with low WC had higher EAT same as to say for BMI. It can certainly be said that EAT is an individual risk factor for LVSD and LVDD after adjusting all known risk factors among patients with Indian phenotype.