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Epicardial fat in patients with metabolic syndrome

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ABSTRACT

Metabolic syndrome (MS) is bundling of abdominal obesity, insulin resistance, dyslipidemia, and elevated blood pressure. Epicardial fat (EPF) plays a role in cardiovascular diseases because of its anatomic and functional proximity to the myocardium and intense metabolic activity.
Aim: Assess EPF in patients with MS using echocardiography and find its relation to its parameters
Methods: 200 patients with criteria of MS according to National Cholesterol Education Program's Adult Treatment Panel III were subjected to clinical examination, echocardiography; where EPF (end-systolic thickness of echo free space in front of the free wall of right ventricle) was measured, abdominal ultrasonography for detection of non alcoholic fatty liver disease (NAFLD) and lipid and liver profiles. Fasting blood sugar (FBS), uric acid (UA) and hs-CRP were assessed as well. Patients were classified into two groups, group 1; 176 patients with EPF and group 2,28 patients with no EPF.
Results: 55% patients in group 1 were females vs 50% in group 2. Patients in group 1 had higher waist circumference (WC) 123 ± 7 vs 107 ± 2, $p < 0.05$, higher triglycerides level 240 ± 45 vs 181 ± 40, $p < 0.05$, higher filling pressure 16 ± 2 vs 11 ± 3, $p < 0.05$, and higher (ALT) 88 ± 17 vs 42 ± 7, $p < 0.05$. 28% of patients in group 1 had high hs-CRP, and 89% have NAFLD. EPF correlated significantly with WC, $r = 0.45$, NAFLD, $r = 0.48$, filling pressure, $r = 0.39$, and hs-CRP $r = 0.57$.
Conclusion: EPF as measured by echocardiography reflects visceral fat and severe MS. Echocardiography is an easy and reliable imaging technique to classify patients with high risk.

1. Introduction

Abdominal obesity is associated with insulin resistance on peripheral glucose and fatty acid utilization, often causing type 2 diabetes mellitus. The associated hyperinsulinemia, hyperglycemia, and adipocyte cytokines (adipokines) may also lead to vascular endothelial dysfunction, an abnormal lipid profile, hypertension, and vascular inflammation, all of which encourage the progress of atherosclerotic cardiovascular disease (CVD).[1–4] A similar profile can be seen in individuals with abdominal obesity who do not have an excess of total body weight.[5]

The metabolic syndrome (MS) has been accepted as a proinflammatory, prothrombotic state, associated with elevated levels of C-reactive protein, interleukin (IL)-6, and plasminogen activator inhibitor (PAI)-1[4] increasing the risk for subsequent CVD and type 2 diabetes.[6–8]

There are several definitions for (MS). The National Cholesterol Education Program (NCEP/ATP III) and International Diabetes Federation (IDF) definitions are the most widely used.[9–11]

Current ATP III criteria define the metabolic syndrome as the presence of any three of the following five traits:

- Abdominal obesity, defined as a waist circumference WC in men >102 cm (40 in.) and in women >88 cm (35 in.).
- Serum triglycerides (TG) ≥150 mg/dL (1.7 mmol/L) or drug treatment for elevated triglycerides.
- Serum HDL cholesterol <40 mg/dL (1 mmol/L) in men and <50 mg/dL (1.3 mmol/L) in women or drug treatment for low HDL-C.
- Blood pressure ≥130/85 mmHg or drug treatment for elevated blood pressure.
- Fasting plasma glucose (FPG) ≥100 mg/dL (5.6 mmol/L) or drug treatment for elevated blood glucose.

It had been suggested that visceral fat distribution plays an important part in the development of an unfavorable metabolic and cardiovascular risk profile.[12]

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Epicardial fat (EPF) is a metabolically active organ that secrets numerous bioactive substances, which may alter cardiac function. This small, visceral fat depot had been accepted as a rich source of free fatty acids and a number of bioactive molecules, such as adiponectin, resistin and inflammatory cytokines, which could lead to the coronary endothelial dysfunction. Furthermore, epicardial adipose mass might reflect intra-abdominal visceral fat. Epicardial adipose tissue is also clinically related to left ventricular mass and other features of the metabolic syndrome, such as concentrations of LDL cholesterol, fasting insulin and adiponectin, and arterial blood pressure.[12]

Epicardial and intra-abdominal fat evolve from brown adipose tissue during embryogenesis. In the adult heart, fully differentiated white adipose tissue can be commonly found in the atrioventricular and interventricular grooves extending to the apex. Minor foci of fat are also located subepicardially in the free walls of the atria and around the two appendages. As the amount of epicardial fat increases, it progressively fills the space between the ventricles, sometimes covering the entire epicardial surface. A small amount of adipose tissue also extends from the epicardial surface into the myocardium, often following the adventitia of the coronary artery branches. Overall, there appears to be a close functional and anatomic relationship between the adipose and muscular components of the heart. These components share the same coronary blood supply, and no structures resembling a fascia (as found on skeletal muscle) separate the adipose and myocardial layers.[13,14]

There is little evidence to suggest that the extent of epicardial fat is strongly related to overall adiposity. Marchington et al.[14] found no association between epicardial fat mass and the abundance of adipose tissue in other fat depots in a variety of wild and domesticated animals. This finding is in line with observations in humans from autopsy,[15] echocardiography[16] and MRI,[17] and suggests that epicardial fat is more closely related to visceral than total fat. Although autopsy examinations have revealed a relationship between epicardial fat and age,[15] echocardiographic studies have not.[16]

2. Aim of the work

Is to find the prevalence of epicardial fat in patients with MS and to find its relation to the other criteria of MS.

3. Methods

213 patients were recruited from Tanta university hospital cardiology department and Theodor Bilharz Research institute, Cairo, Egypt, from September 2015 till September 2016, they met the criteria of metabolic syndrome according to National Cholesterol Education Program's Adult Treatment Panel III (NCEP: ATPIII). Patients with primary liver disease were excluded as patients with positive markers of viral hepatitis, alcoholic liver disease and heart failure and Patients with left ventricular hypertrophy due to other cause than systemic hypertension as severe aortic stenosis and hypertrophic cardiomyopathy were excluded as well. The patients were subjected to full clinical examination, full echocardiographic examination and abdominal ultrasonography (was performed for detection of fatty liver and was graded as mild, moderate and severe, the method had been described before).[18] Lipid, liver profiles, fasting blood sugar (FBS), glycosylated hemoglobin (HbA1c), uric acid (UA) and high sensitive c-reactive protein (hs-CRP) were measured as well.

The echocardiographic examination was performed at rest by an experienced operator who was blinded to the clinical conditions of the participants, patients were examined in the left lateral decubitus position using a commercially available echocardiographic device (Vivid 7, GE Healthcare, Norway) with a 3.0 MHz transducer to measure left ventricular dimensions and function, left atrial dimension (LAD), left ventricular mass index (LVMI), diastolic function (graded as normal 0 or 1, 2, 3, and 4), left ventricular filling pressure was measured as the ratio (E/e) as E is the trans-mitral Doppler E wave and e is the tissue Doppler of the mitral ring e wave. Examination was performed according to the

Table 1
baseline criteria of the patients in the 2 groups.

	Group 1 Range, mean ± SD	Group 2 Range, mean ± SD	p value	Significance
Number	172 (86%)	28 (14%)	<0.05	S
Age in years	43–59, 58 ± 4	40–60, 53 ± 7	0.91	NS
Women	55%	50%	0.98	NS
Smoking	43%	39%	0.56	NS
CAD	24%	22%	0.80	NS
Valvular heart disease	19%	17%	0.74	NS
Hypertension	35%	32%	0.81	NS
BMI, kg/m^2	29–40, 37 ± 3	26–34, 30 ± 4	<0.05	S
WC in cm	102–141, 123 ± 7	98–114, 107 ± 2	<0.05	S
SBP, mmHg	131–160, 156 ± 14	130–150, 147 ± 7	0.26	NS
DBP, mmHg	98–116, 114 ± 6	90–100, 98 ± 8	<0.05	S
Diabetes	47%	31%	<0.05	S
Dyslipidemia	37%	26%	<0.05	S
HbA1c%	6.5–10, 8.9 ± 0.9	5.3–9, 7 ± 0.3	<0.05	s
FBS, mg%	120–189, 154 ± 15	90–130, 123 ± 21	<0.05	S
TC, mg%	220-289, 280 ± 22	200–270, 265 ± 41	0.54	NS
TG, mg%	180–250, 240 ± 45	160–190, 181 ± 40	<0.05	S
LDL, mg%	98–134, 125 ± 27	100–143, 132 ± 17	0.87	NS
HDL, mg%	17–25, 23 ± 7	22-35, 30 ± 7	<0.05	S
UA, mg%	6–9, 8.9 ± 3	4.5–8, 6 ± 3	<0.05	S
ALT in units	66–90, 88 ± 17	33–44, 42 ± 7	<0.05	S
AST in units	34–55, 50 ± 9	22–49, 42 ± 2	0.12	NS
High sensitive CRP	28%	ZERO		
EPF	100%	ZERO		
NAFLD	89%	3%	<0.05	S

SD = standard deviation, WC = waist circumference, SBP = systolic blood pressure, DBP = diastolic blood pressure, CAD = coronary heart disease, FBS = fasting blood sugar, TC = total cholesterol, TG = triglycerides, LDL = low density lipoprotein, HDL = high density lipoprotein, UA = uric acid, ALT = alanine transaminase, AST = aspartate transaminase, CRP = c-reactive protein, EPF = epicardial fat, NAFLD = non alcoholic fatty liver disease, S = significant, NS = non significant.

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