Research Journal of Medical Sciences 10 (6): 557-564, 2016
ISSN: 1815-9346

Assessment of Epicardial Adipose Tissue Thickness in Patient with Coronary Artery Disease

Mohammed Noori Al-Dujaili, Laith Abdulhussein Al-dabbagh and Mohammad Nassar Karkosh
College of Medical, University of Kufa, Najaf, Iraq

Abstract: One of the most killer and common silent disease worldwide is Coronary Artery Disease (CAD), contributing high morbidity and mortality. The approximate of Epicardial Adipose Tissue (EAT) to the coronary arteries plays an important role within the pathogenesis of CAD previous studies proved that coronary artery disease had been recommended to be directly related to the Epicardial adipose tissue thickness. The purpose of this study is to assess the accuracy of epicardial adipose tissues thickness in predicting coronary artery stenosis. A cross-sectional study carried out at Al-Sader teaching medical city during the period from the first of January to the 15th of October 2014 included 80 with coronary artery diseases underwent cardiac catheterization regardless their age or gender. Patients with valvular hear diseases or poor echocardiographic window were excluded. ECG, the Echocardiographic examination were performed in all patients. Three cut-off points of thickness were used, according to the measurement point; on RV Apex = 8 mm, RV free wall those = 10 mm and right ventricle outlet tract = 13 mm which are considered abnormal accumulation of EAT. The data were analyzed using SPSS version 21 and appropriate statistical tests were applied. The mean of the patients age was 52.1±9.8 (range: 25-77) years, more than two thirds (70%) of the patients were males. Smokers represented (25%), History of hypertension (17.5%), diabetes mellitus(10%). The mean EAT thickness at RV apex was 3.62±0.46 mm at RV free wall was 3.79±0.42 and at the RV outlet tract it was 3.9±0.45 mm, the mean EAT thickness was higher in younger patients and decreased with the advancing age, male gender, smoking, history of hypertension and history of diabetes are associated with larger mean EAT thickness. Because of this EAT thickness is a great predictor of CAD with sufficient sensitivity, specificity and accuracy. Using the echocardiographic assessment of Epicardial adipose tissue thickness can be a great assistance for clinicians to detect the people at risk.

Key words: Adipose tissue, younger patients, diabetes, hypertension, history

INTRODUCTION

The other term of Coronary Artery Disease (CAD) is Coronary Heart Dsease (CHD) which is probably the most killers and common silent diseases contributing high morbidity and mortality worldwide. The CAD is the significant cause of death within adults in the USA; responsible approximately one-third of almost all dead people, mainly, above 35 year old (Hadaegh *et al*., 2009). The CAD occurs because of insufficient oxygen in addition to nutrition supply resulting injury regarding the heart muscle, as a result of the blockade or narrowing of coronary artery which can cause improper blood flow for the heart part. The coronary disease indicated by first of all its latent development exactly where it develops for an advanced stage earlier to be able to any symptoms notice by the patient. The second thing is the symptoms attributed for heart diseases are limited and also mimic quite a few different pathologies. In Western Europe and North America, mortality caused by CAD has been effectively and successfully decreased in the past years, by the improving in treatment; on the other hand, there is an enhancement in mortality rates around Eastern Europe and Asian countries (Fakhrzadeh *et al*., 2008).

Coronary Artery Disease is a multifactorial illness, various factors may possibly stimulated its development; genetic and/or environmental factors are the broadly postulated. Using tobacco, Diabetes Mellitus (DM) and hypertension are the most typical attributable risk factors (Stocker and Keaney, 2004). The interaction within these factors identify if the disease can develop besides its severity (Freitas *et al*., 2008).

Various multiple biochemical processes contributing in CAD development, for example inflammatory response, apolipoprotein metabolism and lipid, blood pressure regulation, platelets and endothelial function, thrombosis and fibrinolysis (Scheuner, 2003).

Prior studies exhibited that biochemical markers, hypertension, dyslipidaemia, family history and DM are

Corresponding Author: Mohammed Noori Al-Dujaili, College of Medical, University of Kufa, Najaf, Iraq

considerably more frequent within CAD patients in comparison with healthy population (Freitas *et al*., 2008).

Risk factors of CAD

The Controllable risk factors of CAD

Cigarette smoking: Smoking and second-hand using cigarettes tobaccos are major risk reasons of CAD advancement in the majority of communities. Various studies strongly related the smoking cigarettes to heart diseases.

Hypertension: This high blood pressure, could lead to thickening, hardening and the coronary arteries narrowing, resulting in impaired blood flow (Gierach *et al*., 2006).

Serum hypercholesterolemia: High serum cholesterol levels regarded as the main causative and the essential element regarding the heart diseases (Nelson, 2013).

Diabetes Mellitus (DM): DM and Hyperglycemia are regarded as the most critical risk factors associated with CAD. Patients have DM are about 3-5 fold higher than non-diabetics for getting coronary atherosclerosis (Kalofoutis *et al*., 2007; Laakso, 2010; Yeboah *et al*., 2014).

Obesity or overweight: Globally, there is a worrying increase in the occurrence of obesity. An excess in general body weight through an abnormal accumulation regarding body fat responsible pertaining to development of several diseases. The association of obesity having CAD is parallel in addition to inter-correlated with certain risk elements of CAD for instance hypertension, DM, low HDL cholesterol. These days' considerable attention and consternation had provided to the association between cardiovascular disease and obesity (Bibbins-Domingo *et al*., 2007; Stocker and Keaney, 2004).

Physical inactivity: Physically inactive people are more liable to have CAD, therefore that the actual physical inactivity is already been proven as more essential risk factor. Thus, the USA Health and Human Services Department stated the physical actions as first priority with regard to the newly revealed objectives (Rafaee and Hazzaa, 2001).

Stress: This particular Controllable risk factor has two opposite influences, it has a great motivation and more productivity impact if it was mild, however, strong stress also has dangerous and harmful {effect|influence}. Dangerous situations, crowds and noise contributed the serious stress status. Marital problems, demanding jobs and family troubles triggered a serious stress. The chronic and recurrent exposure to stress might harm the arteries as well as could worsen some other risk elements (Holmes *et al*., 2006).

The uncontrollable risk factors of CAD

Age: Age is probably the most vital and important risk factors that have a close association with the heart diseases incidence. The standard risk rate to obtain cardiovascular disease around people aged that between 30-34 years is around 3%, increases to 49% in people aged 90 years old. Generally, CAD developed in females about 10 years right after men in addition to the average risk can be about 32% through aged 90 years. To ensure, it had been recorded that CAD is an illness of advancing age group and greater risk a with occurrence of genetic predisposition (Savji *et al*., 2013; Scheuner, 2003).

Family history: Presence with family history of earlier heart disease has been observed to be related to the development of early CAD and will be attributed to good genetic factors that are more probable to have an impact on young instead of old people (Freitas *et al*., 2008).

Gender: Males are more liable to have excess chance for cardiovascular illness compared with women in the same age. The risk at menopause in women rises, caused by lack of the protecting role associated with estrogens hormone. Nevertheless, this has been complicated to substantiated, wherever the estrogen treatment cannot prove to decrease the prevalence of cardiovascular illness in women (Tamaki *et al*., 2006; Roeters van Lennep *et al*., 2002).

Epicardial adipose tissue and CAD: It is widely recognized that large visceral adipose amount related to the cardiovascular diseases development. Epicardial adipose tissue is one of visceral adipose tissue part which is associated with the heart as well as the coronary arteries placed between the pericardium and heart (Iacobellis and Willens, 2009). The EAT nearness towards the coronary arteries can be played an essential role in the CAD pathogenesis. Autopsy and imaging and studies evaluated the quantity and location of EAT in both females and males, without or with CAD. EAT is actually located close to epicardial coronary vessels on the interventricular and atrioventricular grooves in another locations for EAT tend to be about the free wall along with the right ventricle apex as well as about the atria and this can also propagate into the myocardium. However, the lateral right of ventricular wall showed the main site for EAT (as shown in Fig. 1) (Iacobellis and Willens, 2009;

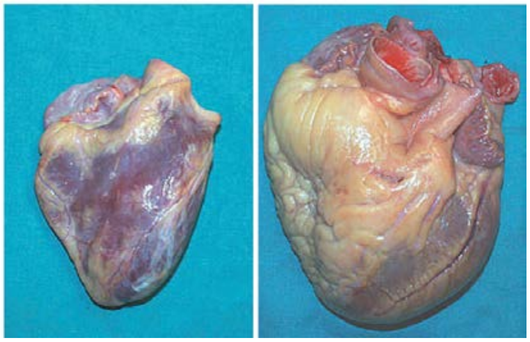

Fig. 1: Macroscopic appearance of EAT in the anterior site of a normal (210 g) and a hypertrophic (900 g) heart, respectively. EAT correlates with the weight of the heart Adopted from Iacobellis *et al*. (2005)

Rabkin, 2007; Sacks and Fain, 2007). It has been recommended that coronary atherosclerosis advancement influenced by an endocrine in addition to paracrine activity regarding EAT besides the pro-inflammatory secretion and also anti-inflammatory cytokines, chemokines for example interleukin-6 as well as tumor necrosis element alpha. Furthermore, bioactive molecules that may affect the cardiac function tend to be developed through the epicardial fat that considered like a metabolically effective organ (Fain *et al*., 2010; Iacobellis *et al*., 2005; Marcus *et al*., 2010; Mazurek *et al*., 2003).

Coronary artery disease has been recommended to be directly related to the EAT thickness, coming from other viewpoint, EAT thickness related to hypertrophy, visceral fat left and ventricular mass and in addition it is indicated to be correlated along with the CAD severity (Corradi *et al*., 2004; Iacobellis and Bianco, 2011; Wong *et al*., 2011).

Purpose of the study: The aim of this study is to assess the significance of Epicardial Adipose Tissue (EAT) thickness in predicting coronary artery disease.

MATERIALS AND METHODS

Study design, setting and time: A cross-sectional study conducted at Al-Sader teaching medical city during the period from the first of January to the 15th of October 2014.

Patients: The total of 80 patients all with proved diagnosis of coronary artery diseases were registered in this study.

Inclusion criteria:

- Patients with suspected ischemic heart disease underwent cardiac catheterization.
- Patients were enrolled regardless their age or gender

Exclusion criteria: Patients were excluded from this study if they had one or more of the following:

- Patients with valvular hear diseases.
- Patients with poor echocardiographic window.
- Patient did not desire to participate further in the study

Data collection: Data were collected by using data collecting sheet and the data were obtained by detailed medical history, complete physical examination and lab investigation. In all patients, ECG was performed, the Echocardiographic examination was performed and the EAT thickness was assessed.

Echocardiography protocol

Evaluation of epicardial adipose tissue: The Echocardiographic examination was performed in all patients with Two-dimensional transthoracic echocardiography Philips iE33 with 3.5 MHz transducer in the remaining lateral decubitus position. Measurements were conducted by the same cardiologist and the same system in all patients to get rid any possible variations in the Echocardiographic examination (inter observer or system variation) and to get excellent reproducibility of echocardiographic measurements. Epicardial adipose tissue has been determined as echo-free area in the layers of pericardial for this two-dimensional echocardiography in addition to its thickness has been measured perpendicularly with the right ventricle free wall at the ending of systole, frequent more than three cardiac cycles. With the standard of two echocardiographic windows, short-axis and parasternal long-axis were utilized. The EAT thickness has been measured on right ventricle free wall, with right ventricle apex (measured at end diastole through parasternal long axis view of 3 cardiac cycle) and right ventricle outlet (measured at end diastole through parasternal short axis).

EAT thickness cut-off points: The cut off points for abnormal accumulation of EAT was different according to the point of measurements as abnormal EAT accumulation was considered accordingly:

- On RV Apex = 8 mm
- On RV free wall those = 10 mm
- On right ventricle outlet tract = 13 mm

Ethical issue:

- This study protocol was authorized by the internal medicine department, Medical college-University of Kufa
- Verbal consent of the patient was obtained
- Patients names or identification information that directly refer to the patients were hided and replaced with codes and serial numbers
- Data were used for purpose of this study and didn't disclose to non-authorized individuals

Statistical analysis: Data of the patients were analyzed by using the Statistical Package for Social Sciences (SPSS) Version 21. Data presented as mean, Standard Error of mean (SE), Standard Deviation (SD), frequencies (number of patients) and proportions (%). Non-parametric statistical tests were used; Mann Whitney test was used to compare two means and Kruskal-Wallis H test was utilized to compare more than two means of EAT thickness at different points of measurements. Chi square and Fisher's exact tests are utilized, alternatively to compare categorical variables. Level of significance (p-value) of = 0.05 indicated a significant difference or correlation. Results and finding were presented in tables and figures accordingly.

RESULTS

Patients' demographical characteristics: There were 80 patients enrolled in this study all were underwent cardiac catheterization and coronary angiography with a mean age of 52.1±9.8 (range: 25-77) year, furthermore by distribution of the patients according to 4 age intervals, it had been found that 7 patients (8.8%) aged <40 year, 22 (27.5%) aged 40-49 year, 32 patients (40%) aged 50-59 year and 19 patients (23.8%) aged = 60 year. More than two thirds, 56/80 (70%) of the patients were males and the remaining 24/80 (30%) were females with a male to female ratio of 2.3:1 (Table 1). Regarding the smoking habit, smokers were 20 represented (25%) of the studied group compared to 60 non-smoker patients. History of hypertension (HT) was reported in 14 patients (17.5%), Diabetes Mellitus (DM) in 8 patients (10%) and 10 patients (12.5%) had history of both diseases.

Abnormal EAT deposition: According to the above mentioned different cut-off values of the three points of measurements of EAT, the total patients with abnormal EAT deposition was 46 patients represented 57.5% of the studied group which shows in Table 2.

Epicardial Adipose tissue Thickness (EAT): The mean EAT thickness at RV apex was 3.62±0.46 (range: 0.30-12) mm at RV free wall 3.79±0.42 (range: 0.44-12) and at the RV outlet tract it was 3.9±0.45 (range: 0.10-12.7) mm.

Table 1: Demographic characteristics of the studied group

Characteristics	No.	(%)
Age groups		
<40	7	8.8
40-49	22	27.5
50-59	32	40.0
≥60	19	23.8
Sex		
Male	55	69.6
Female	24	30.4
HT	14	17.5
DM	8	10.0
HT and DM	10	12.5
Smoker	19	23.8

Table 2: Rquency and distribution of abnormal and normal EAT deposition of the studied group

Group	No. of patients	Percent	Valid percent
Abnormal	46	57.5	57.5
Normal	34	42.5	42.5
Total	80	100.0	100.0

Table 3: Mean EAT thickness at different points of measurement

Point	Mean±SE	Min.-Max.
RV apex	3.62±0.46	0.30-12.0
RV free wall	3.79±0.42	0.44-12.0
RV outlet tract	3.90±0.45	0.10-17.2
Average	3.77±0.43	0.44-11.0

NS: Not Significant

Table 4: Comparison of EAT thickness measured at RV Apex according to demographic parameters of the patients

Demographic parameter	No. of patients	Mean EAT (mm) RV Apex	p-values
Age (years)			
<40	7	5.34	0.041
40-49	22	4.22	
50-59	32	3.93	
≥60	19	1.77	
Sex			
Male	56	4.97	0.027
Female	24	3.04	
Smoking			
Smoker	20	5.29	0.045
Non-smoker	20	3.14	
Hypertension			
Yes	24	4.46	0.008
No	56	1.67	
Diabetes			
Yes	18	4.40	0.015
No	62	0.95	

Correlation of mean EAT thickness with demographic variables: Table 3-5 demonstrate the correlation of EAT thickness at different points (RV apex, RV free wall and RV outlet) with different demographic variables

Age: It had been found that in all points of measurements, the mean EAT was higher in younger patients, below 40 year and the EAT thickness decreased with the

Table 5: Comparison of EAT thickness measured at RV free wall according to demographic parameters of the patients

Demographic parameter	No. of patients	Mean EAT (mm) RV free wall	p-values
Age (years)			
<40	7	5.37	0.040
40-49	22	4.43	
50-59	32	3.96	
≥60	19	2.23	
Sex			
Male	56	5.92	0.001
Female	24	2.89	
Smoking			
Smoker	20	5.51	0.034
Non-smoker	20	3.30	
Hypertension			
Yes	24	4.66	0.003
No	56	1.79	
Diabetes			
Yes	18	4.64	0.001
No	62	0.89	

advancing age; at RV apex point the mean EAT of the patients <40 year old was 5.34 mm compared to 4.22 in those their ages between 40-49 year, 3.93 in those ages 50-59 year old and the lowest value in those aged = 60 year. Similar trends were observed in the other two points of measurements, however the difference was statistically significant in RV Apex and RV free wall measurement points ($p<0.05$) and insignificant in RV outlet point.

Sex: The comparison in mean EAT measured at different points according to the sex, revealed that males had significantly larger mean EAT thickness than females, at RV apex (4.97 vs. 3.04 mm, p = 0.027), at RV free wall (5.92 vs. 2.89 mm, p = 0.001) and at RV outlet tract (5.55 vs. 3.01, p = 0.006), respectively.

Smoking: Smokers had significantly thicker EAT than non-smokers in all points of measurements of EAT, $p<0.05$.

Hypertension and diabetes: Patients with hypertension, diabetes or both had significantly larger mean EAT thickness $p<0.05$.

EAT thickness cut-off points and categorization: According to different cut-off points the EAT thickness was categorized. On RV Apex, patients with = 8 mm (abnormal deposition of EAT) were 13 represented (16.3%), on RV free wall those with = 10 mm (abnormal deposition) were 33 patients (41.3%) while on RVOT those with = 13 mm EAT were 38 patients (47.5%) which shows in Table 6 and 7.

Table 6: Comparison of EAT thickness measured at RV outlet according to demographic parameters of the patients

Demographic parameter	No. of patients	Mean EAT (mm) RV outlet tract	p-values
Age (years)			
<40	7	4.93	0.5
40-49	22	4.59	
50-59	32	4.32	
≥60	19	2.02	
Sex			
Male	56	5.55	0.006
Female	24	3.01	
Smoking			
Smoker	20	5.89	0.016
Non-smoker	20	3.32	
Hypertension			
Yes	24	4.73	0.022
No	56	1.96	
Diabetes			
Yes	18	4.72	0.020
No	62	1.09	

p-value = 0.05 is statistically significant

Table 7: Distribution of EAT thickness at different points of measurements according to cut-off points

Point of measurements	No. of patients	(%)	p-values
EAT on RV Apex			
<8 mm	67	83.8	<0.001
≥8 mm	13	16.3	
EAT on RV free wall			
<10 mm	47	58.8	
≥10 mm	33	41.3	
EAT on RVOT			
<13 mm	42	52.5	
≥13 mm	38	47.5	

DISCUSSION

The amount of EAT close to the heart was proven as an important independent estimator of coronary artery diseases. Many studies that concerned with this subject proved that EAT thickness is a good indicator and independent predictor of CAD of great interest than pericardial adipose tissue (Yanez *et al*., 2014). The current cross sectional study has been conducted to assess the accuracy of EAT thickness as predictor regarding coronary artery stenosis for this purpose, measurements of epicardial fat distance were compared in 80 patients who underwent coronary angiography at three points of measurements. The mean age of the studied group was 52.1±9.8 (range: 25-77) years and about two thirds (63.8%) of them aged 50 years or more. Males were the dominant, represented 70% of the studied group with a male to female ratio of 2.3:1; these findings indicated the higher prevalence of CAD among males and advancing age.

Although, smoking is a main risk factor of CAD, the current study reported that smokers were 25% of the studied group and majority of the patients were non-smokers, nonetheless, this proportion is still higher than that reported in the general Iraqi population in 2006 where the smoking habit reported in 21.9% according to the national survey for risk factors of chronic diseases. Different studies strongly correlate cigarette smoking to heart diseases.

History of hypertension was reported in 17.5%, diabetes mellitus 10% of the patients while in 10 patients (12.5%) both diseases were co-exist. Previous studies concluded that uncontrolled hypertension and DM are deemed of the most essential risk factors in CAD (Kalofoutis *et al*., 2007; Gierach *et al*., 2006; Laakso, 2010).

The present study found that the mean EAT thickness at RV apex was 3.62±0.46 (range: 0.30-12) mm at RV free wall 3.79±0.42 (range: 0.44-12) and at the RV outlet tract it was 3.9±0.45 (range: 0.10-17.2) mm, however, the relative differences in the measurements at different points of measurement were statistically insignificant, p>0.05. Furthermore, by using different cut off of EAT thickness it had been found that on RV Apex, 16.3% of the patients had abnormal accumulation of EAT with = 8 mm EAT thickness, on RV free wall, 41.3% with abnormal accumulation of EAT of = 10 mm thickness (cut off point for this point is 10 mm) and on the RVOT where, cut-off point of 13 mm was used as indicator of abnormal accumulation of EAT those with = 13 mm were 47.5% of the patients, these findings indicated the variation in the distribution of EAT among the patients and even in the same patient, however, the differences were statistically insignificant between the three points of measurements. For the purpose of more precise comparison, the current study depended the mean EAT thickness rather than categories in all comparison and assessment of correlation between EAT thickness and other variables. The current study found that EAT thickness was significantly reduced with advancing age indicated an inverse correlation between the age of the patient and EAT thickness in contrast, our findings disagreed that of (Yanez *et al*., 2014) who found a positive significant association with the age, the disagreement and inverse correlation in our study with that of Yanez-Rivera might attribute to the differences in age of the patients included in both studies where in our study younger and older patients (age range 25-77) years were included while Yañez-Rivera study (Yanez *et al*., 2014) included older patients only (>50 years) in addition older individuals in Iraqi population seem to be thinner than younger individuals.

The current study identified that EAT thickness is significantly larger in males than females, similarly, Dagvasumberel *et al*. (2012) concluded in 2012 that EAT thickness in men larger than that of women and was significantly associated with CAD in men. Smoker patients in the present study had significantly thicker EAT than non-smokers, this finding is constant with that by Monti *et al*. (2014) who suggested that cigarette smoking is actually an independent predictor to increase epicardial fat volume. Also, the present study found that patients with history of hypertension had larger mean EAT thickness than those without and also it was thicker (larger mean EAT thickness) in those with history of diabetes than non-diabetic, similar to these findings, many previous studies had reported such associations; Dicker *et al*. (2013) reported in 2013 that hypertensive patients got greater EAT thickness than non-hypertensive and the thickness of EAT =2.4 mm anticipates the occurrence of hypertension and related to a higher calcium score as well as presence of CAD.

Akbas *et al*. (2014) in their control study that has conducted in Turkey in 2014 found that EAT measurements have been significantly greater in diabetic patients if compared with control subjects. The mean EAT thickness at RV apex was 3.62±0.46 (range: 0.30-12) mm, at RV free wall 3.79±0.42 (range: 0.44-12) and at the RV outlet tract it was 3.9±0.45 (range: 0.10-12.7) mm with no significant difference for the measurements at these three points, p-value >0.05. These findings were close to that of a study was conducted by Ahn *et al*. (2008) who revealed a mean thickness of epicardial fat of 4.3 mm in patients with unstable angina and our findings were lower than that reported by Toufan *et al*. (2012) who reported an overall mean EAT thickness of 5.36 mm, however, these differences might attributed to the differences in the patients included in that study in addition to ethnic variation. Nonetheless Ahn *et al*. (2008) studied the relationship between the EAT thickness upon the free wall regarding the CAD and RV in patients who have conventional coronary angiography because of chest pain and identified a positive relationship between EAT thickness along with the significant coronary stenosis presence (luminal narrow -50%) along with the amount of coronary arteries through significant stenosis. In contrast, a study by Chaowalit *et al*. (2006) throughout 139 patients that were referred to get conventional coronary angiography have not indicate a significant correlation in between EAT thickness with atherosclerotic coronary segments. The current study used different cut-off point for EAT thickness at different points of measurements to differentiate between normal and abnormal EAT deposition, according to these cut-off points the overall prevalence of abnormal EAT deposition was 57.5% which indicated a significant higher prevalence of abnormal EAT deposition among patients with CAD and that EAT thickness could be a good predictor for CAD. Different previous studies concluded, approximately, similar findings and most of these studies considered EAT thickness as an important and good predictor of CAD, independent of the age, gender or ethnicity. Other studies used EAT thickness for evaluation and prediction of severity of stenosis and outcomes associated with CAD (Demircelik *et al*., 2014).

Other study was conducted by Toufan *et al*. (2012) found that RV EAT= 10 mm, RV apex EAT = 8 mm and RVOT EAT = 13 mm could predict coronary stenosis = 50% and Acute Coronary Syndrome (ACS). On the other hand, Kaya *et al*. (2013) and his colleagues found that even lower cut-off point of EAT thickness of = 5.8 mm could predict the presence of CAD with 77% sensitivity and 70% specificity and that an echocardiographic EAT assessment is independently associated with the presence of CAD. Another study was conducted by Bachar *et al*. (2012) found that lower cut-off point of EAT thickness of 2.4 mm is the optimal cutoff for prediction of presence of significant CAD.

CONCLUSION

The EAT thickness was higher in younger patients, less than 40 years and the EAT thickness decreased with the advancing age.

Male gender, smoking, history of hypertension and history of diabetes are associated with larger mean EAT thickness. Epicardial Adipose Tissue (EAT) thickness is a good predictor for coronary artery diseases.

LIMITATIONS

Some limitations were interfering with this work; the single-center study and restriction in time affected the sample size.

RECOMMENDATIONS

- Using the echocardiographic assessment of EAT thickness as a non-invasive method, can be a good assistance to clinicians pertaining to detect those patients at risk in addition to help them to experience additional evaluations with other procedures
- Subsequently it would be able to use EAT in predicting CAD incidence, its degree and the probability of deterioration of the patient status
- Further studies with bigger sample size and multi-center are recommended to promote the power of the results and enabling the use of the findings clinically
- Further studies to detect relation between the thickness of EAT and the number of the affected coronary arteries.

REFERENCES

Ahn, S.G., H.S. Lim, D.Y. Joe, S.J. Kang and B.J. Choi *et al*., 2008. Relationship of epicardial adipose tissue by echocardiography to coronary artery disease. Heart, 94: 7-7.

Akbas, E.M., H. Hamur, L. Demirtas, E.M. Bakirci and A. Ozcicek *et al*., 2014. Predictors of epicardial adipose tissue in patients with type 2 diabetes mellitus. Diabetology Metab. Syndrome, 6: 55-62.

Bachar, G.N., D. Dicker, R. Kornowski and E. Atar, 2012. Epicardial adipose tissue as a predictor of coronary artery disease in asymptomatic subjects. Am. J. Cardiol., 110: 534-538.

Bibbins-Domingo, K., P. Coxson, M.J. Pletcher, J. Lightwood and L. Goldman, 2007. Adolescent overweight and future adult coronary heart disease. N. Eng. J. Med., 357: 2371-2379.

Chaowalit, N., V.K. Somers, P.A. Pellikka, C.S. Rihal and J.F. Lopez, 2006. Subepicardial adipose tissue and the presence and severity of coronary artery disease. Atherosclerosis, 186: 354-359.

Corradi, D., R. Maestri, S. Callegari, P. Pastori and M. Goldoni *et al*., 2004. The ventricular epicardial fat is related to the myocardial mass in normal, ischemic and hypertrophic hearts. Cardiovasc. Pathol., 13: 313-316.

Dagvasumberel, M., M. Shimabukuro, T. Nishiuchi, J. Ueno and S. Takao *et al*., 2012. Gender disparities in the association between epicardial adipose tissue volume and coronary atherosclerosis: A 3-dimensional cardiac computed tomography imaging study in Japanese subjects. Cardiovasc. Diabetology, 11: 106-106.

Demircelik, M.B., O.C. Yilmaz, O.M. Gurel, Y. Selcoki and I.A. Atar *et al*., 2014. Epicardial adipose tissue and pericoronary fat thickness measured with 64-multidetector computed tomography: potential predictors of the severity of coronary artery disease. Clin., 69: 388-392.

Dicker, D., E. Atar, R. Kornowski and G.N. Bachar, 2013. Increased epicardial adipose tissue thickness as a predictor for hypertension: A cross-sectional observational study. J. Clin. Hypertension, 15: 893-898.

Fain, J.N., H.S. Sacks, S.W. Bahouth, D.S. Tichansky and A.K. Madan *et al*., 2010. Human epicardial adipokine messenger RNAs: Comparisons of their expression in substernal, subcutaneous and omental fat. Metab., 59: 1379-1386.

Fakhrzadeh, H., F. Bandarian, H. Adibi, T. Samavat, H. Malekafzali, E. Hodjatzadeh and B. Larijani, 2008. Coronary heart disease and associated risk factors in Qazvin: A population-based study. Eastern Mediterranean Health J., 14: 33-41.

Freitas, A.I., I. Mendonca, M. Brion, M.M. Sequeira, R.P. Reis, A. Carracedo and A. Brehm, 2008. RAS gene polymorphisms, classical risk factors and the advent of coronary artery disease in the Portuguese population. BMC Cardiovasc. Disord., Vol. 8. 10.1186/1471-2261-8-15

Gierach, G.L., B.D. Johnson, C.N.B. Merz, S.F. Kelsey and V. Bittner *et al*., 2006. Hypertension, menopause and coronary artery disease risk in the Women's Ischemia Syndrome Evaluation (WISE) study. J. Am. Coll. Cardiol., 47: 50-58.

Hadaegh, F., H. Harati, A. Ghanbarian and F. Azizi, 2009. Prevalence of coronary heart disease among Tehran adults: Tehran lipid and glucose study. Eastern Mediterranean Health J., 15: 157-166.

Holmes, S.D., D.S. Krantz, H. Rogers, J. Gottdiener and R.J. Contrada, 2006. Mental stress and coronary artery disease: A multidisciplinary guide. Progress Cardiovasc. Dis., 49: 106-122.

Iacobellis, G. and A.C. Bianco, 2011. Epicardial adipose tissue: Emerging physiological, pathophysiological and clinical features. Trends Endocrinol. Metab., 22: 450-457.

Iacobellis, G. and H.J. Willens, 2009. Echocardiographic epicardial fat: A review of research and clinical applications. J. Am. Soc. Echocardiography, 22: 1311-1319.

Iacobellis, G., D. Corradi and A.M. Sharma, 2005. Epicardial adipose tissue: Anatomic, biomolecular and clinical relationships with the heart. Nat. Clin. Pract. Cardiovasc. Med., 2: 536-543.

Kalofoutis, M.D.A., C. Kalofoutis, C. Piperi, A. Kalofoutis and F. Harris *et al*., 2007. Type II diabetes mellitus and cardiovascular risk factors: Current therapeutic approaches. Exp. Clin. Cardiol., 12: 17-28.

Kaya, H., F. Ertas, M. Oylumlu, M.Z. Bilik and A. Yýldýz *et al*., 2013. Relation of epicardial fat thickness and brachial flow-mediated vasodilation with coronary artery disease. J. Cardiol., 62: 343-347.

Laakso, M., 2010. Cardiovascular disease in type 2 diabetes from population to man to mechanisms: The Kelly West Award Lecture 2008. Diabetes Care, 33: 442-449.

Marcus, G.M., L.M. Smith, K. Ordovas, M.M. Scheinman and A. Kim *et al*., 2010. Intracardiac and extracardiac markers of inflammation during atrial fibrillation. Heart Rhythm, 7: 149-154.

Mazurek, T., L. Zhang, A. Zalewski, J.D. Mannion and J.T. Diehl *et al*., 2003. Human epicardial adipose tissue is a source of inflammatory mediators. Circulation, 108: 2460-2466.

Monti, M., A. Monti, G. Murdolo, D.P. Renzi and M.R. Pirro *et al*., 2014. Correlation between epicardial fat and cigarette smoking: CT imaging in patients with metabolic syndrome. Scand. Cardiovasc. J., 48: 317-322.

Nelson, R.H., 2013. Hyperlipidemia as a risk factor for cardiovascular disease. Primary Care: Clin. Office Practice, 40: 195-211.

Rabkin, S.W., 2007. Epicardial fat: properties function and relationship to obesity. Obesity Rev., 8: 253-261.

Rafaee, S.A.A. and H.M.A. Hazzaa, 2001. Physical activity profile of adult males in Riyadh City. Saudi Med. J., 22: 784-789.

Roeters van Lennep, J.E., H.T. Westerveld, D.W. Erkelens and E.E. van der Wall, 2002. Risk factors for coronary heart disease: Implications of gender. Cardiovasc. Res., 53: 538-549.

Sacks, H.S. and J.N. Fain, 2007. Human epicardial adipose tissue: A review. Am. Heart J., 153: 907-917.

Savji, N., C.B. Rockman, A.H. Skolnick, Y. Guo and M.A. Adelman *et al*., 2013. Association between advanced age and vascular disease in different arterial territories: A population database of over 3.6 million subjects. J. Am. Coll. Cardiol., 61: 1736-1743.

Scheuner, M.T., 2003. Genetic evaluation for coronary artery disease. Genet. Med., 5: 269-285.

Stocker, R. and J.F. Keaney Jr., 2004. Role of oxidative modifications in atherosclerosis. Physiol. Rev., 84: 1381-1478.

Tamaki, J., H. Ueshima, T. Hayakawa, S.R. Choudhury and K. Kodama *et al*., 2006. Effect of conventional risk factors for excess cardiovascular death in men NIPPON data80. Circulation J., 70: 370-375.

Toufan, M., R. Azarfarin, B. Sadati and S.E. Golzari, 2012. The association between epicardial adipose tissue and coronary artery disease: An echocardiographic cut-off point. J. Cardiovasc. Thoracic Res., 4: 31-36.

Wong, C.X., H.S. Abed, P. Molaee, A.J. Nelson and A.G. Brooks *et al*., 2011. Pericardial fat is associated with atrial fibrillation severity and ablation outcome. J. Am. Coll. Cardiol., 57: 1745-1751.

Yanez, R.T.G., B.M.A. Gonzalez, B.J.L. Castillo, T.M.E. Hernandez and T.J.E. Lopez *et al*., 2014. Relationship between epicardial adipose tissue, coronary artery disease and adiponectin in a Mexican population. Cardiovasc. Ultrasound, 12: 35-35.

Yeboah, J., R. Erbel, J.C. Delaney, R. Nance and M. Guo *et al*., 2014. Development of a new diabetes risk prediction tool for incident coronary heart disease events: The multi-ethnic study of atherosclerosis and the heinz nixdorf recall study. Atherosclerosis, 236: 411-417.