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Original Article

Epicardial adipose tissue thickness in newly diagnosed adult onset asthma

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Abstract

Aim: In recent years, asthma, a respiratory disease with an increasing incidence , is often associated with obesity. The parallel increase in the prevalence of asthma and obesity is shown as a reason for this relationship and joint studies are conducted for both diseases. Epicardial adipose tissue (EAT) is a visceral adipose tissue on the pericardium and is associated with visceral obesity rather than general obesity. In this study, we planned to investigate the relationship between asthma and EAT, which is considered as an indicator of visceral adiposity.

Material and Methods: The study group consisted of 104 newly diagnosed asthma patients and 99 healthy controls. Transthoracic echocardiography was performed for all participants and EAT was measured.

Results: There was no statistically significant difference between baseline clinical and echocardiografic data between groups. Also EAT values were similar in both groups (5.40±0.61 mm in case subjects and 5.29±0.47 mm in healthy subjects ; p=0.142).

Conclusion: We did not find any difference between EAT measurements of newly diagnosed adult patients with asthma compared to healthy population.

Keywords: Epicardial adipose tissue; echocardiography; obesity; asthma.

INTRODUCTION

Asthma is a chronic disease characterized by airway inflammation, airway hyperresponsiveness and varying degrees of airway obstruction, characterized by symptoms of wheezing, shortness of breath, and cough. Asthma is a serious public health problem that is estimated to affect approximately 300 million people worldwide (1,2). Obesity is associated with many other diseases, primarily cardiovascular diseases, due to its metabolic complications (3). In recent years, the relationship between asthma and obesity has been investigated and different hypotheses have been proposed. The relationship between these two diseases has been investigated in recent years due to the simultaneous increase in the prevalence of asthma and obesity, and different hypotheses have been proposed. Although the reason for the relationship between obesity and asthma is not entirely clear, the mechanical effect of obesity on the lungs, the effect on the immune system and inflammatory response, and hormonal changes are thought to be possible factors (4,5).

Epicardial adipose tissue (EAT) is visceral adipose tissue that surrounds the heart between the myocardium and the inner stratum of the pericardium. It is accepted as a visceral adipose tissue surrounding myocardium, which is thought to be a good indicator of visceral adiposity rather than general adiposity, is considered as an important risk factor in diseases such as metabolic syndrome and coronary artery disease (6,7). Echocardiography has been previously validated as a cheap and simple method in the evaluation of EAT and its results were compatible with MRI (8).

In the literature, there are very few studies evaluating EAT thickness in asthmatic patients and investigating its clinical significance. In these studies, there are factors that may affect the results such as treatment of patients and having other additional diseases in the patient population (9,10). Taking this into consideration, we aimed to investigate whether echocardiographicly measured EAT is

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different from the normal population in newly diagnosed adult-onset astmatic patients without additional disease and without a history of use of a drug.

MATERIAL and METHODS

This study was a cross-sectional study. It consisted of 104 patients with newly diagnosed asthma who were admitted to the chest diseases outpatient clinic of Mehmet Akif İnan Training and Research Hospital between September 2016 and March 2018 and 99 patients with a similar age and gender served as controls. The patients with asthma were diagnosed according to the criteria of the Global initiative for Asthma guidelines (GINA) , with the pulmonary function test (PFT) results and clinical data of the patients by a pulmonogist(11). Routine biochemical blood analysis and anthropometric measurements were performed on the same day with polyclinic evaluation. Patients with a history of cardiovascular or any systemic disease, who had a history of drug use, who were smoking and under the age of 18, were excluded from the study. The study was conducted in accordance with the Helsinki Declaration. Ethical approval was obtained by the local ethics committee and informed consent forms were obtained from all participants.

Transthoracic echocardiography was performed by two cardiologists experienced in echocardiography who were aware of the clinical data of the participants by using the 2.5-3.5 MHz ultrasound probe in the left lateral position (Philips iE33, Andover, Md., USA). The recordings were taken with three cardiac cycles accompanied by a single derivation electrocardiogram. All subjects were evaluated with the standard two-dimensional by taking parasternal long axis, short axis, apical four chamber and two chamber images in accordance with the recommendations of the American Society of Echocardiography criteria (12).

EAT was detected as the echo-free distance between the right ventricle and the inner leaf (visceral layer) of the pericardium. It was measured from a point vertical to the aortic anulus over the right ventricular free wall at the end of the diastole during 3-4 heart cycles. Measurements were obtained by two different cardiologists blinded to the study subjects twice at different times and the average of the values of both cardiologists were taken.

Twenty randomly selected patients' recordings were used to determine intra-observer and inter-observer variability of EAT measurementby two blinded cardiologists with two way Anova approach. The intra-observer and inter-observer variability values were0,97 (95% CI; 0,91–0,99) and 0,96 (95% CI; 0,90–0,97)respectively.

Statistical Analysis

While evaluating the findings of the study, SPSS software program (version 21.0; SPSS Inc., Chicago, IL, USA) was used for statistical analysis. While evaluating the study data, descriptive statistical methods (mean, standard deviation), as well as the comparison of normally distributed quantitative data, Student t test was used. The results were evaluated at 95% confidence interval and p <0.05 at significance level.

RESULTS

A total of 203 participants (104 subjects in case group, 99 subjects in control group) were included in the study. All patients in the case group consisted of patients with mild-stage asthma. Clinical features of the study groups and laboratory results are presented in Table 1. There was no significant difference between the groups in terms of age, gender, body mas index (BMI), heart rate, blood pressure values and laboratory parameters except high sensitive C-reactive protein.

Baseline echocardiographic measurements of the study population are shown in Table 2. Echocardiographic values were similar in both groups. There is not any significant difference in EAT values between gruops (5.40 ± 0.61mm in astmatics and 5.29 ± 0.47 in controls; p=0.142).

Table 1. Clinical, laboratory and pulmonary function test data of the study population

	Patients (n=104)	Controls (n=99)	P Value
Age (years)	25.9±4.4	25.6±4.1	0.403
Male (n)	68	64	0.847
BMI (kg/m2)	26.1±2.3	25.7±2.4	0.149
SBP (mmHg)	122.7±6.5	121.5 ±6.4	0.410
DBP (mmHg)	77.4±6.1	77.1 ±6.4	0.714
Heart rate (beats/minute)	80±9	75 ± 9	0.206
HsCRP (mg/dl)	0.51±0.33	0.40±0.44	0.007
Leukocytes (G/l)	7.38±0.37	7.02±0.39	0.551
Hemoglobin (g/dl)	13.9±1.9	14.0±1.3	0.255
FEV1	92.5±9.7	95.8±9.1	0.051

BMI: body mass index, DBP: diastolic blood pressure, FEV1: forced expiratory volume in 1 second, hsCRP: high sensitive C- reactive protein, SBP: systolic blood pressure, Bolded data indicate significance

Table 2. Echocardiographicdata of the study population

	Patients (n=104)	Controls (n=99)	P Value
LV EF (%)	65.19±3.91	66.14±3.11	0.215
LV EDD (mm)	45.94±4.42	44.97±3.55	0.105
LV ESD (mm)	26.70±2.11	26.55±2.35	0.419
Septum (mm)	9.40±0.40	9.50±0.90	0.624
Posterior wall (mm)	9.30±0.70	9.50±0.90	0.365
LA diameter (mm)	36.91±2.52	37.27±2.95	0.486
RA diameter (mm)	32.35±2.92	32.57±3.16	0.516
RV diameter (mm)	28.12±2.46	28.35± 2.90	0.324
EAT (mm)	5.40±0.61	5.29±0.47	0.142

EAT: epicardial adipose tissue, EF: ejection fraction, LA: left atrium, LV EDD: left ventricle end-diastolic dimension,LV EDD: left ventricle end-systolic dimension,RA: right atrium, RV: right ventricle

DISCUSSION

Epicardial adipose tissue (EAT) is a visceral adipose tissue surrounding the heart located between the myocardial tissue and the inner layer of the pericardium. According to previous studies, visceral adipose tissue is metabolically more active than subcutaneous adipose tissue and therefore more dangerous for the cardiovascular system. Epicardial adipose tissue is considered as an indicator of visceral adipose tissue surrounding myocardium which is correlated with central obesity and is considered as an important risk factor in diseases such as metabolic syndrome and cardiovascular diseases (6,7,13).

According to the data obtained from a national survey, the obesity prevalence ranges from 21% to 32% in patients with asthma (14). Many studies reported the the relationship between asthma and obesity (4,15). It has been reported that subclinical inflammation is increased in obese patients. Because chronic inflammation also plays a role in the etiopathogenesis of asthma, this may be the reason why asthma is common in obese patients. But it is not still clear that whether which type of obesity (general or visceral) is more important in understanding the relationship between asthma and obesity (4).

The most important feature of EAT, which distinguishes it from other visceral fat tissues, is that it can affect myocardium and coronary arteries as paracrine with many proinflammatory, proatherogenic molecules that EAT secretes besides systemic effects. EAT cells secrete many metabolically active molecules, such as tumor necrosis factor α, monocyte chemoattractant protein-1, resistin, interleukin-6, interleukin-8 and interleukin-1β. These molecules, as mentioned above, cause systemic inflammation as well as paracrine effects directly to the myocardium and coronary arteries that create an inflammatory environment (7,13,16). These molecules have also been shown to play a role in chronic airway inflammation in the pathogenesis of asthma (17).

Nowadays, waist circumference values, which are considered as a practical measurement of visceral adiposity, do not always reflect the truth and it does not help to distinguish between visceral fat and subcutaneous fat. This has led researchers to find a cheap, accessible and quantitative marker that can be used instead of waist circumference measurement. A significant relationship was reported between the echocardiographic measurements of epicardial fat tissue and the clinical and anthropometric parameters of the metabolic syndrome. Therefore, as the epicardial fat mass reflects the amount of intraabdominal visceral fat, it is suggested that echocardiographic evaluation of this tissue may work as a reliable marker for visceral fat (6,18). Methods such as cardiac MRI or computed tomography to determine the amount of epicardial adipose tissue provide a more accurate measurement than transthoracic echocardiography, but these methods are both difficult to access and expensive. Moreover, the studies showed that the EAT thickness measured by echocardiography was highly correlated with MRI measurements (r=0.864) (8). Previous studies in asthma patients have shown that EAT measurement values are similar in comparison to the normal population (9,10). In our study, we found that the EAT values were not different from the normal population, confirming the results of these two studies. But the patient group in our study consisted of patients with newly diagnosed, untreated and mild asthma without any additional disease. The reason we did our study in such a patient group was to determine the effect of pure asthma disease on EAT by excluding other factors that might affect EAT measurement. Previous studies had factors such as drug use and comorbidity in patients with asthma, which may affect EAT measurement but their study group consisted of all stages of asthma (9,10). We do not know if we could have a different result if we could include more advanced stages of asthma patients who provided criterias for our study, because the patient group that provided our study criterias was only in the mild stage. Although previous studies have shown the relationship between asthma and visceral adiposity, we have obtained findings suggesting the opposite in our study (4,19).

Limitations

Our study is a cross-sectional study and presents only the data of a single center. In addition, only early-stage asthma patients were included in our study and we do not know if we could have a different result if patients in advanced stages were included. Although EAT measurement by transthoracic echocardiography is not a gold standard method, this method was used in our study because it is easily applicable and cheap and its results are compatible with MRI measurements.

CONCLUSION

In our study, we obtained similar EAT values which is an accepted indicator of visceral obesity in asthma patients compared to the normal population. According to this result, we can conclude that there is no relationship between visceral fat and asthma.

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