Turkish Journal of Geriatrics
2025; 28(2):130–136

DOI: 10.29400/tjgeri.2025.429

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Received : Feb 21, 2025
Accepted: May 08, 2025

Cite this article as:
Ayhan Albayrak G, Bardakçı Mİ, Bülbün G. The Relationship Between Epicardial and Paracardial Fat Thickness and Disease Severity in Elderly Patients with Chronic Obstructive Pulmonary Disease. Turkish Journal of Geriatrics 2025; 28(2):130–136. doi: 10.29400/tjgeri.2025.429

ORIGINAL ARTICLE

THE RELATIONSHIP BETWEEN EPICARDIAL AND PARACARDIAL FAT THICKNESS AND DISEASE SEVERITY IN ELDERLY PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Abstract

Introduction: Cardiovascular diseases are frequently observed in patients diagnosed with chronic obstructive pulmonary disease. The Body-mass index, airflow Obstruction, Dyspnea and Exercise index was used to assess the prognosis of patients with chronic obstructive pulmonary disease. Although epicardial fat thickness is associated with cardiovascular diseases, few studies have investigated its relationship with the Body-mass index, airflow Obstruction, Dyspnea and Exercise index. Therefore, we investigated this relationship in patients diagnosed with chronic obstructive pulmonary disease.

Materials and Method: We enrolled 78 patients with severe chronic obstructive pulmonary disease and 65 healthy controls. All patients underwent pulmonary function tests and a six-minute walk test. We used thoracic computerized tomography at admission to measure the epicardial and paracardial fat thickness. Participants' demographic data (age, sex, body mass index, and smoking status) and biochemical parameters (e.g., glucose, urea, creatinine) were measured.

Results: The epicardial fat thickness values of patients with chronic obstructive pulmonary disease were higher than those of the control group ($p < 0.05$). The B-Modified Medical Research Council dyspnea scale scores and Body-mass index, airflow Obstruction, Dyspnea and Exercise indices did not differ significantly between the low and high paracardial fat thickness/body mass index ratios.

Conclusion: This study demonstrated an independent and negative relationship between epicardial fat thickness with the Body-mass index, airflow Obstruction, Dyspnea and Exercise index, which indicates the severity of chronic obstructive pulmonary disease.

Keywords: Pulmonary Disease, Chronic Obstructive; Epicardial Adipose Tissue; Severity of Illness Index.

INTRODUCTION

Epicardial fat tissue (EFT) is the visceral adipose tissue of the heart. The relationship between visceral fat metabolic syndrome (MS) and cardiovascular diseases has previously been demonstrated. Many studies have identified a correlation between visceral fat tissue and EFT. Unlike other visceral fat tissues, the EFT has several local effects on the heart and supports the thermoregulation, and mechanical protection of the myocardium, anti-inflammatory cytokine release, and free fatty acid provision for the myocardium. However, its adverse effects have also been observed, including intrinsic inflammatory effects, high free fatty acid synthesis and release, reduction of glucose uptake and glucose delivery, high lipolysis, intrinsic insulin resistance, fat infiltration into the myocardium, proinflammatory transcriptome, proinflammatory secretome, atherogenic lipid and glucose metabolism, and mechanical obstruction of the heart (1). Several studies have investigated the relationship between EFT thickness, coronary artery disease (CAD), development of atrial fibrillation, and left ventricular diastolic dysfunction (2–4).

The thickness of EFT is a potential diagnostic marker or treatment target for cardiometabolic diseases because it can be measured using imaging methods and responds rapidly to pharmacological and surgical treatments targeting fat burning. EFT is located between the myocardium and the visceral layer of the pericardium. A local interaction occurs between the EFT and the underlying coronary arteries and myocardium (5). The EFT is defined as an endocrine and inflammatory organ that secretes proatherogenic and proinflammatory cytokines. The relationship between the EFT and MS, diabetes mellitus, and CAD has been demonstrated in many studies (6). Various imaging methods have been used to measure the EFT. Transthoracic echocardiography (TTE) is the preferred method for measuring EFT because of its superior advantageous features such as easy accessibility, low cost, lack of radiation exposure, and simultaneous acquisition of other cardiac parameters. The thickness of EFT measured using TTE is strongly correlated both with the epicardial and abdominal visceral fat volume and the anthropometric and metabolic parameters measured using magnetic resonance imaging (7).

Previous research studies indicated that visceral fat tissue is metabolically more active than subcutaneous fat tissue and is, therefore, imposes a greater burden on the cardiovascular system. EFT is the visceral fat tissue located around the heart and is approximately 5 mm thick in healthy individuals (8). It surrounds the coronary arteries and exerts paracrine, endocrine, and inflammatory effects. It is also implicated in the development of CAD, MS, insulin resistance, and hypertension (9). Although epicardial fat tissue is essential for regulating vascular functions and providing energy in healthy conditions, an increase in epicardial fat tissue makes it a lipolytic, prothrombotic, and pro-inflammatory organ. Studies have shown that EFT thickness increases significantly in hypertensive patients compared with normotensive individuals and that the amount of EFT is positively correlated with blood pressure levels (10). This study aimed to elucidate the relationship between EFT and the Body-mass index, airflow Obstruction, Dyspnea and Exercise (BODE) index in patients diagnosed with chronic obstructive pulmonary disease (COPD).

MATERIALS AND METHOD

A total of 143 patients were enrolled in this single center, retrospective study. Ethics committee approval was granted by our institution (protocol number 2695/09.07.2024), and since the data of the study were obtained by scanning patients' files from the hospital automation system, informed consent was not obtained from the participants

The study group comprised 78 patients with severe COPD, and the control group comprised 65 individuals. All patients underwent pulmonary function tests and a six-minute walk test. Thoracic computed tomography at admission measured parameters such as EFT and paracardial fat

thickness (PFT). Participants' demographic data (age, gender, body mass index [BMI], and smoking status) and biochemical parameters (e.g., glucose, urea, creatinine, alkaline transaminase (ALT), aspartate transaminase (AST), C-reactive protein (CRP), hemoglobin (Hgb) levels, white blood cell (WBC), and platelet (PLT) counts) were analyzed.

Statistical Analysis

We analyzed the patients' data using Statistical Package for the Social Sciences for Windows 26.0 (IBM Corp., Armonk, NY). The frequencies and percentages for the categorical data and the means and standard deviations for the continuous data were assessed as the descriptive values. For comparisons between the two groups, an independent samples *t*-test was used. Pearson's chi-square test was used to compare the categorical variables. The results were considered statistically significant when the *p*-value was less than 0.05.

RESULTS

Based on our study data, COPD courses more severely in our male patients, and older population

Table 1. Demographic, biochemical data and pulmoner function tests findings, BODE index, EFT and PFT thickness parameters of the patient, and control groups.

Variables	Patient group (n = 78)	Control group (n = 65)	p-value
Sex n (%)			
Female	19 (24.4)	32 (49.2)	0.002**[1]
Male	59 (75.6)	33 (50.8)	
Age , years: median (Min–Max)	69.5 (64–96)	68 (65–83)	0.033*[2]
BMI kg/m^2 median (Min–Max)	26.1883 (15.57–46.66)	26.2227 (20.55–33.2)	0.651[2]
Smoking history median (Min–Max)	40 (26–56)	40 (22–52)	0.855[2]
FBG, mg/dL median (Min–Max)	99 (75–197)	98 (76–135)	0.361[2]
BUN, median (Min–Max)	38.5 (17–153)	33 (13–72)	0.028*[2]
Creatinine median (Min–Max)	0.965 (0.32–3.82)	0.96 (0.48–1.55)	0.053[2]
ALT median (Min–Max)	15 (7–71)	23 (8–59)	0.0001**[2]
AST median (Min–Max)	20 (10–62)	25 (6–60)	0.015*[2]
CRP median (Min–Max)	6.16 (0.52–199.81)	4.15 (0.39–19.33)	0.001**[2]
WBC, 10^9/L: median (Min–Max)	8.165 (3.6–16.07)	7.3 (4.21–12.45)	0.022*[2]
HB, g/dL mean ± SD (Min–Max)	13.31 ± 1.8 (9.9–17.6)	13.5 ± 1.7 (8.4–17.3)	0.526[3]
HCT (%) mean ± SD (Min–Max)	40.38 ± 5.2 (30.1–53.2)	40.33 ± 4.7 (27.7–51.2)	0.953[3]
PLT. 10^9/L median (Min–Max)	241 (108–713)	258 (110–412)	0.534[2]
FVC (L) median (Min–Max)	1.34 (0.41–2.58)	1.88 (0.97–4.46)	0.0001**[2]
FVC (%) median (Min–Max)	43.5 (15–68)	63 (43–104)	0.0001**[2]
FEV1 (L) median (Min–Max)	0.95 (0.38–1.57)	1.47 (0.85–3.39)	0.0001**[2]
FEV1 (%) median (Min–Max)	38 (15–49)	64 (38–101)	0.0001**[2]
FEV1/FVC mean ± SD (Min–Max)	71.41 ± 13.1 (39–100)	80.98 ± 8.2 (61–101)	0.0001**[3]
BODE index median (Min–Max)	8 (4–11)	3 (0–8)	0.0001**[2]
EFT median (Min–Max)	17.25 (6.9–24.8)	14.6 (6.8–27.8)	0.0001**[2]
PFT, mm median (Min–Max)	205.555 (54.2–585.32)	157.3 (16.63–412.39)	0.022*[2]

[1] Chi-square test; [2] Mann–Whitney U test; [3] Independent samples t-test; * $p < 0.05$; ** $p < 0.01$. Abbreviations: BMI: Body mass index, FBG: Fasting blood glucose, BUN: Blood urea nitrogen, ALT: Serum alanine aminotransferase, AST: Aspartate aminotransferase, CRP: C-reactive protein, WBC: White blood cell count, HB: Hemoglobin, HCT: hematocrite, PLT: Platelet, FVC:Forced vital capacity; FEV1: Forced expiratory volüme in 1 sec; BODE index: (B) Body mass index; (O) airflow obstruction measured by the forced expiratory volume in one second (FEV_1); (D) dyspnoea measured by the modified Medical Research Council (MRC) scale; and (E) exercise capacity measured by the 6 minute walk distance (6MWD), EFT: Epicardial fat thickness, PFT: Paracardial fat thickness

relative to the age-matched healthy study participants (p = 0.002 and p = 0.033, respectively). Serum urea (p = 0.028), CRP (p = 0.001), and WBC (p = 0.022) levels were significantly higher in the patient group. The ALT (p = 0.0001) and AST (p = 0.015) levels were significantly higher in the control group (Table 1). Forced vital capacity (FVC [L; p = 0.0001]), forced expiratory volume in 1 second (FEV1 [L; p = 0.0001]), FEV1 (%; p = 0.0001), and FEV1/FVC (p = 0.0001) were significantly lower in the patient group. Additionally, the BODE index was significantly higher in the patient group than in the control group (3; p = 0.0001; Table 1). The EFT thickness was significantly higher in the patient group (17.09 ± 4) than in the control group (14.86 ± 3.6; p = 0.0001). Similarly, the PAT was significantly higher in the patient group (229.69 ± 109.9) than in the control group (187.03 ± 95.6; p = 0.022; Table 2). We found that the BODE index was significantly higher in the patient group than in the control group (3; p = 0.0001). There was a statistically significant difference between the FEV1 (%) of those with low and high PFT/BMI ratios (p = 0.013). Accordingly, the cutoff values for EFT/BMI and PFT/BMI were determined as 0.64 and 8.48, respectively. The cut-off values were determined by taking the averages of the epicardial fat thickness/BMI (EFT/BMI), and paracardial fat thickness (PFT/BMI) parameters in the patient group (Table 3).

DISCUSSION

The BODE index is a significant prognostic predictor of COPD. It is a multidimensional scoring system that combines information on various clinical factors, including the BMI, airflow obstruction (forced expiratory volume in 1s [FEV1]), dyspnea (Medical Research Council Dyspnea Scale), and exercise capacity (six-minute walk distance), with scores ranging from 0 to 10 (11). Our study detected

Table 2. Comparison of EFT and PFT between patient and control groups

	Patient group (n = 78)			Control group (n = 65)			p-value
	Median	Min–Max	Mean ± SD	Median	Min–Max	Mean ± SD	
EFT	17.25	6.9–24.8	17.09 ± 4.00	14.6	6.8–27.8	14.86 ± 3.6	0.0001**
PFT	205.555	54.2–585.32	229.69 ± 109.9	157.3	16.63–412.39	187.03 ± 95.6	0.022*

Abbrevistions: EFT, Epicardial fat thickness; PFT: Paracardial fat thickness Statistically significant at levels:;* p < 0.05; ** p < 0.01.

Table 3. Comparisons based on parameters of EFT and PFT

EFT/BMI	Low (< 0.64; n = 44)			High (≥ 0.64; n = 34)			p-value
	Median	Min–Max	Mean ± SD	Median	Min–Max	Mean ± SD	
FEV1 (%)	39	15–49	38.32 ± 8.8	36	15–49	36.41 ± 9.2	0.371
B-mMRC	3	1–4	2.89 ± 0.9	3	1–4	2.91 ± 1.0	0.849
BODE index	8	4–10	7.3 ± 1.8	8	4–11	7.82 ± 2.1	0.223
PFT/BMI	**Low (< 8.48; n = 46)**			**High (≥ 8.48; n = 32)**			**p-value**
	Median	**Min–Max**	**Mean ± SD**	**Median**	**Min–Max**	**Mean ± SD**	
FEV1 (%)	40.5	15–49	39.46 ± 9.00	34.5	15–49	34.66 ± 8.2	0.013*
B-mMRC	3	1–4	2.8 ± 1.0	3	1–4	3.03 ± 0.9	0.302
BODE index	7	4–11	7.2 ± 2.1	8	4–10	8.0 ± 1.7	0.073

Abbreviations: EFT: Epicardial fat thickness; BMI: Body mass index; BODE: Body-mass index, airflow Obstruction, Dyspnea and Exercise; B-mMRC; B-modified Medical Research Council Scale; PFT: paracardial fat thickness; FEV1. Forced Expiratory Volume in 1 sec

no statistically significant differences between the FEV1, B-mMRC, or BODE index of those with low and high EFT/BMI ratios.

Extensive research has demonstrated that a significant proportion of individuals diagnosed with COPD also exhibit characteristics of metabolic syndrome (MS) and obesity. The coexistence of MS has been strongly correlated with elevated systemic inflammation levels, which play a crucial role in the pathophysiology of both conditions. Epicardial fat, a specialized type of visceral adipose tissue, is located between the myocardium and the visceral layer of the epicardium. This fat depot serves as a source of various endocrine and inflammatory mediators, contributing to metabolic dysregulation and cardiovascular complications (12). Evidence suggests that epicardial fat accumulation plays a pivotal role in the development of atherosclerosis and is linked to unfavorable cardiometabolic outcomes. A study by Demir et al. revealed that patients with COPD exhibit significantly increased epicardial fat thickness (EFT) compared to healthy individuals (13).

The observed elevation in EFT is believed to be a direct consequence of the systemic inflammatory nature of COPD. Moreover, an EFT measurement exceeding 6.75 mm has been identified as a reliable biomarker for predicting MS in COPD patients, with a sensitivity of 83% and a specificity of 65%. These findings support the notion that the presence of MS further exacerbates the expansion of epicardial fat in individuals with COPD. Key diagnostic indicators of MS, such as elevated plasma glucose levels and hypertension, may also be influenced by the long-term administration of corticosteroids, which are commonly prescribed for COPD management. These metabolic alterations can persist over an extended period, further complicating disease progression. However, the accumulation of epicardial fat is typically a gradual and progressive process. Consequently, the non-invasive assessment of EFT may serve as a valuable tool for early detection and monitoring of MS in COPD patients. Additionally, our findings indicate that both EFT and pulmonary function test (PFT) values were substantially higher in COPD patients, underscoring a potential link between epicardial fat expansion and impaired respiratory function (13).

Zagaceta et al. examined the relationship between COPD, epicardial fat thickness (EFT), and the BODE index, highlighting their significance as indicators of disease severity (14). Kiraz et al. were the first to conduct a comprehensive study assessing the link between EFT and COPD progression, as measured by the BODE index, in a cohort comprising 157 COPD patients and 45 healthy control subjects (15). To better understand this association, patients were stratified into four distinct quartiles based on their BODE index scores: Q1 (0–2 points), Q2 (3–4 points), Q3 (5–6 points), and Q4 (7–10 points). The results demonstrated that individuals with COPD exhibited markedly higher EFT levels compared to those in the control group, suggesting a possible connection between increased epicardial fat deposition and disease advancement. Among patients with COPD, the highest EFT values were observed in Q1 compared with the other BODE index quartiles ($p < 0.05$ for all). The EFT values decreased from Q1 to Q4. Furthermore, EFT was independently associated with the BODE index ($\beta = 0.405$, $p < 0.001$), CRP ($\beta = 0.300$, $p < 0.001$), and diabetes ($\beta = 0.338$, $p < 0.001$). The EFT was independently and negatively associated with disease severity, as indicated by the BODE index, in patients with COPD (15).

A study involving 202 patients examined the link between EFT thickness and the severity of CAD, suggesting that EFT measurements may serve as a useful marker for risk assessment in CAD patients. (16). Ahn et al. conducted an echocardiographic evaluation of EAT thickness in 527 patients undergoing coronary angiography for the first time and reported a significant correlation between EFT thickness and both the activation and

progression of CAD. Additionally, their findings indicated a strong association between EFT, insulin resistance, and systemic inflammation, highlighting its potential role in metabolic dysregulation. (17). Ulucan et al. demonstrated that an increase in EFT thickness was closely linked to a higher incidence of long-term major adverse cardiovascular events, further emphasizing its prognostic importance (18).

In research by Sridhar and Bhaskar, a sensitivity of 95.6% and a specificity of 77.8% were determined for a threshold EFT measurement of 6.5 mm (Area Under the ROC Curve AUC = 0.872; 95% confidence interval [CI]: 0.697–1.000) (19). Similarly, Naik et al. observed that EFT thickness was 4.82 ± 1.31 mm in CAD patients compared to 4.06 ± 1.25 mm in the control group, with a cutoff value of 3.9 mm found to be statistically significant (p = 0.005) (20). For a cut-off value of 3.9 mm, the sensitivity and specificity were 84% and 55%, respectively (AUC = 0.68, 95% CI: 0.58–0.79). Park et al. investigated the effect of the BMI on the relationship between CAD and EFT thickness in 643 participants (21). The patients were divided into two groups according to their BMI values (BMI < 27 kg/m^2 and ≥ 27 kg/m^2). The median EFT thickness values were higher in the low (3.5 mm and 1.5 mm, $p < 0.001$) and high (4 mm and 2.5 mm, $p = 0.001$) BMI groups compared to the control group. An ROC analysis showed that the EAT thickness measurement was more effective in predicting CAD in the low BMI group (AUC = 0.735 and 0.657) compared with the high BMI group. Park et al. hypothesized that the EFT is more potent in predicting CAD in patients with a BMI < 27 kg/m^2 (21). Ahn et al. found a significant relationship between the BMI of groups with an EFT above and below 3mm (22).

CONCLUSION

The results of this study suggest that BODE index which measures the severity of COPD increases in line with the increase in epicardial, and paracardial fat tissue thickness.

Conflict of Interest: The authors declare that they have no conflict interests.

Funding: There is no specific funding related to this research.

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