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ORIGINAL RESEARCH

Epicardial Adipose Tissue in Rheumatoid Arthritis Patients: More Than A Simple Fat Deposit?

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ABSTRACT

Rheumatoid arthritis (RA) is a multifaceted autoimmune disorder characterized by chronic joint inflammation, leading to progressive disability and significantly impacting patients' quality of life. The exact mechanisms underlying RA pathogenesis remain unclear. Epicardial adipose tissue (EAT), a metabolically active form of adipose tissue, has been linked to systemic inflammation and increased cardiovascular risk. One emerging area of research in RA is the role of EAT in the pathogenesis of the disease. This study aims to investigate the relationship between EAT and clinical and laboratory features in patients with RA. A total of 342 patients were recruited from the rheumatology department of a single center cross-sectional study. The participants were divided into two groups: 200 patients with RA and 142 age-matched healthy controls (HC). Laboratory and radiology results were obtained from the electronic registration database. Data were analyzed and compared between groups. EAT was measured using computed tomography (CT), and its correlations with clinical features and inflammatory markers were evaluated. EAT was found to be significantly higher in the RA than HC. There was a strong positive correlation between EFV and activity score (DAS28) ($p<0.001$) and weak positive correlation between EFV and age, leukocyte, lymphocyte, RDW and CRP ($p<0.05$). The EAT did not differ significantly in terms of treatment. ROC analysis showed a moderate discriminatory power of EAT with an AUC of 0.604 (95% CI: 0.542-0.666, $p<0.001$) with a sensitivity of 69.0% and a specificity of 51.4% at a cut-off value of 123.68. EAT may be an indicator of active inflammation in RA patients.

Keywords: Epicardial adipose tissue. Inflammation. Rheumatoid Arthritis.

Romatoid Artrit Hastalarında Epikardiyel Yağ Dokusu: Basit Bir Yağ Dokusundan Daha Fazlası mı?

ÖZET

Romatoid artrit (RA), kronik eklem iltihabı ile karakterize, ilerleyici sakatlığa yol açan ve hastaların yaşam kalitesini önemli ölçüde etkileyen bir otoimmün hastalıktır. RA patogenezinin altında yatan kesin mekanizmalar henüz netlik kazanmamıştır. Metabolik olarak aktif bir yağ dokusu olan epikardiyal yağ dokusu (EYD), sistemik inflamasyon ve kardiyovasküler riskle ilişkilendirilmiştir. EYD 'nin RA patogenezindeki rolü yeni araştırma alanlarından biri olmuştur. Bu çalışma, RA hastalarında EYD ile klinik ve laboratuvar özellikleri arasındaki ilişkiyi değerlendirmeyi amaçlamaktadır. Tek merkezli kesitsel çalışmaya Romatoloji kliniğinde takipli toplam 342 birey dahil edildi. Katılımcılar iki gruba ayrıldı: 200 RA hastası ve 142 yaş eşleştirilmiş sağlıklı kontrol (SK). Laboratuvar ve radyoloji sonuçları elektronik veritabanından elde edildi. Veriler analiz edildi ve gruplar arasında karşılaştırıldı. EYD bilgisayarlı tomografi (BT) kullanılarak ölçüldü ve klinik özellikler ve inflamasyon belirteçleriyle korelasyonları değerlendirildi. EYD'nin RA'da SK'ye göre anlamlı olarak yüksek olduğu bulundu. EYD ile aktivite skoru (DAS28) ($p<0,001$) arasında güçlü, yaş, lökosit, lenfosit, RDW ve CRP ($p<0,05$) arasında zayıf pozitif korelasyon vardı. Tedavi açısından karşılaştırıldığında anlamlı bir fark yoktu. ROC analizi, EYD'nin 123,68 kesme değerinde %69,0 duyarlılık ve %51,4 özgüllükle 0,604'lük bir AUC (95% CI: 0,542-0,666, $p<0,001$) ile orta düzeyde bir ayırt edici güce sahip olduğunu gösterdi. EYD, RA hastalarında aktif inflamasyon göstergesi olabilir.

Anahtar Kelimeler: Epikardiyal yağ dokusu. İnflamasyon. Romatoid Artrit.

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The multidimensional autoimmune disease known as rheumatoid arthritis (RA) is typified by persistent joint inflammation that eventually results in disability. Numerous systems may experience complications as a result of RA, many of which have the potential to substantially raise a patient's risk of mortality and adversely impact their quality of life. The precise mechanisms that underlie the pathogenesis of RA are still unknown. It is now acknowledged that RA emerges as a result of a complex interaction between immune system dysregulation, environmental exposures, epigenetic influences, and genetic predisposition. Inflammatory processes, increased cell proliferation, invasion, migration, and angiogenesis are all factors in the pathophysiology of RA, leading to the progressive degradation of the joints. The development of RA is caused by a complex interplay between immune cells and various cytokines that promote synoviocyte growth and lead to the deterioration of bone and cartilage.[1] A deeper understanding of the pathogenesis of the disease is crucial for developing innovative diagnostic markers and treatment approaches. Epidemiological data suggest that RA serves as an independent risk factor for cardiovascular disease (CVD). Recent epidemiological findings indicate that CVD are responsible for nearly 50% of all deaths associated with RA[2]. An emerging field of study in RA is the significance of epicardial adipose tissue (EAT) in the disease's pathogenesis[3]. Recently, numerous studies have shown that EAT is linked to insulin resistance, heightened cardio-metabolic risk, inflammatory markers, and coronary artery disease[4]. Recent studies have suggested that EAT may also be involved in the inflammatory mechanisms linked to RA, rendering it an intriguing target for further investigation.

EAT, located between the myocardium and visceral pericardium, has been identified as a metabolically active tissue with a role in inflammation and cardiovascular (CV) risk. EAT is highly vascularized and possesses endocrine and paracrine functions. EAT, unlike subcutaneous fat, interacts directly with the myocardium and coronary arteries, excretes numerous proinflammatory and proatherogenic cytokines, chemokines, and adipokines. Aberrant EAT accumulation alters the anti-inflammatory and pro-inflammatory balance, aggravates oxidative stress, and causes atherosclerosis. As a result, EAT promotes the progression of subclinical atherosclerosis via vascular inflammation and endothelial dysfunction. Several studies have shown that EAT is associated with negative CV outcomes that vary from asymptomatic to overt coronary artery disease, regardless of other risk factors[4,5].

EAT volume (EFV) can be measured with echocardiography, computed tomography (CT), or magnetic resonance imaging (MRI). CT is a trustworthy and replicable method for quantifying EFV, independent of cardiac cycle phase[6,7]. Increased EFV has been associated with autoimmune disease (AID), such as systemic lupus erythematosus (SLE), systemic sclerosis (SSc), psoriasis (PSO), and RA, and with cardiometabolic disease[8-11]. The goals of this study were to assess EFV using CT in RA patients compared to age- and sex-matched controls and to identify risk factors for EFV.

Material and Method

Study Design and Population

The study included 200 consecutive patients who presented to a tertiary rheumatology outpatient clinic between 2020 and 2021, aged ≥ 18 years, and diagnosed with RA based on the 2010 American College of Rheumatology/European League Against Rheumatism Rheumatoid Arthritis Classification Criteria[12]. In this cross-sectional study, 142 healthy subjects (HC) who were matched for age and sex were included at the same time. The HC group was chosen from among participants who had normal blood tests, no known chronic diseases, applied to the internal medicine outpatient clinic with cough and dyspnea, underwent an elective thoracic CT, and had no pathological findings. Exclusion criteria were pregnant or breast-feeding patients, coronary heart disease, cerebrovascular and peripheral artery disease, heart failure, body mass index (BMI) ≥ 30 kg/m2, cardiomyopathy, moderate to severe valvular disease, serious arrhythmia, pericardial effusion, chronic liver, pulmonary and kidney disorders, malignancy, prior cardiac surgery or interventions, severe infections, and combinations of other autoimmune/autoinflammatory disorders. Additionally, patients with poor-quality imaging that prevented accurate measurement of EAT were excluded. The study protocol was approved by the institutional ethics committee (Approval Number: 2021/297), and all procedures were performed in line with the principles of the Declaration of Helsinki.

Clinical and Laboratory Assessments

Patient history, physical examination and laboratory test results were obtained from medical records and information provided by patients. The onset of their disease, smoking habits, ongoing treatment for RA, existence of additional medical conditions, tender joint count (TJC), swollen joint count (SJC), as well as the visual analogue scale evaluations by both the patient and physician (0–100 cm) were documented. The evaluations of disease activity for the RA patients were conducted in accordance with the disease activity scores derived from 28 joints (DAS28-CRP). DAS-28 is calculated according to the formula that is composed of the number of tender joints and swollen joints, and CRP[13].

Laboratory parameters included neutrophil, lymphocyte, monocyte, platelet count, hemoglobin, mean platelet volume (MPV), and red cell distribution width (RDW). Renal and hepatic function tests were performed to exclude patients with significant organ dysfunction. The erythrocyte sedimentation rate (ESR; 0–20 mm/hour), C-reactive protein (CRP; 0–8 mg/l), autoantibodies [anti-citrullinated protein antibodies (anti-CCP) and rheumatoid factor (RF)] of the patient group were recorded.

Imaging assessments

EAT was assessed using non-contrast-enhanced CT (Somatom Scope 16 or Somatom Definition Flash, Siemens Healthcare, Germany). The patient was placed in the supine position and CT scans were taken at full inspiration. EFV was measured following a standardized protocol using axial CT slices. EAT was defined as the fat surrounding the myocardium, bounded externally by the visceral pericardium using semi-automated volumetric analysis. Typical chest CT image acquisition parameters included a pitch of 1-1.5, 1.2-mm collimation, 3-mm slice thickness, 2-3 mm reconstruction interval, 80-130 kVp, and 100-250 mAs. All imaging assessments were conducted independently by two experienced radiologists who were blinded to the patients' clinical and laboratory data, and inter-observer variability was calculated.

Statistical Analysis

The Statistical Package for Social Sciences software was used for all procedures (IBM SPSS Statistics 21.0, IBM Corporation, Armonk, NY, USA). The normal distribution of the scale variables was ascertained using the Kolmogorov–Smirnov test. For continuous numerical variables, descriptive statistics are presented as mean and standard deviation. Categorical variables are represented by the number of cases and the percent. The Chi-square test was used to compare categorical variables, and the Student's t-test and Mann-Whitney U test were used to compare continuous numerical variables. Receiver operating characteristic (ROC) curve analysis was used to assess diagnostic performance. If the area under the curve was found to be significant, the Youden index was used to verify the best cut-off point. The sensitivity and specificity of diagnostic performance indicators were calculated. To ascertain the relationship between EAT and laboratory values, Pearson correlation analysis was used. Unless otherwise specified, the results were deemed statistically significant at $p<0.05$.

Results

200 RA patients with a mean age of 56.21 ± 11.10 years were included in the study. The control group included 142 HC with a mean age of 54.08 ± 11.54. Thyroid disease, asthma, diabetes mellitus, and hypertension were among the additional comorbidities that were present in 83 RA patients (41.5%). A total of 151 patients (75.5%) utilize conventional synthetic disease-modifying anti-rheumatic medications (csDMARDs), which include methotrexate, leflunomide, hydroxychloroquine, and sulfasalazine, or targeted synthetic disease-modifying anti-rheumatic medications (tsDMARDs: Janus kinase inhibitors). Eight patients receiving DMARD treatment were receiving Janus kinase (JAK) inhibitors. Biological disease-modifying anti-rheumatic medications (bDMARDs) were administered to 86 RA patients (44.0%). These included the tumor necrosis factor-alpha (TNF) inhibitors: infliximab, adalimumab, golimumab, certolizumab pegol and etanercept, the CD80/CD86 costimulation inhibitor abatacept, the interleukin-6 (IL-6) inhibitors, and the CD-20 depleting agent rituximab. Of them, 37 were taking concurrent csDMARDs, while 49 were solely taking bDMARDs. Most patients were taking concomitant low-dose ≤5 mg corticosteroids. Every participant was treated with either a biologic or nonbiologic DMARD for a minimum of six months. The laboratory test results for the RA patients are shown in Table I. Table II shows the demographic characteristics of RA patients and the HC. The groups did not differ significantly in terms of age and gender ($p>0.05$). The EFV was found to be significantly higher in the RA patient group than in the HC ($p=0.003$). Within the RA patient group, the EFV was significantly higher with additional disease ($p=0.018$) and DMARD usage ($p=0.049$). The EFV did not differ significantly in terms of treatment (Table III). Table IV shows the results of the ROC analysis. The EFV demonstrated diagnostic performance in differentiating RA patients from HC. The AUC was 0.604 (0.542-0.666) ($p<0.001$), with a sensitivity of 69.0% and a specificity of 51.4% at a cut-off value of 123.68. Pearson correlation analysis was performed to determine the relationships between the EFV and laboratory values of RA patients (Table V). A strong positive correlation was found between EFV and the activity score (DAS28) ($p < 0.001$). There was a weak positive correlation between EFV and age, leukocyte, lymphocyte, RDW and CRP ($p<0.05$).

Table I. Clinical characteristics and laboratory findings of RA patients (n=200)

Leukocyte (10^9/L) *	7.79 ± 2.6
Hemoglobin (g/L) *	12.92 ± 1.77
Platelet (10^9/L) *	289.45 ± 89.72
Neutrophile (10^9/L) *	6.03 ± 19.66
Monocyte (10^9/L) *	0.64 ± 0.48
Lymphocyte (10^9/L) *	2.18 ± 0.90
Red cell distribution width (RDW) (%) *	15.68 ± 3.58
Mean platelet volume (MPV) (FL) *	8.43 ± 1.22
C-reactive protein (CRP) (mg/L) *	14.22 ±19.10
Erythrocyte sedimentation rate (ESR) (mm/H) *	30.64 ±22.8
Disease duration	7.94 ± 4.81
DAS-28 activity score	4.27 ± 1.28

* Data are presented as mean ± standard deviation. ** Data are presented as counts, with percentages in brackets.DAS-28: disease activity scores based on 28-joints, RA: rheumatoid arthritis

Table II. Demographic and clinical characteristics of RA patients and healthy control group

	RA patients (n=200)	Healthy control (n=142)	*p-value*
Gender (female), n (%) *	155/200 (77.5)	112/142 (78.9)	0.762
Female	155 (58.1)	112 (41.9)	
Male	45 (60.0)	30 (40.0)	
Age, (years) **	56.21 ± 11.10	54.08 ± 11.54	0.088
Epicardial Fat Tissue volume (cm^3) **	167.76 ± 81.95	139.96 ± 86.16	**0.003**

* Chi-Square Tests, data are presented as counts, with percentages in brackets; ** Independent sample *t*-test, data are presented as mean ± standard deviation. RA: rheumatoid arthritis

Table III. Comparisons RA Patients according to comorbitiy and treatment

	Epicardial Fat Tissue volume (cm^3) **	*p-value*
Additional disease		***0.018****
Negative	156.28 ± 78.66	
Positive	183.92 ± 84.25	
DMARD treatment		***0.049***
Negative (49)	150.51 ± 63.56	
Positive (151)	173.25 ± 86.55	
Biological agent therapy		*0.327*
Negative (114)	162.81 ± 87.31	
Positive (86)	174.31 ± 74.27	

* Independent sample *t*-test, data are presented as mean ± standard deviation. * Mann-Whitney U test, data are presented as median (min-max).RA: rheumatoid arthritis, DMARD: disease-modifying anti-rheumatic medications

Table IV. ROC analysis results of epipericardial fat tissue used for diagnosis of patients with RA.

	AUC (95% CI)	*p-value*	Cut-off	Sensitivity	Specificity
Epipericardial fat tissue	0.604 (0.542-0.666)	**0.001**	> 123.68	69.0	51.4

AUC: Area under the curve, 95% CI: 95% confidence interval. RA: rheumatoid arthritis

Table V. Correlation of epipericardial fat tissue volume and RA patients features(n=200)

	rs	*p*-value
EFV – Age	**0.280**	**<0.001**
EFV– Disease duration	-0.034	0.636
EFV– Activity score (DAS28)	0.734	**<0.001**
EFV – Hemoglobin	0.115	0.105
EFV– Leukocyte	0.179	**0.011**
EFV– Platelet	-0.054	0.450
EFV– Neutrophile	-0.045	0.527
EFV– Monocyte	0.067	0.345
EFV– Lymphocyte	**0.227**	**0.001**
EFV– RDW	**0.148**	**0.036**
EFV– MPV	0.084	0.236
EFV– CRP	**0.211**	**0.003**
EFV– ESR	0.037	0.601

rs: Pearson's rho correlation coefficients, BMI: Body mass index, MPV: mean platelet volume, RDW: red cell distribution width, PCT: platelets, ESR: erythrocyte sedimentation rate, CRP: C-Reactive Protein, RA: rheumatoid arthritis, DAS28: disease activity scores based on 28-joints,EFV: Epipericardial fat tissue volume

Discussion and Conclusion

RA is a chronic autoimmune inflammatory disease and is a major cause of disability worldwide, with a significant impact on the quality of life of patients[1]. Despite the paucity of research on EAT in RA, it is hypothesized that EFV is elevated in RA patients and is associated with a higher risk of CVD. We found that RA patients had higher EFV than subjects without the condition, which is in line with earlier studies. EAT's intrinsic inflammatory status makes it a promising therapeutic target for RA patients[3].

Mazurek et al. compared EAT and subcutaneous adipose tissue, observing that EAT produced more inflammatory cytokines[14]. EAT is an ectopic fat depot with endocrine and paracrine functions, secreting a range of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), leptin, IL-1β, and monocyte chemoattractant protein-1 (MCP-1). They hypothesized that the presence of proinflammatory mediators in the tissues surrounding the coronary arteries can lead to an enhancement of vascular inflammation and neovascularization through apoptosis. The biologically active substances secreted

by EAT may act on coronary and myocardial tissues by paracrine and vascular secretion, thereby increasing the inflammatory response and influencing the structure and function of the arteries and myocardial tissues. This is because there is no anatomical barrier between the EAT and the myocardium, and the coronary artery, and the microcirculation is identical in both[15]. Packer et al. proposed that EAT acts as an inflammatory reservoir, exacerbating systemic autoimmune conditions[16].

In several studies evaluating the EAT in rheumatic diseases, patients with psoriatic arthritis, inflammatory bowel disease, familial Mediterranean fever (FMF), spondyloarthritis (SpA), SSc and SLE were found to have an increased EAT[17-26]. Lipson et al. identified elevated EAT thickness (EATT) in SLE, linking adipose-driven inflammation to heightened CV risk[23]. Increased EAT is related to endothelial dysfunction in SpA. Systematic review and meta-analysis also show that FMF patients and PSO patients have a higher EAT compared to control subjects[26-28]. PSO patients had higher EATT values than HC patients, and EATT was linked to psoriasis disease activity scores in a study involving 115 patients[27]. It was shown that EATT was connected with disease activity scores in a study involving SSc patients[28]. In the previous studies conducted by Keleşoğlu Dinçer AB, et al., Saha et al., Fatma et al., Lima et al., Ekinci et al., Yao H, et al, and Başpınar et al., EATT was considerably greater in RA patients in comparison to HC, consistent with our results[29-34]. A cross-sectional research involving a cohort of 34 RA patients and 16 age- and body mass index-matched controls revealed that individuals with RA exhibited greater EAT compared to those without R[11]. Another cross-sectional study involving 76 RA patients and 50 age and gender matched controls reported that EATT was higher in RA patients[29]. A recent cross-sectional study in 90 RA patients and 59 age and gender-matched controls showed that RA patients had a fatter EAT[34]. However, EATT was not substantially higher in RA patients than in HC patients in the Ormseth et al. study[35].

The present study showed a significant correlation between EAT and systemic inflammatory markers in RA without CVD. These results underscore the potential role of EAT as an inflammation-related factor in RA. Although the findings are statistically significant, they are clinically limited. In addition, higher EAT was observed in RA patients with higher disease activity scores in our study. Comparable to our research, Alpaydın et al. EATT was shown to have a positive correlation with DAS28 scores[36]. Temiz et al. revealed that EATT had a significant relationship with ESR, CRP, and Health Assessment Questionnaire scores, but not with DAS28 scores[19]. Saha S, et al., found that RA patients had higher EFV, left ventricular mass, and diastolic dysfunction than HC patients. Additionally, DAS28, disease duration, RF, anti-CCP, and inflammatory markers (ESR, hs-CRP) were linked to elevated EFV in RA patients[30]. EAT was shown to have statistically significant relationships with CRP and other disease activity markers by Keleşoğlu Dinçer AB et al[34]. In a study by Delkash P et al., EATT was correlated with RF, Anti-CCP, ESR, and systolic blood pressure. High EATT is correlated with more CVD and is an early sign and independent predictor of atherosclerosis in RA patients, which greatly underlines the importance of CV assessment in RA patients[37]. Petra et al. and Ormseth et al. found no significant relationships between EATT values, DAS28-CRP scores, CRP, and ESR, which is in contrast to our findings[38].

In a study including 34 female RA patients, Lima-Martinez et al. found that, in comparison to RA patients, healthy women had the lowest EATT levels. EATT was lower in RA patients on biological DMARDs than in those on non-biological DMARDs. RA patients using TNF-α therapies had lower EAT, according to a case-control study[11]. Regardless of the medication, we discovered a substantial association between EAT and our RA patients. Secondly, it is unable to draw any inferences about how DMARDs affect EAT due to the cross-sectional design. According to Kitterer et al., individuals receiving high doses of steroids had noticeably greater EAT than those receiving low doses[39]. However, there was no discernible change in EAT between steroid-naive and low-dose steroid-using individuals. The majority of our patients were on low-dose steroids, notwithstanding the possibility that corticosteroid usage contributed to the increased EFV in RA patients compared to healthy controls in our research.

Karpouzas et al. found that RA patients with higher EAT volumes had increased coronary atherosclerosis, independent of traditional CV risk factors. EATV and coronary plaque were assessed by CT angiography in 139 RA patients and 139 non-RA controls. Only RA patients had EATVs linked to increased plaque load, noncalcified component plaques, and susceptibility characteristics, despite the EATVs in RA patients and controls being identical[40]. RA patients without CVD symptoms had noticeably thicker EAT than controls, according to Wang T. et al. While EAT accumulation is linked to a reduced global longitudinal strain, RA-related LV myocardial systolic dysfunction may be more strongly influenced by advanced age and increased disease activity[20]. According to Petra et al.'s research, EAT and elevated arterial stiffness in RA patients were independently associated[38]. Fatma et al. found a favorable correlation between EAT and both the duration of illness and hypertension in RA patients[29]. In our study, no association was observed between EFV and the duration of the illness. These results may suggest that, particularly in the early

phases of the disease, the heightened risk of CV events is more strongly driven by disease activity rather than the length of disease progression. Furthermore, the present study identified that an elevated disease activity score in patients with RA constitutes an independent risk factor for increased EFV. Particularly, its relationship with the disease activation score is significant. EAT serves as a source of various inflammatory mediators, and evidence exists that illustrates the involvement of inflammation in the progression of atherosclerosis among individuals with RA. Furthermore, due to its inherent inflammatory characteristics, EAT has the potential to act as a therapeutic target for individuals suffering from RA. Packer et al. proposed that targeting EAT through pharmacological or lifestyle modifications could mitigate its detrimental effects on the myocardium. Weight loss and physical activity have been shown to reduce EFV and recover cardiac function[16]. Further research should also explore the potential of lifestyle modifications, pharmacological interventions, and targeted therapies to mitigate the effects of EAT accumulation in autoimmune diseases.

In the context of RA, studies have demonstrated that patients without overt cardiac disease exhibit increased EAT thickness. Increased EAT is linked to disease severity and systemic inflammation in RA patients. But EFV is not a strong diagnostic marker alone. Our study suggests that EFV assessment using non-invasive imaging modalities, such as CT, could provide valuable insights into risk in RA. Overall, the relationship between RA and EAT is a promising area of research that has the potential to uncover new insights into the pathogenesis of the disease and identify novel therapeutic targets. By understanding the role of EAT in RA, we may be able to develop more personalized and effective treatments for this debilitating disease. Incorporating EAT assessment into routine clinical practice may enhance risk stratification and facilitate early intervention, ultimately improving long-term prognosis in this high-risk patient population. In conclusion, Increased EFV is linked to disease severity and systemic inflammation in RA patients.

Limitations

Our research presents a number of limitations. First, this research was conducted at a single institution and employed a cross-sectional design, which limits our ability to establish causal relationships between EAT and the outcomes, making it impossible to generalize the findings. Additionally, the variability in EAT measurement techniques across different imaging modalities and analysis methods may introduce inconsistencies. Another limitation is the lack of functional assessment of cardiac performance. Furthermore, potential confounders such as concomitant medications, obesity, metabolic factors, and genetic predispositions were not extensively evaluated.

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