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Original Article

The Relationship Between Obesity Paradox and Inflammation Markers in STEMI Short: Obesity Paradox in STEMI

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Abstract

Objectives: Inflammation plays a very important role in the pathogenesis of coronary artery disease (CAD) and its prognosis. Especially; C-reactive protein (CRP) is associated with poor prognosis in patients with CAD. In this study, the relationship between CRP levels and body mass ındex (BMI) was investigated in patients who underwent primary coronary intervention (PCI) due to ST elevation myocardial infarction (STEMI).

Methods: Between January 2015 and February 2016, 132 patients who underwent PCI due to acute STEMI were included in the retrospective study. Patients were classified into two groups: (Group 1: BMI <25 kg/m^2 n=27 and BMI >35 kg/m^2 n=9, total: 36 patients; Group 2: 25 <BMI <30 kg/m^2 n=58 and 30 <BMI <35 kg/m^2 n=38, total 96 patients). Class 2, 3 obese patients and normal weight patients constituted Group 1 whereas pre-obese and Class 1 obese patients were included in Group 2. The patients are grouped in this way because the prognosis of the first group is worse in obesity paradox studies.

Results: There was no statistically significant difference between the two groups regarding demographic features, risk factors and left ventricular ejection fraction. CRP was significantly higher in group 1 (p=0.004). Among the inflammation markers, only CRP was significantly higher in Group 1.

Conclusion: CRP was found to be significantly lower in STEMI patients with 25 >BMI <35. Whereas, it was significantly higher in STEMI patients with 25 <BMI >35. One of the reasons for the better prognosis of mildly overweight and Class 1 obese patients with STEMI diagnosis may be the low values of CRP which has many effects on atherosclerotic plaque formation.

Keywords: C-reactive protein; obesity paradox; ST elevation myocardial infarction.

ST Elevasyonlu Mi Tanisi İle Başvuran Obez Hastalarda Obezite Paradoksunun Enflamasyon Belirteçleri İle Olan İlişkisi

Özet

Amaç: İnflamasyon, koroner arter hastalığının (KAH) patogenezinde ve prognozunda çok önemli bir rol oynar. Özellikle; C-reaktif protein (CRP), KAH'lı hastalarda kötü prognozla ilişkilidir. Bu çalışmada, ST yükselmeli miyokard enfarktüsü (STEMI) nedeniyle primer koroner girişim (PKG) geçiren hastalarda CRP düzeyleri ile Vücut Kitle İndeksi (VKİ) arasındaki ilişki araştırılmıştır.

Gereç ve Yöntem: Ocak 2015-Şubat 2016 tarihleri arasında akut STEMI nedeniyle primer koroner girişim uygulanan 132 hasta retrospektif çalışmaya dahil edildi. Hastalar iki gruba ayrıldı: (Grup 1: VKİ <25 kg/m², n=27 ve VKİ >35 kg/m², n=9, toplam: 36 hasta; Grup 2: 25 <VKİ <30 kg/m², n=58 ve 30 <VKİ < 35 kg/m², n=38,

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toplam: 96 hasta). Sınıf 2, 3 obez hastalar ve normal kilolu hastalar Grup 1'i oluştururken, pre-obez ve sınıf 1 obez hastalar Grup 2'yi oluşturdu. Hastalar bu şekilde gruplandırılmaktadır çünkü obezite paradoksu çalışmalarında ilk grubun prognozu daha kötüdür.

Bulgular: Demografik özellikler, risk faktörleri ve LVEF açısından iki grup arasında istatistiksel olarak anlamlı fark yoktu. İnflamasyon belirteçlerinden sadece CRP, Grup 1'de anlamlı olarak yüksekti (p=0,004).

Sonuç: CRP, VKİ: 25-35 kg/m² olan STEMI hastalarında anlamlı olarak daha düşük bulunmuştur. Buna karşın, VKİ: <25 kg/m² ve >35 kg/m² olan STEMI hastalarında anlamlı olarak daha yüksekti. Hafif kilolu ve sınıf 1 obez STEMI tanılı hastaların daha iyi prognozunun nedenlerinden biri, aterosklerotik plak oluşumu üzerinde birçok etkisi olan CRP'nin düşük değerleri olabilir.

Anahtar sözcükler: C-reaktif protein; obezite paradoksu; ST yükselmeli miyokard enfarktüsü.

Introduction

The prevalence of obesity has increased significantly worldwide, becoming a major health and social problem.[1,2] Obesity is associated with increased risks of hypertension, metabolic syndrome, and Type 2 diabetes mellitus, all strong risk factors for coronary artery disease (CAD).[3–5] Despite these adverse cardiovascular effects of obesity, numerous studies have revealed better cardiovascular outcomes in obese individuals which are defined as "obesity paradox."[6–11] The etiology of obesity paradox remains largely unexplained.

Weight that is higher than what is considered as a healthy weight for a given height is described as overweight or obese. Body mass ındex (BMI) is used as a screening tool for overweight or obesity. According to the World Health Organization; BMI was categorized as follows: Underweight (BMI <18.5 kg/m^2), normal (BMI 18.5 ≤24.9 kg/m^2), overweight (BMI 25 ≤30 kg/m^2), and obesity (BMI ≥30 kg/m^2). Obesity is classified as Class I for a BMI between 30 and 34.9 kg/m^2, Class II for a BMI between 35 and 39.9 kg/m^2, and Class III for a BMI ≥40 kg/m^2.

Inflammation plays a very important role in the pathogenesis of CAD and its prognosis.[12,13] Especially; many clinical studies indicate that C-reactive protein (CRP) is associated with poor prognosis in patients with CAD.[14,15]

In this study, the relationship between CRP levels and BMI was investigated in patients who underwent primary coronary intervention (PCI) due to ST-elevation myocardial infarction (STEMI).

Materials and Methods

Between January 2015 and February 2016, 132 patients who underwent PCI due to acute STEMI were included in the retrospective study. Informed consent was provided by all patients before their inclusion in the study. The confidential information of the patients was protected according to current national normative. The study protocol was approved by the Ethics Committee of University of Health Sciences Kartal Koşuyolu High Specialization Training and Research Hospital (date: April 16, 2024; no: 2024/08/785) and was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Acute coronary syndrome (ACS) with ST-segment elevation was defined as the presence of chest pain with persistent ST-segment elevation of at least 0.1 mV in at least two contiguous leads or a new left bundle-branch block.

Major exclusion criteria included cardiogenic shock, clinically significant hepatic disease, infection, patients who were followed up by non-PCI medical treatment, and CRP >10 mg/dL. This study evaluated demographic characteristics, risk factors and laboratory findings. Patients were classified into two groups: (Group 1: BMI <25 kg/m^2 n=27 and BMI >35 kg/m^2 n=9, total: 36 patients; Group 2: 25 <BMI <30 kg/m^2 n=58 and 30 <BMI <35 kg/m^2 n=38, total 96 patients). Class 2, 3 obese patients and normal weight patients constituted Group 1 whereas pre-obese and Class 1 obese patients were included in Group 2. The patients are grouped in this way because the prognosis of the first group is worse in obesity paradox studies. The characteristics of the patients consisted of medical history (diabetes mellitus, hypertension, hyperlipidemia, previous CAD, smoking, and family history) and laboratory findings (glucose, creatinine, cardiac enzymes, serum cholesterol, CRP, hemoglobin, hematocrit, leukocyte, lymphocyte, neutrophil, mean platelet volume, platelets, albumin, total protein, bilirubin, and left ventricular ejection fraction) (LVEF).

Statistical Analysis

Numerical variables were mean ± standard deviation; categorical variables were frequency and percentage. Patients were divided into two groups according to BMI. The Student's t-test was used to compare normal distribution variables and the Mann–Whitney U test was used to compare non-normal distributions. The Chi-square test was used to compare categorical variables. Patients were divided into four groups according to BMI and one way analysis of variance was applied to compare CRP values. Statistical Package for the Social Sciences 16.0 program was used for statistical analysis of the data in the study. p<0.05 was considered statistically significant for all tests.

Results

The demographic characteristics of 132 patients' (115 men, 17 women), risk factors, laboratory results and LVEF are listed Tables 1 and 2. There was no statistically significant difference between the two groups regarding demographic features, risk factors and LVEF. Total cholesterol (192.50±43.99; 175.30±41.22, p=0.044), hemoglobin (13.92±1.40; 13.23±1.98, p=0.026), hematocrit (42.42±4.19; 40.51±5.61, p=0.037), and triglyceride (179.59±99.13; 140.25±53.12, p=0.026) levels were significantly higher in Group 2 com-

Table 1. Baseline demographics and medical history of the study population

Patient characteristics	Group 1 BMI>35 and BMI<25 (n=36)	Group 2 35≥BMI>25 (n=96)	p
Age years	59.5±9.82	54.3±12.2	0.250
Gender, male, n	31	84	0.518
BMI (kg/m^2)	26.2±6.0	28.8±2.3	0.015
Diabetes mellitus, n	13	32	0.459
Hypertension, n	14	41	0.423
Dyslipidemia, n	12	40	0.252
Smoker (current), n	16	42	0.679
Smoker (ex), n	13	29	0.392
Chronic kidney disease, n	6	8	0.143
Previous CAD history, n	10	19	0.224
Family CAD history, n	14	50	0.124

BMI: Body mass index; CAD: Coronary artery disease.

Table 2. Laboratory results of the study population

Laboratory data	Group 1 BMI>35 and BMI<25 (n=36)	Group 2 35≥BMI>25 (n=96)	p
CRP (mg/dL)	1.88±2.14	0.75±0.81	0.004
Hemoglobin	13.23±1.98	13.92±1.40	0.026
Hematocrit	40.51±5.61	42.42±4.19	0.037
Leukocyte	12.86±3.12	12.29±3.78	0.423
Lymphocyte	1.96±0.87	1.94±0.92	0.906
Neutrophil	10.01±3.42	9.57±3.65	0.446
Mean thrombocyte volume	8.34±0.96	8.62±1.03	0.159
Thrombocyte	238.58±47.02	234.48±52.97	0.685
Neutrophil/lymphocyte ratio	7.07±5.79	5.98±3.28	0.295
LDL	112.36±35.49	121.08±38.15	0.236
HDL	36.11±7.59	38.23±8.85	0.204
Triglyceride	140.25±53.12	179.59±99.13	0.026
Total cholesterol	175.30±41.22	192.50±43.99	0.044
Glucose	163.47±63.27	168.97±84.01	0.722
Urea (mg/dL)	38.96±22.75	33.75±10.46	0.193
Creatinine (mg/dL)	0.99±0.62	0.81±0.24	0.115
Initial troponin	15.28±25.97	19.21±29.51	0.482
Bilirubin	0.64±0.32	0.60±0.32	0.512
Albumin	3.69±0.44	3.78±0.38	0.217
Total protein	6.43±0.59	6.42±0.48	0.943
LVEF	49.58±11.29	49.06±10.24	0.801

BMI: Body mass index; CRP: C-reactive protein; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; CAD: Coronary artery disease; LVEF: Left ventricular ejection fraction.

pared to Group 1. On the other hand, CRP was significantly higher in Group 1 (p=0.004) (Table 2). Among the inflammation markers, only CRP was significantly higher in Group 1. There was no statistically significant difference between the two groups in other markers.

Subgroup analysis was performed to assess CRP according to the patients' BMI. Patients were divided into four subgroups according to BMI: Subgroup 1 BMI <25 (n=27), subgroup 2 25 <BMI <30 (n=58), subgroup 3 30 <BMI <35 (n=38), and subgroup 4 BMI >35 (n=9). The mean CRP values of the subgroups are given in Table 3 and the statistical relations of the subgroups with each other are given in Table 4. The distribution of CRP values according to BMI is shown in Figure 1.

Table 3. CRP values of the subgroups

	Subgroup 1	Subgroup 2	Subgroup 3	Subgroup 4
CRP mg/dl	1.77±2.23	0.77±0.91	0.72±0.66	2.22±1.92

CRP: C-reactive protein; Subgroup 1: BMI<25; Subgroup 2: 25≤BMI<30; Subgroup 3: 30≤BMI<35; Subgroup 4: BMI≥35.

Table 4. Statistical relations of the subgroups with each other (p-value)

	Subgroup 1	Subgroup 2	Subgroup 3	Subgroup 4
Subgroup 1	–	0.017	0.022	0.848
Subgroup 2	0.017	–	0.999	0.027
Subgroup 3	0.022	0.999	–	0.027
Subgroup 4	0.848	0.027	0.027	–

Subgroup 1: BMI<25; Subgroup 2: 25≤BMI<30; Subgroup 3: 30≤BMI<35; Subgroup 4: BMI≥35.

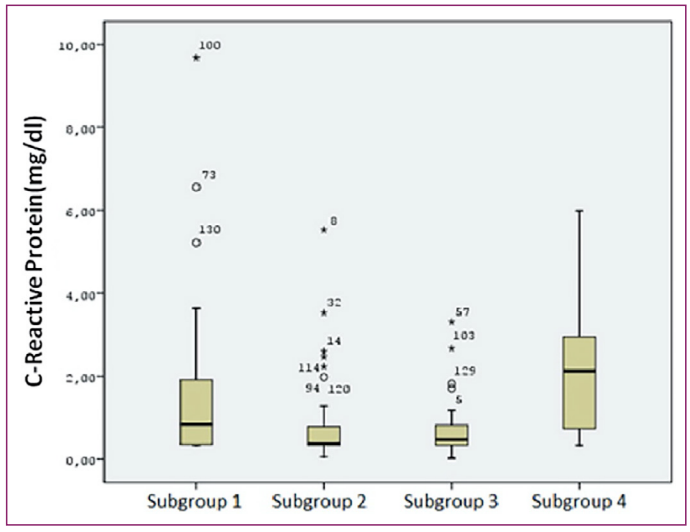

Figure 1. The distribution of C-reactive protein values according to body mass index.

Subgroup 1: BMI<25; Subgroup 2: 25≤BMI<30; Subgroup 3: 30≤BMI<35; Subgroup 4: BMI≥35.

Discussion

CRP was found to be significantly lower in STEMI patients with 25 >BMI <35. Whereas, it was significantly higher in STEMI patients with 25 <BMI >35. CAD is one of the most important causes of death in the world. Rupture of atherosclerotic plaques and plaque erosion in the coronary arteries cause ACS. CRP is an acute phase reactant that plays a role in atherosclerotic plaque formation and plaque rupture. In addition to CRP, inflammation markers such as leukocyte, fibrinogen, and ınterleukin (IL-6) have been associated with

cardiovascular events.[16] The reference hs-CRP value has been found to be associated with a poor prognosis when measured >3 mg/L in stable CAD and >10 mg/L in ACSs.[17] JUPITER and PROVE-IT clinical trials have shown that clinical outcome is better with low CRP in patients receiving statin therapy.[18,19] The relationship between inflammation and very weak and morbidly obese patients, who are reported to have poor prognosis for CAD, has been investigated in this clinical trial. In the obesity paradox of CAD, overweight patients and Type 1 obese patients have been shown to have a better prognosis than normal weight patients, Type 2 and 3 obese patients.[20–23]

CRP is an acute-phase protein produced in liver cells in response to IL-6 and Tumor necrosis factor α cytokines. It has been shown that CRP is also produced by the atherosclerotic intima layer.[24] It is highly sensitive, and may indicate non-specific inflammation, tissue damage, and infection. Increased risk of cardiovascular disease has been detected in patients with increased inflammatory markers such as CRP, leukocyte, fibrinogen, and IL-6.[25] One of the most investigated markers of inflammation in ACSs is the CRP.[26] CRP has many effects on atherosclerotic plaque formation.[27]

In our study, there was no statistical difference between the groups in terms of inflammation markers except CRP. Significantly different levels of CRP among the groups may indicate that CRP-mediated inflammation may be one of the causes of obesity paradox. In the subgroup analysis, the distribution of CRP levels in groups was similar to the U-shaped curve in previously reported obesity paradox studies.

It has been suggested that adiponectin released from adipose tissue may be cardioprotective with anti-inflammatory, anti-apoptotic, and anti-hypertrophic effects.[28–30] Obesity complications are expected to be less frequent in overweight and Type 1 obesity compared to Type 2 and 3 obesity. As a result, the protective effects of adiponectin may be expected to be more prominent in overweight and Type 1 obese patients. The anti-inflammatory effects of adiponectin may lead to decrease in CRP levels and suppression of CRP related tissue effects. One of the reasons for the worse survival rates of normal weight people may be the lack of cardioprotective effects of adipose tissue.

Limitations

The limitations of our clinical study were retrospective, single-centered, and small number of patients.

Conclusion

CRP was found to be significantly lower in STEMI patients with 25> BMI <35. Whereas, it was significantly higher in STEMI patients with 25 <BMI >35. One of the reasons for the better prognosis of mildly overweight and Class 1 obese patients with STEMI diagnosis may be the low values of CRP which has many effects on atherosclerotic plaque formation.

Disclosures

Ethics Committee Approval: The study was approved by the Kartal Koşuyolu High Specialization Training and Research Hospital Ethics Committee (no: 2024/08/785, date: 16/04/2024).

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