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Review Article / Derleme Makalesi

Acute Coronary Syndrome and Obesity Paradox

Akut Koroner Sendrom ve Obezite Paradoksu

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ABSTRACT

Acute coronary syndrome (ACS) is one of the most prevalent cardiovascular conditions worldwide, and although its etiologies are diverse, the underlying pathophysiological mechanisms are largely similar. In clinical practice, biomarkers such as cardiac troponins and myoglobin are critical for diagnosis and prognostic assessment, and they guide acute management. Importantly, modifiable risk factors and lifestyle behaviors substantially influence both the onset and the course of ACS; among these, dietary fattyacid composition, smoking, alcohol consumption, and physical activity are particularly significant. Specifically, diets with favorable fattyacid profiles and regular moderate physical activity have been associated with protective and therapeutic effects against ACS. Moreover, the relationship between obesity and ACS is complex, because large cohort studies have reported a Ushaped association between body mass index and mortality: both underweight and morbidly obese patients experience higher mortality, whereas overweight and mildly obese patients sometimes show lower mortality risk. Consequently, these findings highlight the heterogeneous impact of adiposity on ACS prognosis and underscore the need for individualized risk assessment and targeted lifestyle interventions.

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ÖZET

Akut koroner sendrom (AKS), dünya genelinde en yaygın kardiyovasküler hastalıklardan biridir ve etiyolojileri farklılık göstermekle birlikte, altta yatan patofizyolojik mekanizmalar büyük ölçüde benzerdir. Klinik uygulamada, kardiyak troponinler ve miyoglobin gibi biyobelirteçler tanı ve prognostik değerlendirme açısından kritik öneme sahiptir ve akut yönetimi yönlendirmektedir. Önemle belirtmek gerekir ki, değiştirilebilir risk faktörleri ve yaşam tarzı davranışları hem AKS'nin ortaya çıkışını hem de seyrini önemli ölçüde etkilemektedir; bunlar arasında özellikle diyetin yağ asidi bileşimi, sigara kullanımı, alkol tüketimi ve fiziksel aktivite dikkat çekmektedir. Özellikle, yağ asidi profili açısından elverişli diyetler ve düzenli, orta düzeyde fiziksel aktivitenin AKS'ye karşı koruyucu ve tedavi edici etkilerle ilişkili olduğu gösterilmiştir. Ayrıca, obezite ile AKS arasındaki ilişki karmaşıktır; zira geniş kohort çalışmalarında, vücut kitle indeksi ile mortalite arasında U-şekilli bir ilişki bildirilmiştir: hem düşük kilolu hem de morbid obez hastalar daha yüksek mortalite riskine sahipken, fazla kilolu ve hafif obez hastalarda bazen daha düşük mortalite riski görülmektedir. Bu bulgular, adipozitenin AKS prognozu üzerindeki heterojen etkilerini vurgulamakta ve bireyselleştirilmiş risk değerlendirmesi ile hedefe yönelik yaşam tarzı müdahalelerinin gerekliliğini ortaya koymaktadır.

1. Acute Coronary Syndrome

Acute Coronary Syndrome refers to all of the clinical manifestations caused by myocardial ischemia as a result of reduced coronary artery blood flow. Acute Myocardial Infarction (AMI), unstable angina pectoris, and sudden cardiac death are all conditions that can occur as a result of an abrupt worsening in myocardial blood supply. Even though it is not always possible to distinguish clearly between acute myocardial infarction and unstable angina pectoris, enzymes that demonstrate myocardial necrosis have been proven to be beneficial(Lip et al., 2010; Cannon et al., 2013).

Pathophysiologically, acute coronary syndrome (ACS) is defined as an acute atherothrombotic event within an atheroma plaque. The event is triggered by injury to a coronary plaque. This injury often does not cause critical lumen narrowing but is problematic because of a thin fibrous cap, large lipid core, and intense inflammatory activity. The damaged plaque loses its endothelial covering; when this antithrombotic layer is lost, circulating platelets are exposed to subendothelial collagen and von Willebrand factor. Sequential reactions on this thrombogenic substrate lead to accumulation of platelets, fibrin, and erythrocytes, which may occlude the coronary artery lumen (Edmondson et al., 2012).

The most common symptom of ACS is thought to be a compressive chest pain felt in the middle of the chest, radiating to both shoulders, arms, back, upper abdomen and lower jaw (Crea & Liuzzo, 2013).

1.1. Classification of Acute Coronary Syndrome

Acute Coronary Syndrome includes ST Elevation Myocardial Infarction (STEMI), Non-ST Elevation Myocardial Infarction (NSTEMI) and Unstable Angina Pectoris (USAP) (Nikus et al., 2010).

1.1.a ST Elevation Myocardial Infarction (STEMI)

A rupture of a plaque in one of the major coronary arteries results in the formation of an occlusive thrombus that cannot be compensated for by collateral circulation. As a result, complete occlusion occurs (Chang et al., 2018).

1.1.b Non-ST Elevation Myocardial Infarction (NSTEMI)

It is a clinical condition characterized by the presence of positive clinical indicators reflecting myocardial necrosis without ST-segment elevation, electrocardiographic ST-segment depression, and/or significant change in T waves, with appropriate clinical conditions developing on the background of coronary artery disease (Nikus et al., 2010).

1.1.c Unstable Angina Pectoris (USAP)

It is a clinical condition that cannot be precisely localized in the chest or arms, originates deep, is rarely defined as pain, feels more like heaviness or pressure, is associated with physical activity or emotional stress, and resolves in 5-25 minutes with rest or sublingual nitrate intake (Nikus et al., 2010).

1.2. Pathogenesis of Acute Coronary Syndrome

The number of studies on the pathogenesis of ACS has increased in recent years. Angiographic and angioscopic studies indicate that rupture of an atherosclerotic plaque is a common mechanism in ACS formation (Makki et al., 2015). Atherosclerotic coronary artery disease begins in childhood and usually progresses insidiously for many years without symptoms. Symptoms typically start when an atheroma enlarges enough to limit blood flow in the lumen; this clinical picture is called Stable Angina Pectoris (SAP). Alternatively, plaque rupture with subsequent thrombus formation can partially or completely occlude the coronary lumen. Clinical manifestations then range from unstable angina to myocardial infarction (MI), depending on the degree of thrombotic occlusion. The clinical outcome also depends on many factors: plaque composition, extent of rupture, local hemodynamics and coagulation status at the time of rupture, treatments applied, and whether the resulting thrombus fully occludes the vessel. Finally, unstable angina and non–STelevation myocardial infarction (NSTEMI) are characterized by an imbalance between myocardial oxygen supply and demand (Otsuka et al., 2014).

Atherosclerosis is a disease characterized by the formation of intimal plaques that contain lipids, fibroblasts, macrophages, smooth muscle cells, and extracellular matrix in varying proportions, leading to progressive arterial stenosis or occlusion and loss of arterial compliance and antithrombotic function (Libby, 2021). Coronary artery disease (CAD) is the main cause ACS; ACS is a clinical manifestation of CAD. Monocytes adhere to endothelial cells via adhesion molecules and take up oxidized lowdensity lipoprotein (LDL). This process produces foam cells and, macroscopically, the earliest lesion known as a fatty streak. Smooth muscle cells migrate into the intima and adopt a phenotype that produces a fibrous cap. Over time macrophages and lipid/cholesterol crystals accumulate within the plaque (Libby, 2021). The endothelial surface over an advanced plaque may erode or rupture, exposing the thrombogenic core. Thrombus formation at the site of plaque disruption can then precipitate ACS (Romagnoli et al., 2012).

2. Biochemical Indicators in Acute Coronary Syndrome

2.1. Myoglobin

Myoglobin is an iron-containing protein with a low molecular weight. It is released into the circulation by damaged myocardial cells and is detectable in the blood ½-2 hours after the onset of AMI. The duration of myoglobin in serum is 12-18 hours. It is found in both heart and skeletal muscle and tissue; It is thought to have a minor role in the diagnosis of AMI. Clinical studies have demonstrated that determining myocardial necrosis using more specific markers and myoglobin may be useful for the early identification of AMI (Scirica, 2010; Wu et al., 2018).

2.2. Creatine Kinase

Creatine kinase (CK), which is an essential enzyme of muscle metabolism, reversibly catalyzes the phosphorylation of creatinine via Adenosine Triphosphate (ATP). Creatine kinase isoenzymes are dimeric molecules formed by the combination of B and M chains. Therefore, CK has 3 isoenzymes as follows; CK-MM, CK-MB, CK-BB. BB form is found in the brain and kidneys. Even though skeletal muscles contain mostly MM form, 1-2% also contain MB form. There are both MB and MM forms in the heart muscle. Following AMI, the affected muscle releases Creatine Kinase-MB into the circulation. It is known that Creatine Kinase begins to be released approximately 2-4 hours after AMI, peaks at 24 hours and returns to normal after 36-72 hours (Wu et al., 2018; Timmis, 2015).

2.3 Cardiac Troponins

Troponins regulate the interaction between actin and myosin. Within the myocyte cell, cardiac troponins are found in two pools. The first of these is the pool where they are free in the cytosol, and these troponins are released into the plasma following myocardial damage. This first pool contains 3-5% of the total troponins, and because the concentration is so low, the amount that enters the plasma during the early phase is also quite low. On the other hand, the second pool, which is connected to the contractile structure, contains a significantly higher concentration of troponin and releases it much more slowly into the plasma. Troponin levels may remain increased for an extended period of time following cardiac injury because of this second pool. The two isoforms, troponin T and I, begin to rise within 3-12 hours of ischemia. It peaks at 12-24 hours. Troponin T may remain at high levels for 8-21 days, and troponin I for 7-14 days. High levels were found to be correlated with the extent of pathologically detected myocardial necrosis (Ramasamy, 2011; Dekker et al., 2010).

In summary, cardiac troponins are the most sensitive and specific markers for the diagnosis of MI. Cardiac troponins are capable of detecting necrosis in a very small area. While creatine kinase-MB is not as specific as cardiac troponins, it is an additional useful test for diagnosis. Myoglobin alone is not suggested for diagnostic purposes (Moe & Wong, 2010).

3. Risk Factors in Acute Coronary Syndromes and Lifestyle Modifications

3.1. Fatty Acid Pattern of Dietary Content

Nutritional habits play a significant role in the development of ACS. Dietary habits have an effect on the development of atherosclerosis and CAD via the effects of dietary fatty acids and cholesterol on plasma LDL-C levels. Saturated fatty acids in the diet increase LDL-C, whereas poly and monounsaturated fatty acids reduce LDL-C. However, it is believed that the risk of CAD associated with diet is more complex, as different fatty acids may have thrombogenic or antithrombogenic qualities. Along with dietary fatty acids, dietary fiber and carbohydrates derived from vegetables have been shown to act as protective factors (Yadav et al., 2010; Varela et al., 2020).

It was hypothesized that oxidative modification of lipoproteins in the arterial wall contributes to atherogenesis. Observational population studies suggested that diets high in antioxidantrich foods (for example, those containing alphatocopherol, betacarotene, and vitamin C were associated with a lower risk of coronary artery disease (CAD). However, randomized interventional trials of antioxidant vitamin supplementation did not show a reduction in CADrelated mortality or overall cancer incidence (Haque et al., 2022; Stein et al., 2021). Subsequent longterm trials have reported similar negative findings: dietary supplementation with antioxidant vitamins failed to reduce cardiovascular morbidity, mortality, or cancer development. In one randomized study, alphatocopherol and betacarotene supplementation provided no benefit in smoking men with angina pectoris (Monzani et al., 2018).

Two randomized interventional studies of diet for secondary prevention after myocardial infarction (MI) demonstrated significant reductions in total mortality when the dietary fattyacid composition was changed. In the first study, the intervention group was recommended to take 0.3 g/day of eicosapentaenoic acid (EPA), while the control group consumed 0.1 g/day of EPA and oily fish; this intervention produced a 29% reduction in overall mortality within two years after MI. In the second study, MI patients were advised to follow a Mediterraneanstyle diet rich in alphalinolenic acid (ALA). Over five years, the ALArich diet was associated with

a 70% reduction in overall mortality, a 76% reduction in cardiac mortality, and a 73% reduction in coronary events. Serum lipids, blood pressure, and body mass index remained similar in both groups throughout the studies (Buscemi et al., 2014; Sanchis-Gomar et al., 2016).

3.2 Smoking

Smoking is a significant, independent, and modifiable risk factor for the development and progression of acute coronary syndrome (ACS). Substances such as nicotine and carbon monoxide have toxic effects on the cardiovascular system. Nicotine raises arterial blood pressure and heart rate by stimulating catecholamine release and the sympathetic nervous system. The risk of myocardial infarction (MI) and cardiac death is 2.7 times higher in male smokers and 4.7 times higher in female smokers than in nonsmokers. Smoking lowers HDLC levels and damages the arterial endothelium by increasing LDLC oxidation and platelet aggregation. Smoking accounts for approximately 30% of cardiovascular deaths. After an ACS event, smoking cessation reduces the risk of MI and mortality; risk approaches that of nonsmokers after about three years (Snaterse et al., 2015; Notara et al., 2015). Overall, smoking increases the risk of coronary artery disease by 2–4 times and is implicated in a large proportion of CADrelated deaths and sudden cardiac deaths. There is a roughly 50% reduction in the risk of death from CAD within the first year after smoking cessation (Snaterse et al., 2015; Notara et al., 2015).

According to the Heart Diseases and Risk Factors Study in Turkish adults, smoking is the most common risk factor in the country. Individuals who smoked more than ten cigarettes per day had a 1.7fold greater risk of coronary events and a 2–2.5fold increased risk of mortality from any cause. In Türkiye, men's smoking prevalence is declining while women's is increasing; given the high CAD mortality among Turkish women compared with other European countries, this trend is particularly concerning. In addition to active smoking, environmental exposure to cigarette smoke (passive smoking) is now recognized as a modifiable cardiovascular risk factor (Andsoy et al., 2015).

3.3 Alcohol Consumption

Epidemiological studies demonstrate that low to moderate drinkers have a 40-50% lower risk of AMI and CAD compared to non-drinkers or heavy drinkers (Mathews et al., 2015). Low alcohol consumption tends to increase plasma HDL-C, which may account for part of alcohol's protective effect. On the other hand, alcohol has an inhibitory effect on platelet aggregation and a reducing effect on fibrinolytic factors. Increased alcohol consumption, on the other hand, can result in a rise in blood pressure and an increased risk of developing cerebrovascular disease. Alcohol and blood pressure have a more complicated relationship than appears. Alcohol and blood pressure studies reveal the presence of a J-shaped association, with moderate drinkers having the lowest blood pressure. Heavy drinkers have the highest blood pressure levels (Toma et al., 2017; Goel et al., 2018).

3.4 Physical Activity

A sedentary lifestyle is an important risk factor for ACS. Compared with individuals who do not exercise regularly, the death rate from CAD is twice as high. Regular exercise has been shown to be associated with a lower risk of CAD among a variety of groups of workers in the United States and England, and this association was observed regardless of smoking, obesity, blood pressure, or a family history of CAD. Apart from weight loss and blood pressure reduction, exercise increases HDL-C, decreases blood TG, and decreases insulin resistance (Ter Hoeve et al., 2015). Maintaining a healthy level of physical activity and performance can operate as a direct protective factor against coronary heart diseases, regardless of other risk factors. Meta-analyses of randomized studies of cardiac rehabilitation in patients with acute myocardial infarction, including those that incorporate an exercise program as part of a multifactorial rehabilitation strategy, have demonstrated that cardiac rehabilitation can result in a 20%–25% reduction in total mortality and cardiovascular mortality. Aerobic exercises lasting 30 minutes (150 min/week) 4-5 times a week are recommended (Huffman et al., 2016). Regular exercise prevents obesity or causes weight loss, and lowers blood pressure and cholesterol. Physical exercise has been demonstrated to help reduce the development and progression of atherosclerosis by lowering risk factors in CAD (Pirhonen et al., 2017).

4. Obesity Paradox

Uretsky et al. (2007) followed 22,576 patients with coronary artery disease and hypertension for two years and reported a Ushaped relationship between body mass index (BMI) and cardiovascular mortality. Both underweight and morbidly obese individuals had higher mortality risk, whereas normalweight and overweight individuals had lower mortality. The authors suggested this pattern may relate to lower systemic vascular resistance and lower plasma renin activity in obese patients with hypertension (Uretsky et al., 2007).

In a large metaanalysis pooling cohorts followed for three to eight years, the association between BMI and mortality in patients with coronary artery disease was examined. The relationship between obesity and total mortality was inconsistent across studies, and similar heterogeneity was seen for cardiovascular events. Overall, the analysis found higher cardiovascular and total mortality among patients with low BMI, whereas overweight and mildly obese patients (for example, overweight or Class I obesity by BMI categories) had lower mortality risk compared with those with low BMI (Bucholz et al., 2012; Romero-Corral et al., 2006).

Oreopulos et al. (2007) conducted an observational meta-analysis study in which 28,209 patients diagnosed with ACS were followed up for 2.7 years. According to the study, those who are slightly overweight or obese and have been diagnosed with ACS have a lower mortality rate than those who are underweight (Oreopoulos et al., 2008). Several hypotheses have been proposed to account for the paradoxical observations linking higher BMI with more favorable short-term outcomes after ACS. One explanation is that patients with greater adiposity possess larger metabolic reserves, which may confer resilience during the catabolic stress of acute illness and thereby reduce early mortality. In addition, adipose tissue secretes a range of adipokines and cytokines that can exert complex, sometimes cardioprotective, effects; for example, increased circulating levels of soluble tumor necrosis factor (TNF) receptors and certain anti-inflammatory adipokines have been suggested to buffer deleterious inflammatory cascades. Hemodynamic and neurohormonal adaptations have also been implicated: obese individuals often exhibit lower circulating atrial natriuretic peptide concentrations and altered sympathetic and renin–angiotensin system activity, which could modify cardiovascular responses in the acute setting. Furthermore, clinicians may prescribe or tolerate higher absolute doses of cardioprotective medications in heavier patients, and differences in pharmacokinetics may influence drug distribution and apparent therapeutic effect. However, these proposed mechanisms must be interpreted cautiously because most observational studies rely on BMI or total body weight as the principal measure of adiposity, whereas central adiposity typically assessed by waist circumference or waist-to-hip ratio—is a stronger predictor of cardiometabolic risk and may have distinct associations with outcomes after ACS. Consequently, relying solely on BMI can mask heterogeneity in fat distribution and metabolic health (for example, metabolically healthy obesity versus sarcopenic obesity), and may lead to residual confounding by factors such as fitness level, smoking status, comorbidities, and inflammatory burden. For these reasons, future research should incorporate more granular phenotyping (including measures of visceral adiposity, body composition, functional status, and biomarkers of inflammation and neurohormonal activation) and apply robust statistical methods to address confounding and reverse causation. Clinically, these limitations underscore the need for individualized risk assessment rather than simplistic reliance on BMI when prognosticating after ACS (Niedziela et al., 2014; Costanzo et al., 2015).

5. Conclusion and Perspectives

Acute coronary syndrome is an important cause of disability and death worldwide. Diagnosis of acute coronary syndrome in electrocardiography can talk about different classifications. It is emphasized that the most important laboratory parameters in the diagnosis of acute coronary syndrome are myoglobin and cardiac troponins. Modifiable risk factors, which are risk factors that can lead to acute coronary syndrome, factors such as diet fatty acid pattern, physical activity, smoking habits affect the prevention and treatment process of the disease. The relationship between obesity and acute coronary syndrome has been tried to be explained by the obesity paradox. In studies with large samples, it is stated that moderate obesity reduces death from acute coronary syndrome. Although there are contradictions in the results of the study, it has been emphasized that cachectic or extremely obese individuals are at the same risk

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