Smoking Behavior in ST-Elevation Myocardial Infarction: a Risk Factor or Protective Predictor?

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Abstract

ST-elevation myocardial infarction (STEMI) is one of the most serious developments of coronary artery disease. This condition receives a lot of attention because it affects a lot of people, especially those with underlying cardiac diseases. STEMI can lead to several cardiac complications, such as transmural myocardial injury and obstruction of the heart microvasculature, which can result in clinical heart failure. One of the major modifiable risk factors of cardiovascular diseases is smoking. Many studies reported that this behavior can increase the risk of STEMI, especially in some populations. Smoking can cause damage to the coronary microvascular components, hence the infarct of related cardiac tissues. However, there were several findings that considered smoking as a protective factor for STEMI patients. This review article aims to evaluate the role of smoking behavior in STEMI cases from prior studies.

Keywords: STEMI; coronary artery disease; cardiac complication; smoking; heart failure

1. Introduction

Cardiovascular diseases are known to be primary causes of death across the globe. In 2017, 31% of the global death cases were from cardiovascular causes. One-third of the total deaths in the United States were also caused by cardiovascular diseases. Affecting more than 17.9 million people in the world per year, cardiovascular diseases remain to get lots of attention in the medical field[1–3].

The morbidity and death rate of acute myocardial infarction is comparatively high when compared to other cardiovascular disorders. One of the five main signs of coronary artery disease is myocardial infarction; the other four are heart failure, unstable angina, stable angina, and sudden death. Based on the electrocardiography (ECG) presentation, myocardial infarction is typically categorized into two forms in clinical settings: non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). Because of the greater obstruction in the afflicted coronary arteries, STEMI is regarded as the more dangerous of the two infarctions[2].

Epidemiologically, based on the data released by Jakarta Acute Coronary Syndrome Registry, in Indonesia there were 654 cases of STEMI in 2008-2009. In European nations, the incidence of STEMI ranges between 43 to 144 per 100.000 people in a year period. A similar incidence rate is also found in the United States, showing 50 STEMI cases per 100.000 people in a year[4].

There are two main categories of risk factors for STEMI: modifiable and nonmodifiable. Non-modifiable risk factors include age, sex, and a family history of heart disease, whereas modifiable risk factors include smoking, dyslipidemia, hypertension, obesity, lack of physical exercise, and alcohol use. Smoking is regarded as a significant factor among these due to the severe harm it causes to the vascular system. Nonetheless, because smoking is a behavior that can be changed, it is known that the vascular dysfunctions it causes are preventable. The purpose of this paper is to evaluate earlier research about smoking behavior and STEMI patients[5, 6].

2. ST-Elevation Myocardial Infarction (STEMI)

The absence of oxygen flow to the heart muscles, or myocardium, is the clinical state that forms the basis of the general pathophysiology of STEMI. Coronary artery obstruction is a common cause of this disease. The corresponding cardiac tissue will eventually perish if this inadequate oxygen supply is not corrected. Acute coronary syndrome is the term used to describe the entire clinical event that is occurring, whereas myocardial infarction refers to the death of the heart. A STEMI diagnosis can be made if the electrocardiogram reveals an elevation of the ST segments and the results of myocardial necrosis biomarker testing are positive[7–9].

Heart failure is typically regarded as one of the most serious cardiac problems associated with STMI. The failure of heart pumps and pathological remodeling of the cardiac tissue resulting from transmural damage and/or microvascular blockage (particularly affecting the anterior wall) induced by STEMI can produce clinical heart function failure. Mitral regurgitation is another serious complication of STEMI that typically happens in the subacute period. Mitral regurgitation in STEMI may be strongly associated with dilation of the left ventricle, issues with the chordae tendineae and papillary muscles. Systolic murmurs, pulmonary congestion, or severe dyspnea are the typical symptoms of myocardial infarction[8].

3. Smoking as a STEMI Risk Factor

As was previously noted, one of the main risk factors for STEMI that can be prevented is smoking. Smoking can cause vascular dysfunctions that result in STEMI. Cigarette smoke contains substances that have the potential to decrease blood levels of nitrite oxide (NO) and increase adhesion molecules, both of which can lead to endothelial dysfunction. Additionally, smoking can exacerbate certain inflammatory processes. Smoking can raise platelet counts and macrophages, which can lead to a pro-coagulative and pro-inflammatory environment. Smokers also frequently exhibit tissue remodeling, which might result in atherosclerosis processes. In addition to causing the generation of oxygen radicals, smoking is the primary cause of coronary artery vasospasm [6, 10].

To be more precise, cigarettes include three main ingredients that raise the risk of cardiovascular disease. Nicotine, carbon monoxide (CO), and oxidative gasses are these chemicals. Nicotine raises the risk of cardiovascular disease via impairing insulin resistance, lipid metabolic problems, and endothelial dysfunction. Blood viscosity can rise in response to elevated carbon monoxide levels, which can induce the body to create more red blood cells and increase the risk of hypertension and cardiovascular disorders. By reducing the quantity of endogenous antioxidants, oxidative gasses can cause endothelial dysfunction, inflammation, and oxidation of low-density lipoprotein (LDL) and platelets[11].

Most STEMI patients were active smokers when diagnosed or admitted to inpatient care. A prior study in Abu Dhabi, United Arab Emirates by Callachan and colleagues showed that 61% of STEMI patients had a history of smoking upon admission [12]. Several other studies also reported similar results. In Sri Lanka, a cross-sectional study reported that 55.8% of STEMI patients were active smokers [13]. Another study from Semarang, Indonesia showed that 48.9% STEMI patients had a smoking history [14].

For patients who were already diagnosed with STEMI, results from a recent study stated that smoking is associated with a worse prognosis after undergoing percutaneous coronary intervention (PCI) [15]. In relation to mortality, the Center for Disease Control and Prevention (CDC) declared that smoking is not only a major cause of cardiovascular diseases, but is also the cause of one in four deaths from cardiovascular diseases. The same source stated that 1.9 million people died from tobacco-induced heart disease [16].

A study from 2021 reported that in STEMI patients, smokers had a higher risk of myocardial infarction recurrency [17]. In reperfused STEMI patients, a prior European study found that smokers are more likely to

develop intramyocardial hemorrhage, which is a marker of coronary microvascular damage that causes the erythrocytes to extravasate. The same study also revealed that coronary microcirculation in smokers was more prone to injuries caused by reperfused ischemia [18].

4. Smoker's Paradox in STEMI Cases

Surprisingly, although many studies reported that smoking can increase the risk of STEMI, a significant number of studies stated that smoking can be a relatively good factor for the outcomes of STEMI therapy. This phenomenon is known as the smoker's paradox (or pseudoparadox).

A Singaporean study involving 12,307 STEMI patients reported that smokers had better clinical outcomes after undergoing primary percutaneous coronary intervention (pPCI). However, the fact that more than 90% of smokers in the study population are male could be a potential confounding factor [17].Similar findings were found in a related study conducted by Rakowski and colleagues with patients from Europe. In smoker and nonsmoker STEMI patients, the study evaluated pPCI clinical outcomes and risk profiles (low-risk or high-risk). According to study findings, smokers had superior angiographic PCI outcomes. Based on risk profiling, smoking patients were frequently placed in the low-risk group. Less evidence of prior myocardial infarction, renal failure, PCI, diabetes, anterior wall STEMI, and multivessel illnesses was present in them. [19]. A study from 2019 assessed the 30-day mortality in STEMI patients and the results confirmed the association between smoking activity with better prognosis after pPCI [20].

There is ongoing debate over the existence of the smoker's dilemma and disagreement over the precise mechanism underlying it. Nonetheless, a number of research discovered a connection between this phenomena and smokers' demographics and inflammation. According to a study by Somaschini and colleagues, smokers frequently have a persistent inflammatory state, which preconditions the coronary capillaries. This preconditioned environment reduces the synthesis of platelet activation and inflammatory cell chemotaxis when inflammation occurs, which lowers the acute inflammatory response[20]. Other studies reported that most STEMI patients who are smokers tend to be younger, thus having less comorbidities. STEMI patients with active smoking status are also mostly male, and a study revealed that smoking was associated with a greater risk for STEMI in women than men [21].

5. Conclusions

Despite being a major risk factor for ST-elevation myocardial infarction (STEMI), some studies revealed that smoking can be a protective factor in some clinical situations. Inflammatory preconditioning and the demography of smoker STEMI patients plays a role in this phenomenon.

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