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A LITERATURE REVIEW ON ST-SUPRA AMI, SMOKING AND DIABETES MELIT

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Abstract: Goal: To evaluate the prevalence of smoking in patients who have suffered acute myocardial infarction with ST-segment elevation. **Results:** The findings of this study suggested that predictors of plaque erosion varied with different smoking status in patients with STEMI and broaden clinicians' understanding of clinical and lesion predictors of plaque erosion, especially in relation to smoking. **Conclusion:** In current smokers, diabetes mellitus was negatively associated with plaque erosion compared with plaque rupture. In nonsmokers, higher MLA and close bifurcation were positively related to plaque erosion, but not to plaque rupture. Compared to plaque rupture, the correlation between plaque erosion and current smoking complements clinicians' understanding of plaque erosion. There was a strong indication of the relationship between smoking and the development of STEMI.
Key words: Smoking. Cardiovascular diseases. Myocardial infarction with ST-segment elevation. Necrosis. Angioplasty. Coronary Disease.

INTRODUCTION

Tobacco use has become one of the main risk factors for chronic non-communicable diseases, making it responsible for about 8 million deaths a year. And despite being the leading preventable global cause of morbidity and mortality, an estimated 1.25 billion people are smokers.[1,2] Currently, mortality among smokers is 2 to 3 times higher than in people who have never smoked.[8]

In Brazil, cardiovascular changes are one of the main damages resulting from smoking. There are several mechanisms for cardiac dysfunction, including: endothelial dysfunction, reduction of HDL cholesterol, increased oxidation of LDL cholesterol, increased levels of adhesion molecule and fibrinogen, increased vascular spasm and

increased platelet aggregation. In addition, changes in systemic blood pressure and heart rate can be explained by the exacerbated adrenergic response secondary to increased plasma epinephrine and norepinephrine due to tobacco consumption.

According to the available epidemiological evidence, smoking is the cause of about 50 diseases, of which cardiovascular, respiratory and cancer stand out. About 45% of deaths are from coronary heart disease (AMI), 85% from Chronic Obstructive Pulmonary Disease (COPD), 25% from cerebrovascular disease and 30% from cancer are related to the consumption of tobacco products.[1]

Acute myocardial infarction (AMI) initially occurs with the rupture or erosion of a lipid-laden, atherosclerotic coronary plaque, leading to exposure of the circulating blood to thrombogenic core and matrix materials in the plaque. A fully occluded thrombus results in ST-segment elevation, whereas partial occlusion or the presence of collateral circulation is defined as AMI without ST-segment elevation or unstable angina. [9]

JUSTIFICATION

The number of smokers is still very expressive, and since it is an important factor that causes AMI, the topic has an important relevance in relation to the primary prevention of cardiovascular events. This research provides information for multidisciplinary teams to improve care in this segment, in order to reduce the deleterious effects of cigarettes on the heart and encourage the fight against tobacco.

GOAL

To evaluate the prevalence of smoking in patients who suffered an Acute Myocardial Infarction with ST-segment elevation.

RESULTS

The main findings of the present study were the following: common predictors, including age <50 years, absence of dyslipidemia and single-vessel disease were independently related to culprit plaque erosion, regardless of smoking; the presence of diabetes mellitus significantly increased the risk of rupture-based STEMI but may not reduce the risk of plaque erosion-based STEMI in current smokers; and near bifurcation and higher MLA were associated with plaque erosion in non-smokers, but not in current smokers.

Plaque erosion causing the onset of STEMI had common predictors in both current and non-current smoking groups, including age <50 years, absence of dyslipidemia, and single-vessel disease. In line with our study, other OCT studies showed that age, dyslipidemia, and multivessel disease were independently associated with plaque erosion in patients with ACS. Dyslipidemia and multivessel disease were less frequent with plaque erosion, but younger age was associated with plaque erosion regardless of smoking. Previous pathology studies have reported that acute rupture is more frequent in sudden cardiac death in patients >50 years, while plaque erosion is more common in those aged <50 years. Dyslipidemia is a predictor of acute coronary thrombosis, and ruptured plaque formation is an inflammatory process related to lipid deposition. Autopsy studies revealed that a lower level of CT and a lower CT/HDL-C ratio are specifically associated with plaque erosion compared to plaque rupture. These findings are consistent with our finding that the absence of dyslipidemia was associated with plaque erosion. The angiographic finding of plaque erosion was more frequent in single-vessel disease, and patients with plaque erosion had less complex angiographic features both at the three-vessel coronary level and at the level of the culprit lesion.

Diabetes Mellitus Increase in STEMI based on plaque rupture in current smokers, but not in nonsmokers. The presence of diabetes mellitus significantly increased the risk of STEMI based on rupture but may not have reduced the risk of STEMI based on plaque erosion in the current smoking group. Diabetes mellitus is associated with accelerated coronary atherosclerosis due to the marked proinflammatory and prothrombotic state induced by the associated metabolic abnormalities. OCT evidence suggests that diabetes mellitus can predict TCFA, the precursor to plaque rupture. In addition, smoking induces insulin resistance and hyperinsulinemia. Previous intravascular imaging studies found that increased insulin resistance and smoking-induced hyperinsulinemia were associated with LRPs, which increases the occurrence of plaque rupture. The combined action of smoking of diabetes mellitus contributes to current smokers with diabetes mellitus having a high risk of plaque rupture. These findings support our finding that diabetes mellitus increased the risk of rupture-based STEMI but may not have reduced the risk of plaque erosion-based STEMI in the current smoking group.

Close bifurcation and greater MLA independently associated with plaque erosion in non-current smokers but non-current smokers. As local anatomical factors, close bifurcation and greater MLA promoted erosion-based STEMI in nonsmokers, but were not associated with erosion-based STEMI in current smokers. These findings suggest that hemodynamic factors play a major role in the formation of plaque erosion in nonsmokers.

For current smokers, smoking as a chemical factor contributing to the systemic effects can lead to an imbalance in the supply and demand of oxygen in the coronary arteries, leading to hypoxia, which can damage endothelial cells and consequent thrombosis, causing plaque erosion. This may be the reason why close bifurcation and greater MLA did not have a significant effect on plaque erosion in current smokers. For non-smokers, our study found that local hemodynamic factors, including proximal bifurcation and major MLA, were related to plaque erosion but not plaque rupture. This discrepancy may be due to different thrombus formation between current smokers and non-smokers.

The close bifurcation promoted erosion-based STEMI in nonsmokers, but was not associated with plaque erosion in current smokers. Plaque erosion requires the combined action of hemodynamic disturbances and plaque components. Intravascular imaging studies have identified the proximal bifurcation as an important local hemodynamic factor of plaque erosion. Recently, it has been shown that culprit lesions with an intact fibrous cap are characterized by lower lipid content, less calcification, a thicker overlying fibrous cap, and location close to a coronary bifurcation compared to culprit lesions with a ruptured fibrous cap, which is similar to our results. A close bifurcation will increase major vessel endothelial stress and further accelerate coronary endothelial injury, leading to thrombosis and plaque erosion. Furthermore, local shear stress in vivo directly influences endothelial cell apoptosis in plaques associated with oscillatory shear stress downstream of the plaques, where plaque erosion tends to occur. Furthermore, unlike plaque rupture which is primarily associated with TCFAs, plaque erosion is not only typically associated with fibrous plaque but also with lipid plaque., including LRP and TCFA. Hemodynamic changes around a nearby bifurcation accelerate the development of LRP. However, LRP formation is closely related to hemodynamic disturbances and coronary risk factors such as dyslipidemia,

diabetes mellitus, hypertension, and CKD, which are rarely seen in plaque erosion, and not all LRPs are precursors to plaque rupture. In the PROSPECT study, only <5% of TCFAs actually provoked a clinical event at a follow-up of 3.4 years. 46 A recent study reported that the expression of hyaluronidase 2 (HYAL2, an enzyme that degrades hyaluronan), which is associated with plaque erosion in OCT, is significantly increased in smokers. HYAL2 was not expressed as much in non-smokers, but local hemodynamic factors, such as nearby bifurcation, may play an important role in the formation of plaque erosion in non-smokers.

The present study showed that in non-current smokers with STEMI, a higher MLA was associated with culprit plaque erosion rather than plaque rupture. A culprit vessel with higher MLA often had fibrous plaques and were more prone to erosion; however, plaque rupture occurred mainly in lesions with a high lipid load and a thin fibrous cap. With lipid deposition, lipid components gradually accumulate in the coronary artery tree, the diameter of the lumen gradually decreases, although the plaque rupture appears to have extended into the lipid core. This leads to coronary artery stenosis and consequent reduction in AML. Pathological and intravascular imaging studies have not confirmed the association between plaque erosion and AML in nonsmokers.

CLINICAL SIGNIFICANCE

The findings of this study suggested that predictors of plaque erosion varied with different smoking status in patients with STEMI and expand Doctors' understanding of clinical and lesion predictors of plaque erosion, especially in relation to smoking. Recently, more and more researchers agree that plaque rupture and plaque erosion are different clinical entities. Several clinical studies have revealed that plaque erosion can be treated distinctly from plaque rupture. However, the specific role of smoking in plaque rupture and erosion is still unclear. By delving deeper into this area, ideas for the prevention and management of STEMI patients with different current smoking status will be further explored for their individualized treatment. This in vivo OCT study may lay a foundation for future research. Comparative studies of STEMI based on plaque erosion and STEMI based on plaque rupture under different current smoking status may contribute to the goal of precision medicine and ultimately improve the prognosis of patients with STEMI. More research is needed to investigate the treatment and prognosis of patients with STEMI based on different mechanisms and different smoking status.

CONCLUSIONS

STEMI predictors based on plaque erosion (vs. plaque rupture) varied under different current smoking status. In patients with STEMI, age <50 years, single-vessel disease, and absence of dyslipidemia were independently associated with plaque erosion rather than plaque rupture, regardless of smoking. In current smokers, diabetes mellitus was negatively associated with plaque erosion compared with plaque rupture. In nonsmokers, higher MLA and close bifurcation were positively related to plaque erosion, but not to plaque rupture. Compared to plaque rupture, the correlation between plaque erosion and current smoking complements clinicians' understanding of plaque erosion.

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