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Editorial

Coronary Atherosclerosis from Cigarette Smoking: Emerging Concepts of the Pathogenic Mechanisms

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EDITORIAL

Coronary atherosclerosis due to cigarette smoking has been long-time clearly identified [1-2], although some controversies existed with regard to the responsible mechanisms.

Two main hypotheses are the basis to interpret the coronary artery harm [3-4]: an initially transient mechanism activating endothelial dysfunction with repeatable and constant responses at every smoking exposure primarily due to the effects of nicotine, which lead to atherosclerotic plaque formation, and a progressively structural damage chronically exerted by carbon monoxide with irreversible narrowing and thrombi, which reduce coronary lumen patency up to complete occlusion. However, evidence indicates that both mechanisms co-exist and act by potentiating results [5].

Analyzing the pathological characteristics of coronary artery lesions in the smokers, either observed in active smoking or developed in chronic and passive exposure, clearly emerges that the pathogenic mechanisms of damage follow well-defined pathways. This undoubtedly is a concept to be carefully taken into account since it is very difficult to attribute the course of the alterations to a type of smoking rather the other one.

The onset of coronary atherosclerosis is attributed to the endothelial dysfunction, which has been documented as the first process of lesion formation. In addition, coronary arteries together with carotid and cerebrovascular vessels have shown to be the target organs of cigarette smoking.

An intact endothelium protects against vascular injuries, but when this structure loses its integrity becomes thrombogenic because of several mechanisms, which stimulate platelet aggregation and adhesiveness, inflammatory cell migration on the vascular lesion and lipid material precipitates [3-4]. These factors are also a consequence of adrenergic and sympathetic nervous system activation mediated by the nicotine [6-7] of the cigarettes.

There is evidence that nicotine can activate all those mechanisms that lead to atherosclerotic plaque development and its complication.

Therefore, a question arises: why do coronary atherosclerosis from cigarette smoking differ in the different individuals?

This question requires to be clarified in an attempt to correctly interpret the role of smoking on the coronary artery wall.

Nicotine exerts its effects during smoking exposure, either when a subject smokes a cigarette or, otherwise, when passively exposed every time this occurs, but with a timely limited response. Therefore, coronary damage is a close result of duration and the effect of exposure to the drug. On the contrary, the degree and extent of the coronary lesions mainly depend on the carbon monoxide activity, which begins at the onset of smoking and progressively continues to harm coronary arteries even when a smoker is not exposed to smoke [8-10]. Thus, an emerging and well-demonstrated concept is the benchmark of atherosclerotic damage has to be considered carbon monoxide, which, strictly speaking, is not a chemical compound of tobacco smoke, but a derivative of the combustion between the paper of a burning cigarette and tobacco contained inside [11]. However, initially coronary alterations caused by carbon monoxide are reversible, becoming irreversible lesions when smoking exposure lasts chronically for several years.

As can be seen, factors related to the duration of exposure and individual characteristics may determine a different pattern of coronary damage, although this parameter pathologically keeps the same morphological characteristics.

It is worth noting that the lesions observed in coronary atherosclerosis from cigarette smoking consist of those alterations caused by other coronarogenic factors, although with a major extent, severity, and the number of coronary arteries affected [12].

In conclusion, consistent data provided by pathological and epidemiological studies of subjects exposed to smoking undoubtedly shows that coronary atherosclerosis is closely depending on the effects of nicotine and carbon monoxide with

a major degree and extent of alterations mainly consisting of a greater number of coronary arteries involved, more severe coronary narrowing and thrombi when compared to the atherosclerotic pathology of individuals never exposed to tobacco smoke. In addition, there would be documented evidence that carbon monoxide is the main factor of vascular damage.

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