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Editorial

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Cigarette smoking and acute myocardial infarction (AMI): where is the closest relationship?

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Editorial

On 1995, I wrote as a final remark of the relationship between cigarette smoking and heart on the Journal of Royal Society of Health[1] that "…the following statement may be formulated: Active smoking injures the cardiovascular system chronically causing structural lesions, which become, in the long run, irreversible alterations mainly related to coronary atherosclerosis. In contrast, passive smoking causes transiently impaired cardiac performance, which may be considerably more harmful for those people with established ischaemic heart disease. Therefore, when assessing the harmful effects of smoking, attention should be focused on those with ischaemic heart disease."

These conclusions were a milestone on the effects of smoking on cardiovascular system, as a large number of studies undoubtedly demonstrated.[2–5]

Evidence indicates that AMI is the most harmful event of the ischemic heart disease. Myocardial infarction denotes the necrosis of a part of cardiac muscle because of inadequate blood supply. Therefore, necrosis is the pathological substrate of AMI, which cannot be defined as it without this alteration.[6] Three types of necrosis can be observed in the context of an infarct area[7] determined by different pathogenic mechanisms: coagulation necrosis, colliquative myocytolysis, and coagulation myocytolysis. However, coagulation necrosis is the most common pattern of myocardial infarction. There is evidence that this type of necrosis is strongly associated with coronary pathology being the last also defined as a vascular necrosis. Reduced perfusion of specific areas of myocardium often due to complete coronary artery occlusion is recognized as the basic mechanism.

Coronary pathology is primarily characterized by narrowing of different type and degree of arterial lumen.[8,9] Several lesions may be superimposed on this substrate up to determine partial or total occlusion of the arterial vessel. Commonly, the coronary arteries are diffusely involved by atherosclerotic plaques. The degree of luminal narrowing may vary in the different individuals with AMI, but usually atherosclerotic plaques are present in almost every millimeter of the extramural coronary circulation.

It is worth noting that coronary alterations due to cigarette smoking are practically similar to those observed in the patients who suffer or suffered from AMI.[10]

Conclusion

There is evidence that coronary artery lesions are the strongest parameter, which associates AMI with smoking habit. The morphological alterations due to smoking able to trigger irreversible lesions primarily belong to carbon monoxide.[11,12] On the contrary, nicotine acts transiently but progressively to cause cardiac harm and is able to mask initially the adverse effects due to carbon monoxide.

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Aurelio Leone
Fellow of the Royal Society for Promotion of Health (FRSPH), UK

Correspondence: Aurelio Leone, Fellow of the Royal Society for Promotion of Health (FRSPH), Fellow of the American Heart Association (FAHA), UK, Tel +3934722722215, Email reliol@libero.it

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Conflicts of interest

The rest of the authors declare do not have conflicts of interest.

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