

# A panoramic view of the myocardial infarction: etiology, pathology, and comparison with the past

Volume 15 Issue 2 - 2022

**Aurelio Leone**

Fellow of the Royal Society for Promotion of Health (FRSPH), Italy

**Correspondence:** Aurelio Leone, Fellow of the Royal Society for Promotion of Health (FRSPH), Fellow of the American Heart Association (FAHA), Via Provinciale 27 19033 Castelnuovo Magra, Italy, Email [reliol@libero.it](mailto:reliol@libero.it)

**Received:** April 13, 2022 | **Published:** April 14, 2022

## Editorial

In a COVID-19 era, several harmful diseases have been neglected with regard to their incidence, pathology and clinical outcome. Among these, in my personal opinion myocardial infarction (MI), which undoubtedly is yet a serious problem for public health,<sup>1-3</sup> does not receive the due attention. Although the pathological features of MI have been widely defined allowing to recognize either microscopically or grossly the alterations of myocardial fibers,<sup>4-7</sup> there is evidence that some questions related to the disease are yet far to be correctly interpreted. Firstly, the variable association of the different types of necrosis which usually may be observed in MI needs of a more careful understanding.

Three types of necrosis, each characterized by a different pathogenic mechanism, are the pathological substrate of MI.<sup>8-11</sup> Evidence indicates that there is no established MI without necrosis, although some clinical events, primarily sudden death, related to the disease may occur.<sup>12,13</sup> The purpose of this editorial is to discuss the pathological features of necrosis, their association and/or extent, mechanisms responsible of the incidence and comparison of the pathological pictures actually known with the observations described in the past.

## Etiology of MI

As a large number of studies shows, there is no clear evidence of an etiologic factor always responsible of the occurrence of MI, which however is closely associated with coronary artery pathology.<sup>14,15</sup> A great number of coronary risk factors (Table 1) that usually accompany coronary atherosclerosis, even if with a variable association, have been significantly identified in those patients who suffered MI as table 1 shows. It is worth noting that coronary artery disease alone or accompanied by MI is the most common cause of death and disability in the industrialized countries.<sup>16</sup> The role of coronary risk factors in MI depends on the fact if they are single or associated to exert their power.

## Pathological features of necrosis of MI

As just mentioned, myocardial necrosis is the substrate of MI, although it is worth noting that different types of necrosis exist with a various degree of association and extent among them that may be commonly seen at the histopathologic analysis. Necrosis is the death of a cell or part of body tissue which occurs when blood flow meets a marked reduction due to vessel pathology or injury factors like chemical toxics, radiation, or mechanical stresses as human and experimental findings clearly have documented.<sup>17-20</sup> The three types of myocardial necrosis documented in MI (Table 2) interact with pathogenic mechanisms, which determine clinical features which may determine a different prognosis.

Coagulation necrosis is the most common pattern of myocardial necrosis primarily due to vascular mechanisms consisting of a

reduced perfusion of a myocardial area depending on the supply of the affected coronary artery, which may be partially or completely occluded. Myocardial fibers, although altered, can be recognized for hours or days from the onset of the pathologic process up to the appearance of colliquative myocytolysis. This type of necrosis consists of a liquefaction of the necrotic myocardium affected by the infarct due to an action of hydrolytic enzymes released by autolysis of the damaged material. Colliquative myocytolysis usually can lead to the development of chronic heart failure.

**Table 1** The coronary risk factors more frequently observed in individuals suffering from MI

A.	Modifiable risk factors
	Smoking
	Alcohol consumption
	Psychosocial behaviour
	Diet
B.	Modifiable risk factors associated with an effective therapy
	Abnormal lipid profile
	Diabetes mellitus
	Hypertension
	Metabolic syndrome
	Abdominal obesity
C.	Unmodifiable risk factors
	Coronary atherosclerosis
	Coronary artery disease (inflammatory disease)
	Age
	Gender (male)

Finally, sympathetic and hormonal mechanisms due to catecholamine release are primarily responsible of the coagulative myocytolysis where the myocardial fibers are deeply altered and contract bands due to cell death in hypercontraction similarly to what observed in the stone heart may be documented. Evidence indicates that myocardial fibrosis is the most common result of the healing of necrotic process that determines heart remodeling. As can be seen, a wide spectrum of lesions differently combined are the typical pattern of MI necrosis. Therefore, it depends on the prevailing pathogenic

mechanism, although coagulative necrosis related to coronary artery pathology (vascular necrosis) is the most pathognomonic pattern.

**Table 2** The different types of necrosis described in MI

Types of necrosis	Pathologic pattern	Mechanisms
Coagulation necrosis	Myocardial cell coagulation	Coronary artery lesions
Colliquative myocytolysis	Chemical lysis of myocytes	Enzymatic digestion
Coagulation myocytolysis	Stone heart (contract bands)	Neuro-hormonal action

## Summary of the factors to be focussed in MI

The observations described clearly permit to formulate the following statements.

1. The pathological lesions of MI, as Mallory et al.<sup>5</sup> carefully reported in the past, even today identify that vascular necrosis is the typical determinant of the disease. It is worth noting that in the large transmural infarcts there is a close correlation between the extent and size of the postocclusive supplying area of the infarct artery and the location of the MI.<sup>21,22</sup> Therefore, such a concept would be taken into account making necessary a careful analysis of the coronary circulation in those individuals potentially at risk of MI.
2. With regard to cardiovascular risk factors, an evident factor always acting as a cause of MI has not been identified, although the various risk factors often associated with coronary artery pathology, primarily smoking, hypertension, alteration of the metabolic profiles and obesity, play a significant role to increase the incidence of MI. However, there is evidence of MI caused by acute poisoning from carbon monoxide.<sup>23</sup>
3. MI and sudden cardiac death are the most dramatic events of the ischemic heart disease. It is worth noting that while an acute MI affecting a subject who died after 6 hours after the onset of the symptom of the disease can be diagnosed at the autopsy by microscopic exam, the sudden cardiac death occurring as the first sign of disease usually is asymptomatic without pathological alterations documented at the autopsy. Therefore, the sympathetic-adrenergic response of those individuals who display severe coronary pathology could be carefully studied in an attempt to reduce the incidence of MI.

## Acknowledgments

None.

## Conflicts of Interest

None.

## References

1. Leone A. Myocardial infarction. Pathological relevance and relationship with coronary risk factors. *Curr Pharm.* 2007;7(23):3205–3216.
2. White HD, Chew DP. Acute myocardial infarction. *Lancet.* 2006;372(9638):570–584.
3. Leone A. Toxics of tobacco smoke and cardiovascular system: From functional to cellular damage. *Curr Pharm Des.* 2015;21(30):4370–4379.
4. Baroldi G, Radice F, Schimid G, et al. Morphology of acute myocardial infarction in relation to coronary thrombosis. *Am Heart J.* 1974;87(1):65–75.
5. Mallory GK, White PD, Salcedo-Salgar J. The speed of healing of myocardial infarction. A study of the pathologic anatomy in seventy-two cases. *Am Heart J.* 1939;18:647–671.
6. Cantin M, Leone A. Morphology of myocardial infarction. *Methods Achiev Exp Pathol.* 1981;10:244–284.
7. Leone A. Gross and microscopic analysis of structures involved. In: Passive Smoking and Cardiovascular Pathology, Mechanisms and Physiopathological Basis of Damage, A Leone ed., Nova Science Publishing Inc, New York, 2007;39–54.
8. Eliot RS, Baroldi G, Leone A. Necropsy studies in myocardial infarction with minimal or no coronary luminal reduction due to atherosclerosis. *Circulation.* 1974;49(86):1127–1131.
9. Nabel EG, Braunwald E. A tale of coronary artery disease and myocardial infarction. *N Engl J Med.* 2012;366:54–63.
10. Davies MJ, Woolf N, Robertson WB. Pathology of acute myocardial infarction with particular reference to occlusive coronary thrombi. *Br Heart J.* 1976;38:659–664.
11. Leone A. Coronary atherosclerosis. In: Coronary Circulation in Nonsmokers and Smokers, A Leone ed., Nova Biochemical Books, New York, 2008;119–147.
12. Baroldi G. Myocardial necrosis: the need for definition. *J Mol Cell Cardiol.* 1974;6(4):401–402.
13. Vahatalo JH, Huikuri HV, Holmstrom LTA, et al. Association of silent myocardial infarction and sudden cardiac death. *JAMA Cardiol.* 2019;4(8):796–802.
14. Moser M, Roccella EJ. The treatment of hypertension: a remarkable success story. *J Clin Hypertens.* 2013;15(2):88–91.
15. Leone A, Landini L, Leone A. Epidemiology and costs of hypertension related disorders. *Curr Pharm Des.* 2011;17(28):2955–2972.
16. Leone A. Relationship between cigarette smoking and other coronary risk factors in atherosclerosis: Risk of cardiovascular disease and preventive measures. *Curr Pharm Des.* 2003;9:2417–2423.
17. Csapò Z, Dusek J, Rona G. Early alterations of cardiac muscle cells in isoproterenol-induced necrosis. *Arch Path.* 1972;93:356–365.
18. Leone A, Landini L Jr, Biadi O, et al. Smoking and cardiovascular system: Cellular features of the damage. *Curr Pharm Des.* 2008;14:1771–1777.
19. Falk E. Morphologic features of unstable atherothrombotic plaque underlying acute coronary syndromes. *Am J Cardiol.* 1989;63:E14–E20.
20. Roberts WC. Coronary arteries in fatal acute myocardial infarction. *Circulation.* 1972;45:215–230.
21. Blumgart HL, Zoll PM. Pathologic physiology of angina pectoris and acute myocardial infarction. *Circulation.* 1960;22:301–307.
22. Hort W. Pathology of acute myocardial infarction and the infarct vessel. *Eur Heart J.* 1985;6(Suppl E):5–9.
23. Marius-Nunez AL. Myocardial infarction with normal coronary arteries after acute exposure to carbon monoxide. *Chest.* 1990;97(2):491–494.