MedCrave
Step into the World of Research

Journal of Cardiology & Current Research

Editorial

Open Access

CrossMark

Ultrastructural components of myocardial cell: what is their role in myocardial pathology?

Volume 8 Issue 3 - 2017

Aurelio Leone[1,2,3]

[1]Fellow of the American Society of Hypertension (FASH), USA
[2]Fellow of the Royal Society for Promotion of Health (FRSPH), UK
[3]Editor-in-Chief of the Journal of Cardiology6&Current Research, USA

Correspondence: Aurelio Leone, Fellow of the American Society of Hypertension (USA), Fellow of the Royal Society for Promotion of Health (UK), Editor-in-Chief of the Journal of Cardiology & Current Research (USA), Via Provinciale 27, 19030 Castelnuovo Magra, Italy, Email reliol@libero.it

Received: February 06, 2017 | **Published:** March 03, 2017

Editorial

Usually, the knowledge of the cardiovascular disease identifies its fundamental characteristics in a clinical, pathological, and epidemiological field based on the macroscopic results derived from the widespread type of findings. A poor attention is devoted to ultrastructural alterations of the myocardial cell that are the factors responsible of macroscopic pathology.[1–3] Thus, it is worth noting that all respiratory chains and related enzymes of the metabolic reactions that regulate cellular and organismic homeostasis are located inside the living cells. Therefore, the main patterns of cardiovascular pathology depend on intracellular changes of these components.

Evidence indicates that specific microvascular ultrastructural abnormalities have been described during the early stages of coronary artery occlusion and have been associated with the "no-reflow phenomenon" which consisted of inability to reperfuse a previously ischemic region.[4,5] Mitochondria and ribosomes are the intracellular structures most frequently analyzed by the ultrastructural findings,[6] since they bear the maximum "weight" of the metabolic reactions.

Mitochondria[7,8] consist of intracellular organelles which provide energy to myocardial cell metabolism by converting nutrients to energy through metabolic reactions that utilize sugar as a substrate. Mitochondria contain both RNA and a small amount of DNA primarily damaged by oxygen free radicals, which exert a strong role in the pathogenic mechanisms of atherosclerosis and ischemic heart disease.[9] In addition, RNA metabolism influences DNA replication with changes in intracellular function.

Evidence indicates that all the changes occurred in mitochondria function come before and are the pathological substrate of macroscopic alteration development. Therefore, impeding mitochondria lesions could be an active support to reduce the incidence of cardiovascular disease. Ribosomes are intracellular organelles that are the sites of RNA and protein synthesis that strongly feel the toxic effects due primarily to carbon monoxide including that of cigarette smoking.[10] Thus, it is worth noting that carbon monoxide is the main chemical compound of cardiovascular damage either under morphological or clinical outcome.[11]

As previously mentioned,[4] ultrastructural alterations would seem to precede clinic-pathological signs of the cardiovascular disease. It is worth noting that myocardial contraction, the basic property of heart function, occurs because of the sliding of thin actin filaments in the context of tick myosin filaments without shortening of these proteins and this mechanism recognizes the use of energy produced by the metabolic reactions of the adenosine triphosphate for the contraction coming out from mitochondria, which are about one and third in volume of myofibrils.[12,13] A limitation to a careful study of ultrastructural alterations compared to macroscopic changes of the heart and blood vessels is undoubtedly due to the characteristics of study material. Ultrastructural pathology mainly recognizes experimental findings related to animal material while gross pathology utilizes both animal and human studies that can be more easily conducted.

This assumption is only partly true because experimental findings which analyze both gross and ultrastructural specimens in the same study identify corresponding results particularly in the chronic and established lesions. On the contrary, there is pathological evidence that in the experimental myocardial infarction ultrastructural alterations usually come before a few hours of the gross and clinical sign of the disease appearance.[14,15]

Conclusion

In other words, the commonly observed signs of cardiovascular disease recognize an ultrastructural pathologic substrate that precedes and determines them. This should be carefully taken into account as well is the physio-pathological basis to correctly interpret myocardial and vascular damage. Therefore, trying to assess the ultrastructural alterations could improve our knowledge on the onset and development of both pathological and clinical features of a disease. Thus, this fact is of a great importance with regards to heart and blood vessel disease, which is still today a plague for the living people. Findings on the ultrastructural characteristics of cardiovascular disease should be further promoted and encouraged to better interpret its true meaning.

Acknowledgments

None.

Conflicts of interest

Author declares there is no conflicts opf interest.

Funding

None.

References

1. Leone A. Cardiovascular damage from smoking: a fact or belief? *Int J Cardiol*. 1993;38(2):113–117.
2. Heggtweit HA, Nadkarni BB. Ultrastructural pathology of the myocardium. *Methods Achiev Exp Pathol.* 1971;5:474–517.
3. Jennings RB. Historical perspective on the pathology of myocardial ischemia/reperfusion injury. *Circ Res* . 2013;113(4):428–438.
4. Kloner RA, Rude RE, Carlson N, et al. Ultrastructural evidence of microvascular damage and myocardial cell injury after coronary artery occlusion: Which comes first? *Circulation.* 1980;62(5):945–952.
5. Rezkalla SH, Kloner RA. No-reflow phenomenon. *Circulation.* 2002;105(5):656–662.
6. Leone A, Landini L Jr, Biadi O, et al. Smoking and cardiovascular system: Cellular features of the damage. *Curr Pharm Des.* 2008;14(18):1771–1777.
7. Chang DD, Clayton DA. A mammalian mitochondria RNA processing activity contains nucleus-encoded RNA. *Science.* 1987;235(4793):1178–1184.
8. Van de Bosch BJC, van den Burg CMM, Scoonderwoerd K, et al. Regional absence of mitochondria causing energy depletion in the myocardium of muscle LIM protein knockout mice. *Cardiovasc Res.* 2005;65(2):411–418.
9. Zweier JL, Talukder HMA. The role of oxidant and free radicals in reperfusion injury. *Cardiovasc Res* . 2006;70(2):181–190.
10. Astrup P. Carbon monoxide smoking, and cardiovascular disease. *Circulation*. 1973;48:1167–1168.
11. 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(29):2417–2423.
12. Huxley HE. The double array of filaments in cross-striated muscle. *J Biophys Biochem Cytol.* 1957;3(5):631–648.
13. Huxley HE. The contractile structure of cardiac and skeletal muscle. *Circulation*. 1961;24:328–335.
14. Cantin M, Leone A. Morphology of myocardial infarction. *Methods Achiev Exp Pathol*. 1981;10:244–284.
15. Leone A. Morphological alterations of the heart and blood vessels from tobacco smoke: The steps of the damage. *J Cardiol Ther*. 2015;2(4):355–359.