

COMMENTARY

Cardioprotection: Challenges and Perspectives

Emanuel Tenório Paulino

Institute of Pharmaceutical Sciences, Cardiovascular Pharmacology, Federal University of Alagoas, Brazil.



Open Access

Citation: Paulino, ET.
Cardioprotection: Challenges and Perspectives. *Annals of Cardiology*. 2025; 1(1): 3.

Received: April 11, 2025

Accepted: May 20, 2025

Published: June 09, 2025

Copyright: © 2025. Paulino, ET.

This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Corresponding author:
Emanuel Tenório Paulino,
Federal University of Alagoas, Brazil;
E-mail: emanueltenorio17@gmail.com

Abstract

Myocardial infarction is a disease that causes high levels of death around the world. Despite improvements in the management of this pathology, myocardial infarction continues to be the leading cause of death all over the planet. This study aimed to show the challenges and perspectives for stopping death caused by myocardial infarction. This, here is shown through by commentary article in basis on new strategies for cardioprotective development using translational studies and innovations into therapeutics prototypes for cardioprotective agents. In conclusion, this paper evidences the understanding of molecular pathology of myocardial infarction to promote new agents in cardiovascular therapy focused on the prevention of acute myocardial infarction.

Keywords

Cardioprotection, Myocardial Infarction, Pharmacotherapy

Introduction

Myocardial Infarction is a several diseases affecting million peoples in the world.¹ Commonly clinical outcomes turn peoples unable to work return due limitations of hearts insufficiency.² Despite the health public organizations have progressed in both: the accuracy of diagnosis and management of pharmacotherapy, data annually around the world showed these progressions haven't ameliorated the index of the new cases or prevention of death or inflection outcomes clinical. Maybe due to complex therapeutics care, multiple phases and uses of many classes of drugs to control oxygen supply from hearts, or the need early diagnosis and management care.³ In addition, surgical procedures can be fatal by the time of clinical service versus the progression of ischemia drive to hearts.⁴

Challenges

The scientific community has searched for new methods and ways to stopped myocardial infarction cases.⁵ Of course, in a new era, novel behaviors by people have become more difficult to arrive at this point. High stressful lifestyle by work and increases in anxiety due to social media could be a contributive mechanism to drive new cases annually.⁶ These problems were not considered in the past, where the focus was smoking, hypertension, or age-related, axis-based, considering now a new manner of modern lifestyle.⁷ In 2025, it is necessary to return to the molecular bases of the pathophysiology of myocardial infarction, to comprehensively the question: how to stop the first disease of death in the world?⁸ Injury by hypoxia-reperfusion is a point of the iceberg in the pathophysiology of myocardial infarction; it is not enough to solve oxygen supply, and needs to treat silent movements by pathways in the body before existing injury by oxygen imbalance.³

In phrases of Kübler & Haas: “cardioprotection are all mechanisms and means that contribute to the preservation of heart by reducing or even preventing myocardial damage”.⁹ Thus, the background of the myocardial infarction pathophysiology is needed, because, if seen closer, it is clear that past factors are predictive and the new aligned in turn of the same axis: the autonomic nervous system, overdriving of the sympathetic ways, and regulatory feedback mechanisms to parasympathetic ways loss levels.¹⁰ Hypertension, ageless, lifestyle anxiety, and work burnout are the triggers to excess norepinephrine and lower levels of acetylcholine behind the development of cardiovascular diseases.¹¹ Nevertheless, cholinergic ways are considered cardioprotective in new articles or scientific papers.¹² But, would this be the key to the only fit in the lock, in terms of Paul Erlisch?¹³ No, but why are the defense systems not effective in overcoming the breakdown development of myocardial infarction, such as β -arrestins, opening potassium channels, signaling of the muscarinic and nicotinic receptors to SAFE (Survivor Activating Factor Enhancement) or RISK (Reperfusion Injury Salvage Kinase) pathology pathways, protection of mitochondria disruption by recent discovery channel's exist into mitochondria.^{14,15} Here found the biggest challenges to transpose.

Perspectives

Now, it is necessary to investigate the failure of each mechanism to understand molecular myocardial infarction, which occurs in mammalian animals both small and large; it is equal to occurs in human conditions.¹⁶ At this time, scientists are finding responses to the question and turning to ease translational pharmacology studies. On the other hand to successful of the new strategy proposals in real development, whether through therapeutic innovations, such as immunobiological vaccines using RNAm silencers introduced into adenovirus to prevent new cases or Stem cells by surgical procedures, even if it is expensive for the moment for the majority of the world's population.^{3,17,18} They bring light to translation pharmacotherapy and together reduce costs to all people, similar to what happened recently with COVID-19, or through new compounds to produce cardioprotective actions, based on natural products as a source to new drugs in coherence to Cragg & Newman,2020 studies by introduction nutraceuticals or phytochemicals or synthetic compounds have a potential therapeutic.¹⁹

Conclusion

In conclusion, this commentary sheds new light on cardioprotection area and prevents myocardial infarction, promoting amelioration in this scenario around the world.

Acknowledgments

I would like to thank the Journal for inviting me to write this “commentary article”, free of pay and fees.

Conflict of interest

The author declares no conflict of interest.

References

1. Salari N, Morddarvanjoghi F, Abdolmalek A et al. The global prevalence of myocardial infarction: a systematic review and meta-analysis. *Cardiovasc Disord.* 2023; 23: 206.
2. JenČa D, Melenovsky V, Stehlik J et al. Heart failure after myocardial infarction: incidences and predictors. *ESC. Heart Fail.* 2020; 148(1): 222–237.
3. Tenório, E.P. Development of the cardioprotective drugs class based on pathophysiology of myocardial infarction: A comprehensive review. *Curr Prob Cardiol.* 2024; 49.
4. Yousef S, Sultan I, VonVille H et al. Surgical management for mechanical complications of acute myocardial infarction: a systematic review of long-term outcomes. *Ann Cardiothorac Surg.* 2022; 11(3).
5. Hausenloy D.J. Translating cardioprotection for patient benefit: The EU-CARDIOPROTECTION COST Action. *J American Coll Cardiol.* 2019; 73: 15.
6. Zhang L., Bao Y., Wang X et al. A meta-analysis on the prevalence, associated factors

- and diagnostic methods of mental stress induced myocardial infarction. *J Transl Med.* 2020; 18(1): 218.
7. Mechanic O.J; Gavin M., Grosmann S. Acute myocardial infarction. *Statpearls.* 2023.
8. American Heart Association. Understanding your risks to prevent a heart attack. 2025 Available: <https://www.heart.org/en/health-topic/heart-attack>
9. Klübler, W & Haass, M. Cardioprotection: definition, classification, and fundamental principles. *Heart.* 1996; 75(4): 330–333.
10. Kingma J., Simard D., Rouleau J.R. Influence of cardiac nerve status on cardiovascular regulation and cardioprotection. *World J Cardiol.* 2017; 26(6): 508–520.
11. Mehta P.K., Sharma A Bremner J.D., et al. Mental Stress-induced myocardial ischemia. *Curr Cardiol Reports.* 2022; 4: 2109–2120.
12. Itanchai, K. et al. Revisiting the Cardioprotective Effects of Acetylcholine Receptor Activation against Myocardial Ischemia/Reperfusion Injury. *Int J Mol Sci.* 2018; 19: 1-20.
13. Paul Ehrlich. Biographical. The nobel prize. Available: <http://nobelprize.org/prizes/medicine/1908/erlich/biographical.2025>
14. Hadebe N., Cour M., Lecour S. The SAFE pathway for cardioprotection: is this a promising target?. *Basic Res Cardiol.* 2018; 113(2): 9.
15. Yellow DM., Kalkhoran S.B; Davidson SM. The RISK pathway leading to mitochondria and cardioprotection: how everything started. *Basic Res Cardiol.* 2023; 118(1): 22.
16. Hausenloy DJ., Botker HE., Condorelli G et al. Translating cardioprotection for patient benefit: position paper from the working group of cellular biology of the heart society of cardiology. *Cardiovasc Res.* 2013; 98(1): 7–27.
17. Duan HF., Wang H., Yi J. Adenoviral gene transfer of sphingosine kinase 1 protects heart against ischemia/reperfusion injury and attenuates postischemic failure. *Hum Gene Ther.* 2007; 18: 1119–1128.
18. Madonna R & Caterina R. Stem cells and growth factor delivery system for cardiovascular disease. *J Biotechnol.* 2011; 154: 291–297.
19. Newman DJ & Cragg GM. Natural Products as Source of new drugs over the nearly four decades from 01/1981 to 09/2019. *J Nat Prod.* 2020; 83(3): 770–803.