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Recent Perspectives on Mitochondrial Involvement in Sepsis-induced Cardiomyopathy

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Abstract

Sepsis-induced cardiomyopathy (SIC) represents a critical complication of sepsis, contributing significantly to morbidity and mortality in septic patients. Emerging evidence underscores the pivotal role of mitochondrial dysfunction in the pathogenesis of SIC. This article provides a comprehensive overview of the intricate interplay between mitochondria and SIC, elucidating the underlying mechanisms and potential therapeutic avenues. Mitochondrial dysfunction in sepsis, characterized by impaired ATP production, oxidative stress, and mitochondrial permeability transition pore opening, contributes to myocardial injury and dysfunction in SIC. Dysregulation of mitochondrial dynamics, biogenesis, and energy metabolism further exacerbates cardiac dysfunction in SIC. Therapeutic strategies targeting mitochondria, including mitochondria-targeted antioxidants and pharmacological agents modulating mitochondrial dynamics and biogenesis, hold promise for preserving cardiac function and improving outcomes in SIC. Continued research efforts aimed at unraveling the molecular pathways involved in mitochondrial dysfunction and exploring novel mitochondria-targeted therapies are essential for advancing our understanding and management of SIC.

Keywords: Sepsis • Cardiomyopathy • ICU

Introduction

Sepsis-induced cardiomyopathy (SIC) is a severe complication of sepsis characterized by myocardial dysfunction and contributes significantly to the morbidity and mortality associated with septic shock. While the pathogenesis of SIC is complex and multifactorial, emerging evidence suggests that mitochondrial dysfunction plays a central role in its development. In this article, we delve into the current insights into the intricate interplay between mitochondria and SIC, shedding light on the underlying mechanisms and potential therapeutic strategies [1].

Literature Review

Mitochondria are vital organelles responsible for cellular energy production, calcium homeostasis, and apoptosis regulation. During sepsis, dysregulated host immune responses, oxidative stress, and inflammatory mediators can disrupt mitochondrial function, leading to impaired ATP production, increased reactive oxygen species generation and mitochondrial permeability transition pore opening. These mitochondrial derangements contribute to myocardial injury and dysfunction observed in SIC. Mitochondrial dynamics, encompassing processes like fission, fusion, and mitophagy, play a critical role in maintaining mitochondrial quality control and cellular homeostasis. Dysregulation of these dynamics has been implicated in the pathogenesis of SIC. Excessive fission can result in mitochondrial fragmentation and dysfunction, while impaired mitophagy leads to the accumulation of damaged mitochondria and oxidative stress. Conversely, enhancing mitochondrial fusion and promoting mitophagy may offer protective effects against SIC by preserving mitochondrial function and reducing myocardial injury [2].

Mitochondrial biogenesis, regulated by factors such as PGC-1α and NRF-1, is essential for generating new mitochondria. Sepsis-induced suppression of mitochondrial biogenesis exacerbates mitochondrial dysfunction and energy depletion in cardiomyocytes. Additionally, alterations in energy metabolism, such as the shift from oxidative phosphorylation to glycolysis, contribute to myocardial dysfunction in SIC. Targeting mitochondrial biogenesis and restoring metabolic homeostasis represent promising therapeutic avenues for mitigating SIC-associated cardiac injury. Several therapeutic interventions aimed at preserving mitochondrial function and attenuating myocardial injury in SIC have been proposed. These include mitochondria-targeted antioxidants like MitoQ and MitoTEMPO, which scavenge ROS and mitigate oxidative stress-induced mitochondrial damage. Pharmacological agents modulating mitochondrial dynamics, biogenesis, and metabolism, such as mitochondrial division inhibitor-1 (Mdivi-1) and resveratrol, hold promise for restoring mitochondrial homeostasis and improving cardiac function in SIC [3].

Discussion

Mitochondrial dynamics, including fission, fusion, and mitophagy, play crucial roles in maintaining mitochondrial quality control and cellular homeostasis. Dysregulation of mitochondrial dynamics has been implicated in the pathogenesis of SIC. Excessive mitochondrial fission can lead to mitochondrial fragmentation and dysfunction, whereas impaired mitophagy can result in the accumulation of damaged mitochondria and oxidative stress. Conversely, promoting mitochondrial fusion and enhancing mitophagy may have protective effects against SIC by preserving mitochondrial function and reducing myocardial injury. Mitochondrial biogenesis, the process of generating new mitochondria, is regulated by various transcription factors and coactivators, including PGC-1α and NRF-1. Sepsis-induced suppression of mitochondrial biogenesis further exacerbates mitochondrial dysfunction and energy depletion in cardiomyocytes. Additionally, alterations in energy metabolism, such as the shift from oxidative phosphorylation to glycolysis, contribute to myocardial dysfunction in SIC. Targeting mitochondrial biogenesis and restoring metabolic homeostasis represent promising therapeutic strategies for mitigating SIC-associated cardiac injury. Several therapeutic interventions aimed at preserving mitochondrial function and attenuating myocardial injury in

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SIC have been proposed. These include mitochondria-targeted antioxidants, such as MitoQ and MitoTEMPO, which scavenge ROS and mitigate oxidative stress-induced mitochondrial damage. Pharmacological agents modulating mitochondrial dynamics, biogenesis, and metabolism, such as mitochondrial division inhibitor-1 and resveratrol, hold promise for restoring mitochondrial homeostasis and improving cardiac function in SIC [4-6].

Conclusion

Mitochondrial dysfunction plays a pivotal role in the pathogenesis of sepsis-induced cardiomyopathy, contributing to myocardial injury and dysfunction. Current insights into the intricate mechanisms underlying mitochondrial dysfunction in SIC have highlighted the therapeutic potential of targeting mitochondria to preserve cardiac function and improve outcomes in septic patients. Further research aimed at elucidating the molecular pathways involved in mitochondrial dysfunction and evaluating novel mitochondria-targeted therapies is warranted to advance our understanding and management of SIC.

Acknowledgement

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Conflict of Interest

None.

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