International Journal of Medical Science and Clinical Research Studies

ISSN(print): 2767-8326, ISSN(online): 2767-8342

Volume 03 Issue 11 November 2023

Page No: 2826-2828

DOI: https://doi.org/10.47191/ijmscrs/v3-i11-53, Impact Factor: 6.597

Presentation of Acute Myocardial Infarction Type 2 in a Patient with Septic Shock

Vania Hailed Moreno Mora[1], Christian Alfonso Torres Bazán[2]

[1,2]Servicio de Medicina Interna, Centro Médico Nacional de Occidente, Guadalajara, Jalisco, México.

ABSTRACT

Acute myocardial infarction (MI) in the setting of septic shock presents a multifaceted clinical challenge. In this review, we explore the distinct presentation of type 2 MI among patients grappling with septic shock and coronary artery disease. The coexistence of these conditions poses significant risks, potentially leading to both type 1 and type 2 MIs. We delve into the pathophysiological underpinnings and the clinical implications of these subtypes of MI, emphasizing the altered hemodynamics and increased metabolic demand leading to type 2 MI. Our discussion highlights the differences in management and outcomes, underscoring the critical need for nuanced understanding in clinical practice.

KEYWORDS: Acute Myocardial Infarction, Type 2 MI, Septic Shock, Coronary Artery Disease, Inflammation, Demand Ischemia

ARTICLE DETAILS

Published On:
21 November 2023

Available on:
https://ijmscr.org/

INTRODUCTION

The co-occurrence of septic shock and coronary artery disease poses a complex challenge in clinical medicine [1]. Patients admitted with septic shock confront a heightened risk of developing both type 1 and type 2 myocardial infarction (MI), amplifying the intricacies of their clinical course [2,3].

Septic shock, a state characterized by a dysregulated host response to infection, orchestrates a cascade of pathophysiological events. This dysregulation precipitates profound circulatory, cellular, and metabolic disturbances, creating a precarious environment for patients, potentially culminating in various complications, including myocardial infarction [4].

The mechanistic explanation for type 1 MI within the context of septic shock revolves around the inflammatory state inducing intravascular coagulation, thereby heightening the risk of coronary thrombosis. Conversely, type 2 MI, also known as demand ischemia, manifests when the altered hemodynamics in septic shock exceed the myocardial oxygenation capacity, resulting in an imbalance between metabolic demand and oxygen supply [3,5].

Definition of Septic Shock

Septic shock represents the critical and severe progression of sepsis, wherein the body mounts an overwhelming and dysregulated response to an infection. This aberrant response triggers a complex cascade of events, resulting in life-threatening circulatory dysfunction. It's characterized by refractory hypotension despite adequate fluid resuscitation and is often accompanied by cellular and metabolic abnormalities, contributing to multiple organ dysfunction. This critical state is a medical emergency, demanding immediate intervention. It necessitates aggressive resuscitation, targeted antimicrobial therapy, and supportive care to mitigate its deleterious effects on patient outcomes [5,6].

- Definition of Myocardial Infarction

Myocardial infarction encompasses a spectrum of myocardial ischemic injuries and is commonly categorized into two main types frequently encountered in the context of septic shock. Type 1 MI, the classical form, typically results from the sudden rupture of an atherosclerotic plaque in a coronary artery, leading to acute thrombotic occlusion. On the other hand, type 2 MI arises from a supply-demand mismatch in myocardial oxygenation. It's often associated with conditions such as severe systemic hypotension, tachycardia, or other hemodynamic alterations that challenge the myocardium's oxygen supply relative to its demand [7].

Types of Myocardial Infarction

- Type 1 Myocardial Infarction

Type 1 MI is the classical form primarily attributed to acute coronary events, such as the rupture of an atherosclerotic plaque, leading to acute thrombotic occlusion of a coronary artery. This abrupt occlusion results in myocardial ischemia and subsequent injury. In the context of septic shock, the

heightened inflammatory state predisposes patients to an increased risk of plaque rupture and thrombosis, thereby elevating the incidence of type 1 MI.

The immediate focus in the management of type 1 MI is often directed towards the restoration of coronary blood flow. Prompt recognition and intervention through reperfusion strategies, such as thrombolytic therapy or percutaneous coronary intervention (PCI), are crucial in salvaging myocardial tissue and improving patient outcomes [8].

- Type 2 Myocardial Infarction

Type 2 MI arises from an imbalance between myocardial oxygen supply and demand. In conditions like septic shock, where systemic abnormalities alter hemodynamics, there is an excessive strain on the myocardium, surpassing its oxygenation capacity. This results in myocardial ischemia without the involvement of acute coronary events like plaque rupture or thrombosis [8].

In the management of type 2 MI, the primary focus revolves around addressing the underlying systemic condition that precipitates the imbalance in myocardial oxygen supply-demand. Optimizing hemodynamics through appropriate fluid resuscitation and vasopressor support while treating the underlying cause of shock, such as sepsis or hypovolemia, is critical. However, distinguishing between type 1 and type 2 MIs becomes crucial as their management strategies often differ significantly [8, 9].

- Pathophysiology

The pathophysiological interplay between septic shock and myocardial infarction is complex. In the context of septic shock, the hyperinflammatory state provokes an environment conducive to intravascular coagulation, increasing the risk of type 1 MI. Simultaneously, the altered hemodynamics, particularly the decreased perfusion pressure, impose an excessive metabolic demand on the myocardium, surpassing its oxygenation capacity, leading to the characteristic ischemic injury observed in type 2 MI. Understanding these mechanisms is crucial in differentiating the subtypes and tailoring appropriate management strategies [1-3].

- Management

Managing myocardial infarction within the complexity of septic shock demands a nuanced and comprehensive approach. Timely recognition and differentiation between the types of MI are crucial in tailoring appropriate therapeutic strategies. The management involves optimizing hemodynamics, addressing the underlying cause of septic shock, and initiating targeted therapies while considering the specific features of type 1 and type 2 Mis [9-10].

DISCUSSION

Relationship Between Septic Shock and Myocardial Infarction

The intricate relationship between septic shock and myocardial infarction (MI) underscores the complexity of managing these conditions when they coexist. Septic shock, characterized by a dysregulated host response to infection, induces a hyperinflammatory state and disrupts hemodynamics, predisposing patients to both type 1 and type 2 Mis [1,2,7-10].

The heightened inflammatory state in septic shock contributes to an intravascular prothrombotic environment, increasing the risk of coronary thrombosis and subsequent type 1 MI. Concurrently, the altered hemodynamics, often characterized by reduced perfusion pressure, places an excessive metabolic demand on the myocardium, surpassing its oxygenation capacity and leading to ischemic injury observed in type 2 MI [4, 5].

The presence of septic shock significantly complicates the diagnostic landscape for MI. The systemic inflammation and hemodynamic aberrations in septic shock can mask or mimic the classical presentations of MI, leading to diagnostic challenges. The reliance on traditional biomarkers and electrocardiographic changes, while pivotal in MI diagnosis, might lack the specificity to differentiate between type 1 and type 2 MIs in the context of septic shock [8-10].

Moreover, the coexistence of septic shock and MI poses therapeutic challenges. Tailoring appropriate interventions necessitates a delicate balance. While reperfusion strategies are vital in type 1 MI, managing type 2 MI involves addressing the underlying shock etiology and optimizing systemic conditions.

Understanding the intertwined relationship between septic shock and MI is crucial in refining diagnostic criteria, developing specific management strategies, and improving patient outcomes in this complex clinical scenario [3,7-10].

CONCLUSION

The confluence of septic shock and myocardial infarction presents a complex interplay with profound clinical implications. The relationship between these conditions, both in their pathophysiological connection and the challenges they pose in diagnosis and management, underscores the intricate nature of their coexistence.

Septic shock, characterized by a dysregulated host response to infection, elevates the risk of both type 1 and type 2 myocardial infarctions. The hyperinflammatory state predisposes individuals to intravascular coagulation and coronary thrombosis, contributing to type 1 MI. Concurrently, the altered hemodynamics, often associated with decreased perfusion pressure, imposes an excessive strain on the myocardium, leading to demand ischemia and type 2 MI.

Distinguishing between these MI subtypes within the context of septic shock remains a diagnostic challenge. The reliance on traditional biomarkers and electrocardiographic changes might not sufficiently differentiate between type 1 and type 2 MIs, posing hurdles in prompt and accurate diagnosis.

Therapeutically, tailored approaches are crucial. While reperfusion strategies are pivotal in type 1 MI, managing type 2 MI involves a broader spectrum of interventions, primarily addressing the underlying shock etiology and optimizing systemic conditions.

The need for precise diagnostic criteria and nuanced therapeutic interventions is evident. Enhancing our understanding of the specific pathophysiological mechanisms underlying type 1 and type 2 MIs in the context of septic shock is imperative to refine diagnostic tools and develop targeted therapeutic strategies.

In essence, the intricate relationship between septic shock and myocardial infarction demands a comprehensive approach, emphasizing the necessity for refined diagnostic modalities and tailored management strategies to improve outcomes for these critically ill patients.

REFERENCES

I. Ammann, P., Fehr, T., Minder, E., Günter, C., & Bertel, O. (2001). Elevation of troponin I in sepsis and septic shock. Intensive care medicine, 27, 965-969.

II. Putot, A., Derrida, S. B., Zeller, M., Avondo, A., Ray, P., Manckoundia, P., & Cottin, Y. (2018). Short-term prognosis of myocardial injury, type 1, and type 2 myocardial infarction in the emergency unit. The American journal of medicine, 131(10), 1209-1219.

III. Kamat, I. S., Nazir, S., Minhas, A. M. K., Nambi, V., Kulkarni, P., Musher, D., ... & Jneid, H. (2022). Outcomes of Hospitalizations With Septic Shock Complicated by Types 1 and 2 Myocardial Infarction. The American Journal of Cardiology, 174, 27-33.

IV. Jacobi, J. (2022). The pathophysiology of sepsis—2021 update: Part 1, immunology and coagulopathy leading to endothelial injury. American Journal of Health-System Pharmacy, 79(5), 329-337.

V. Nduka, O. O., & Parrillo, J. E. (2011). The pathophysiology of septic shock. Critical Care Nursing Clinics, 23(1), 41-66.

VI. Cecconi, M., Evans, L., Levy, M., & Rhodes, A. (2018). Sepsis and septic shock. The Lancet, 392(10141), 75-87.

VII. DeFilippis, A. P., Chapman, A. R., Mills, N. L., De Lemos, J. A., Arbab-Zadeh, A., Newby, L. K., & Morrow, D. A. (2019). Assessment and treatment of patients with type 2 myocardial infarction and acute nonischemic myocardial injury. Circulation, 140(20), 1661-1678.

VIII. Wereski, R., Kimenai, D. M., Bularga, A., Taggart, C., Lowe, D. J., Mills, N. L., & Chapman, A. R. (2022). Risk factors for type 1 and type 2 myocardial infarction. European Heart Journal, 43(2), 127-135.

IX. Basalay, M. V., Yellon, D. M., & Davidson, S. M. (2020). Targeting myocardial ischaemic injury in the absence of reperfusion. Basic Research in Cardiology, 115, 1-16.

X. Barnett, C. F., Brusca, S. B., Hanff, T. C., Blumer, V., Kalif, A., & Kanwar, M. (2023). Management of cardiogenic shock unrelated to acute myocardial infarction. Canadian Journal of Cardiology, 39(4), 406-419.