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Case Report

ST-Elevation Mimicking Myocardial Infarction in a Sepsis Condition: A Diagnostic Dilemma

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Abstract

Sepsis-induced myocardial injury can clinically mimic an ST-Elevation Myocardial Infarction (STEMI), presenting a critical diagnostic dilemma. This descriptive qualitative case study aimed to elucidate this phenomenon and highlight appropriate clinical management. The research focused on a single, purposively selected patient: a 31-year-old male with sepsis from a scrotal abscess who presented with chest pain, ST-segment elevation on electrocardiogram (ECG), and significantly elevated cardiac troponins. Data from the patient's medical records, including serial ECGs and coronary angiography results, were analyzed descriptively. Primary percutaneous coronary intervention (PCI) was correctly deferred due to the sepsis condition. Following surgical debridement of the abscess, the patient's ECG normalized, and subsequent angiography confirmed the absence of coronary stenosis. This case demonstrates that sepsis-induced global ischemia can masquerade as a myocardial infarction. The conclusion is that clinicians must consider non-coronary etiologies for ST-elevation in septic patients to prevent misdiagnosis and ensure that appropriate, life-saving infection management is not delayed.

Keywords: Cardiac Enzymes, Diagnostic Dilemma, Myocardial Infarction, Sepsis, ST-Elevation.

1. Introduction

Myocardial injury is a frequently observed complication in patients with sepsis, significantly contributing to morbidity and mortality rates (Singer et al., 2016). Sepsis, defined as a life-threatening organ dysfunction caused by a dysregulated host response to infection, is a systemic inflammatory condition that can profoundly impact the cardiovascular system (Evans et al., 2021). The pathophysiology of this cardiac dysfunction is complex, involving a delicate balance of pro-inflammatory cytokines and metabolic disturbances that can lead to an imbalance in oxygen supply and demand (Zhang & Dhalla, 2024). In a subset of these patients, the resulting myocardial injury can manifest with notable electrocardiogram (ECG) changes, specifically ST-segment elevation, and a significant rise in cardiac enzymes such as troponin (Vogel et al., 2019) This clinical presentation can create a challenging diagnostic scenario, as it closely mimics an ST-Elevation Myocardial Infarction (STEMI), a condition that requires urgent and specific interventions like primary percutaneous coronary intervention (PCI) (Rudiger & Singer, 2007; Kumara et al., 2024).

The physiological mechanisms underlying this phenomenon are multifactorial. Sepsis-induced systemic inflammation triggers the release of a cascade of inflammatory mediators, including pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6) (Yamamoto et al., 2025). These cytokines contribute to widespread vascular dysfunction, causing vasodilation and subsequent hypotension, which reduces myocardial perfusion pressure and compromises oxygen delivery to the heart (Kakihana et al., 2016). Simultaneously, the heightened metabolic state of the

inflammatory response increases myocardial oxygen consumption (Mouton et al., 2020). This critical imbalance between oxygen supply and demand, coupled with direct myocyte damage from inflammatory mediators and microcirculatory disturbances, can induce transmural myocardial ischemia (De Boer et al., 2003). This form of injury is pathognomonic for ST-elevation and elevated troponin levels, presenting a clinical picture that is indistinguishable from true acute coronary syndrome (ACS) without further investigation (Falk et al., 2022; Singer et al., 2016).

The overlap in clinical presentation between sepsis-induced myocardial injury and true STEMI poses a significant diagnostic dilemma for clinicians (Liu et al., 2025). While both conditions present with ST-segment elevation and elevated troponins, their underlying etiologies and management strategies are fundamentally different. A patient with true STEMI requires immediate reperfusion therapy, typically through primary PCI, to prevent irreversible myocardial necrosis and reduce mortality (Boden et al., 2007). However, performing such an invasive procedure on a patient whose ST-elevation is a result of a sepsis condition, and not due to a coronary artery occlusion, may not only be unnecessary but could also be harmful by delaying critical sepsis management (Evans et al., 2021; Rudiger & Singer, 2007). The issues lie in distinguishing between these two etiologies in a time-sensitive emergency setting.

The urgency of this diagnostic challenge is underscored by the potential for misdiagnosis and inappropriate treatment. A delayed or incorrect diagnosis can lead to adverse patient outcomes, including increased mortality (Hanna et al., 2020). For instance, deferring primary PCI in a true STEMI patient due to a suspected sepsis-mimicking condition could have fatal consequences (Pedersen et al., 2014). Conversely, subjecting a septic patient to an unnecessary invasive procedure can expose them to procedural risks and further complicate their already tenuous clinical status (Falk et al., 2022). The rarity of such a complete clinical mimicry, especially in younger patients without cardiovascular risk factors, makes this a particularly challenging and high-stakes diagnostic scenario that requires a high degree of clinical suspicion and a systematic diagnostic approach (Kakihana et al., 2016; Singer et al., 2016; Kumara et al., 2024).

This case report aims to elucidate the diagnostic challenges associated with ST-elevation mimicking STEMI in a sepsis condition through a case-based analysis. Our primary objective is to highlight the importance of a broad differential diagnosis for ST-elevation in patients with a confirmed or suspected sepsis etiology. The urgency of this research is directly tied to the need for timely and accurate clinical decision-making to optimize patient outcomes and prevent inappropriate interventions. Through the presentation of a detailed case illustration of a patient with sepsis-induced myocardial injury who initially presented with classic STEMI signs, we aim to provide illumination on a practical guide for clinicians on how to navigate this diagnostic dilemma. The novelty of this work lies in its detailed case illustration, which provides a tangible example of this rare phenomenon and reinforces the critical role of comprehensive clinical assessment, including surgical intervention to control the source of infection, as a primary and effective therapeutic strategy in such cases (Kumara et al., 2024). Clearly outline the goals of the project and present a suitable context, while refraining from an extensive review of the literature or a recap of the findings.

2. Literature Review

The intersection of sepsis and cardiovascular dysfunction represents a critical area of research and a significant clinical challenge. Sepsis, defined as a dysregulated host response to infection leading to life-threatening organ dysfunction, frequently complicates with

myocardial injury (Cao et al., 2023). This cardiac involvement is a key contributor to the high mortality rates associated with severe sepsis and septic shock. A particularly complex and high-stakes clinical manifestation of this injury is the appearance of ST-segment elevation on an electrocardiogram (ECG), which closely mimics an acute ST-Elevation Myocardial Infarction (STEMI), a condition caused by a blocked coronary artery (Evans et al., 2021). The ability to differentiate between these two distinct pathologies is paramount (Wasserman & Savargaonkar, 2001), as their management strategies are fundamentally different, and an incorrect diagnosis can lead to grave consequences for the patient.

2.1. Pathophysiology of Sepsis-Induced Myocardial Injury

The mechanism by which sepsis causes myocardial injury is complex and multifactorial, moving beyond a simple oxygen supply and demand imbalance. The systemic inflammatory response to an infection triggers the release of potent pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6) (Falk et al., 2022). These mediators cause widespread vasodilation and a decrease in systemic vascular resistance, which leads to hypotension and reduced coronary perfusion. Simultaneously, the hypermetabolic state of the septic patient increases myocardial oxygen demand. The combination of reduced oxygen supply and increased demand creates an environment of global transmural ischemia rather than localized ischemia from a coronary occlusion (Kumara et al., 2024).

Additionally, direct cellular damage plays a role. Inflammatory mediators and microbial toxins can directly injure cardiac myocytes, leading to myocardial edema, microcirculatory dysfunction, and mitochondrial damage (Lafuse et al., 2020). The release of myocyte-damaging enzymes, particularly high-sensitivity cardiac troponin I (hs-cTn I), is a direct consequence of this injury and is a hallmark of the condition (Ragusa et al., 2023). Unlike a true STEMI, where troponin elevation is a result of a focal ischemic event, in sepsis, the elevated troponin level reflects a more diffuse myocardial injury, which can persist throughout the septic episode (Rudiger & Singer, 2007).

2.2. The Diagnostic Dilemma: Sepsis Mimicking STEMI

The most significant clinical challenge arises from the striking similarity in presentation between sepsis-induced myocardial injury and true STEMI. Both conditions can present with chest pain, dyspnea, ST-segment elevation on ECG, and markedly elevated cardiac enzymes. However, the underlying etiology dictates the appropriate treatment. A true STEMI requires urgent reperfusion therapy, such as primary percutaneous coronary intervention (PCI), to save myocardial tissue (Falk et al., 2022). Conversely, the primary treatment for sepsis is early recognition, fluid resuscitation, and aggressive management of the underlying infection, often involving antibiotics and surgical source control (Jimenez & Marshall, 2001).

Misdiagnosis can be catastrophic. Administering reperfusion therapy to a septic patient without a coronary blockage is not only unnecessary but can also be harmful, potentially delaying crucial infection control measures and exposing the patient to the risks of an invasive procedure (Stephens & Whitman, 2015). Similarly, a patient with a true STEMI who is misidentified as having sepsis- induced cardiac injury could have their life-saving PCI delayed, leading to extensive myocardial damage. Therefore, clinicians are faced with a crucial decision-making process that requires a high degree of clinical suspicion and an awareness of these two distinct etiologies when presented with ST-elevation in a patient with a confirmed or suspected infection (Kumara et al., 2024).

2.3. Clinical Management and Prognosis

The correct clinical approach to a patient presenting with ST-elevation in a sepsis condition begins with a careful evaluation of the patient's history, clinical signs, and laboratory markers (Chaulin, 2021). In the absence of definitive signs of coronary artery occlusion, the priority should be to aggressively manage the sepsis. As demonstrated in a specific case illustration, treating the underlying infection—in this instance, through surgical debridement of a scrotal abscess which can lead to a rapid normalization of ECG findings and a decrease in cardiac enzyme levels (Kumara et al., 2024). Subsequent coronary angiography, showing no significant stenosis, can then confirm the diagnosis of sepsis-induced cardiac injury (Kakihana et al., 2016).

The prognosis of sepsis-induced myocardial injury is a serious concern. While the cardiac dysfunction may be reversible upon successful treatment of the underlying infection, it is a marker of severe sepsis and is independently associated with an increased risk of mortality (Rudiger & Singer, 2007; Singer et al., 2016). Therefore, recognizing this condition and initiating prompt, appropriate management of the sepsis is critical not only for resolving the cardiac symptoms but also for improving the patient's overall survival.

3. Methods

3.1. Research Design

This study employed a descriptive qualitative approach with a case study method to thoroughly investigate the diagnostic dilemma presented by ST-elevation in a patient with a sepsis condition (Sugiyono, 2013). This methodological choice is particularly suitable for exploring complex and rare clinical phenomena, as it allows for an in-depth analysis of a single, unique patient's journey, providing rich, context-specific insights that may not be captured in large-scale quantitative studies (Agustianti et al., 2022). The case study method provides a framework to systematically describe and interpret the sequence of events, from the patient's initial presentation to their final diagnosis and outcome, thereby illustrating the clinical reasoning process and management strategies in a high-stakes setting (Evans et al., 2021; Rudiger & Singer, 2007).

3.2. Population and Sample

The population of interest for this research encompasses all patients presenting with ST-segment elevation on an electrocardiogram (ECG) in the context of sepsis. Based on this population, the sample for this study was a single, purposively selected case. The chosen sample was a 31-year-old male patient who presented with both a clinical picture suggestive of ST-Elevation Myocardial Infarction (STEMI) and a confirmed sepsis condition originating from a scrotal abscess. This case was specifically chosen for its classic yet confounding presentation, which exemplifies the diagnostic challenge that is the central focus of this paper (Kumara et al., 2024). The patient's lack of traditional cardiovascular risk factors further highlights the atypical nature of his ST-elevation, making him a compelling subject for this case study (Falk et al., 2022).

3.3. Data Instruments and Analysis Techniques

The primary research instrument in this study was the researchers themselves, who, as clinicians, were directly involved in the patient's care and data collection. The data were collected from the patient's comprehensive medical records, including detailed clinical history, physical examination findings, laboratory test results, and imaging studies (Emzir, 2014). The specific data points analyzed included serial ECGs, cardiac enzyme levels (hs-cTn

I), complete blood count, and imaging results from the coronary angiography. Data analysis was conducted using a descriptive qualitative technique (Singer et al., 2016). The process involved interpreting the collected data in a logical sequence to construct a narrative of the patient's clinical course. This included identifying key findings, such as the initial ST-elevation and high troponin levels, and correlating them with the subsequent clinical decisions and eventual normalization of the ECG after the source of infection was treated. This approach allowed for a robust conclusion that the myocardial injury was a sequela of sepsis, not a primary coronary event (Kakihana et al., 2016; Kumara et al., 2024).

3.4. Research Procedures

The research procedures followed the chronological course of the patient's hospital stay. Initially, the patient was admitted with a provisional diagnosis of inferoposterolateral STEMI. Given the coexisting diagnosis of sepsis, a critical decision was made to defer primary percutaneous coronary intervention (PCI). Following this, the patient was consulted with a surgical team, and debridement of the scrotal abscess was performed to manage the source of the infection. Post-surgically, the patient's clinical condition and laboratory markers were closely monitored. A repeat ECG was performed two days after the debridement, which showed the ST-elevation had returned to normal limits. Finally, a definitive diagnosis was confirmed with a coronary angiography, which showed no significant coronary artery stenosis, thereby supporting the hypothesis that the myocardial injury was caused by sepsis-induced global ischemia (Evans et al., 2021; Falk et al., 2022; Kumara et al., 2024). This sequence of events, from initial presentation to final diagnosis, forms the basis of the case illustration and the findings presented in this paper.

4. Results and Discussion

4.1. Research Results

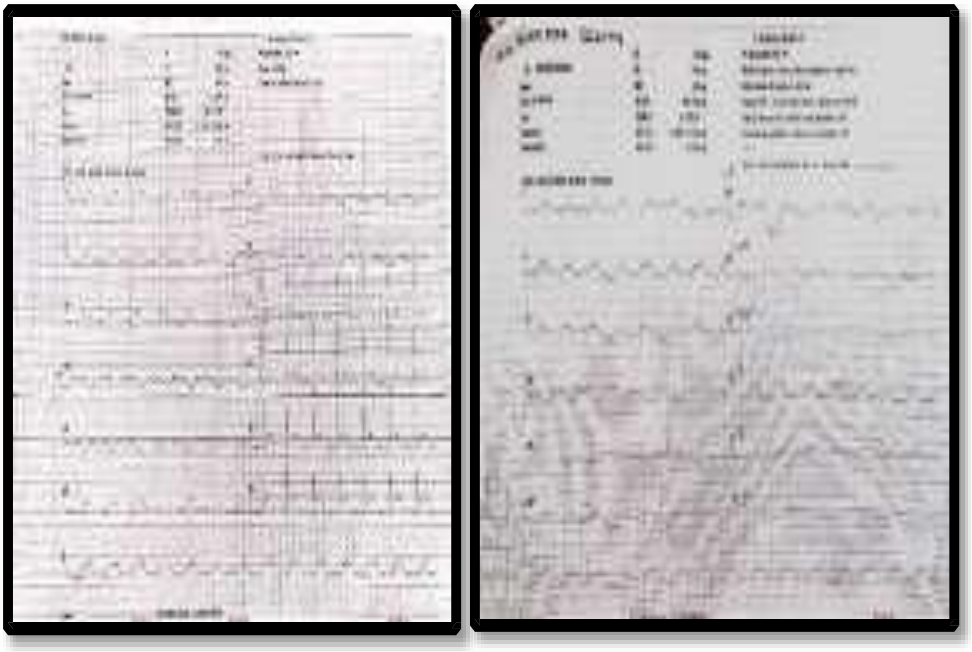

Figure 1. ECG Findings showed ST -Elevation at the inferior posterior lateral leads

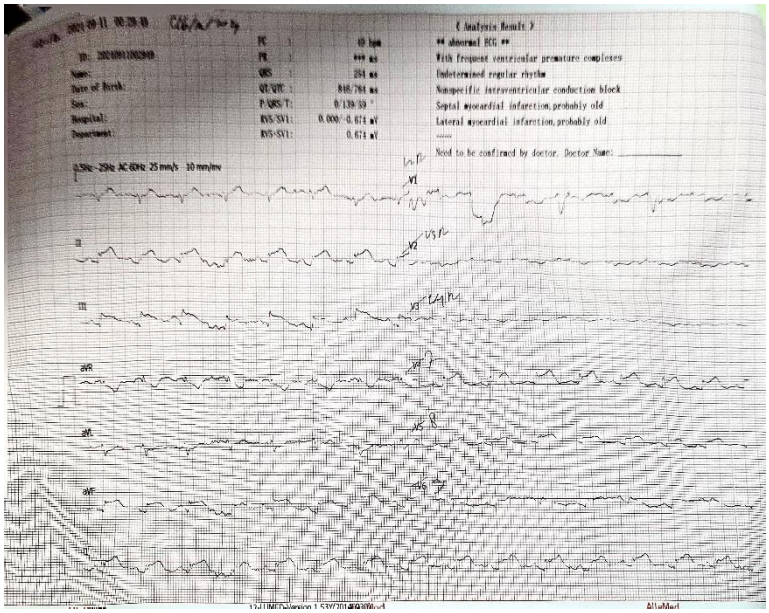

Figure 2. ECG Findings after the inflammation resolved

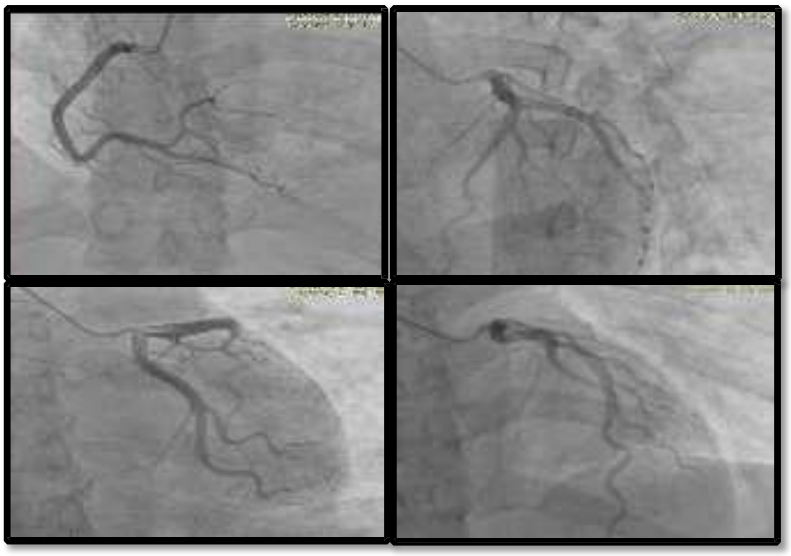

Figure 3. Coronary Angiography showed no evidence of coronary obstructions

This case study centered on a 31-year-old male with a significant diagnostic dilemma. The patient presented to the hospital with a chief complaint of severe epigastric pain radiating to the chest and shortness of breath, symptoms highly suggestive of an acute cardiac event. His initial physical examination revealed a stable but concerning clinical picture, with a blood pressure of 100/70 mmHg and a heart rate of 88 bpm. Crucially, a large scrotal abscess was also identified, providing a clear potential source of systemic infection. These findings immediately raised the suspicion of sepsis as a complicating factor in his cardiac presentation. Initial diagnostic evaluations reinforced the complexity of the case. As shown in Figure 1, the patient's electrocardiogram (ECG) displayed a clear ST-segment elevation in the inferoposterolateral leads, which is the classic electrical signature of a myocardial infarction. Laboratory tests further supported this initial diagnosis with a dramatically elevated high-sensitivity cardiac troponin I (hs-cTn I) level of 40,000 ng/mL, far exceeding the threshold for myocardial injury. However, the patient's leukocyte count was also significantly elevated at 26,080 UL, and his qSOFA score was 2, both indicating a strong systemic inflammatory

response and a high probability of sepsis. The confluence of these cardiac and infectious markers created a challenging scenario where a definitive diagnosis was not straightforward.

The clinical course and subsequent outcomes provided the definitive evidence to resolve this diagnostic conundrum. Given the strong evidence of a sepsis condition, the decision was made to defer primary percutaneous coronary intervention (PCI), a procedure typically reserved for a true STEMI. The patient's primary management focused on controlling the source of infection, and a surgeon performed a debridement of the scrotal abscess. Following this intervention, a repeat ECG performed just two days later showed a complete and rapid normalization of the ST-elevation, with the findings returning to normal limits (see Figure 2). The definitive proof came from a subsequent coronary angiography, which showed absolutely no evidence of significant coronary artery stenosis, thereby ruling out a coronary artery occlusion as the cause of the initial presentation (refer to Figure 3).

4.2. Discussion

This case provides a compelling and vivid illustration of sepsis-induced myocardial injury mimicking ST-Elevation Myocardial Infarction (STEMI). The patient's clinical course directly confirmed the central hypothesis: the cardiac signs and symptoms were not due to a primary coronary event but were a direct consequence of the systemic inflammatory response from a severe infection (Kumara et al., 2024). The complete reversal of ECG abnormalities after successful management of the sepsis, coupled with the definitive coronary angiography findings, provides incontrovertible evidence of this phenomenon. It underscores the critical need for clinicians to maintain a high degree of suspicion for alternative etiologies when a patient presents with ST-elevation in the context of an infectious process.

The pathophysiology in this patient's case is well-supported by established scientific literature. The sepsis-induced systemic inflammation triggered a massive release of pro-inflammatory cytokines, such as TNF-α, IL-1, and IL-6 (Falk et al., 2022). These inflammatory mediators led to widespread vasodilation and hypotension, which significantly reduced myocardial perfusion. This reduction in oxygen supply, combined with the body's heightened metabolic demands in response to the infection, created a critical oxygen supply- demand imbalance. This global, rather than focal, ischemia resulted in transmural myocardial injury, which is the physiological mechanism responsible for both the ST-elevation on the ECG and the massive release of troponin (Kakihana et al., 2016).

The clinical management of this patient highlights a crucial and life-saving decision-making process. The choice to defer PCI, despite the ECG findings, was a deliberate and appropriate course of action based on the full clinical picture. Had the patient been taken for an immediate PCI, the underlying sepsis would have been left untreated, potentially leading to septic shock and death, while also exposing him to the unnecessary risks of an invasive procedure (Evans et al., 2021). Instead, the targeted intervention to control the source of infection through surgical debridement proved to be the definitive treatment for the cardiac symptoms. This demonstrates that in these complex cases, the most effective "cardiac" treatment is often the one that addresses the systemic cause, not the local manifestation.

Ultimately, this case serves as an important reminder of the diagnostic pitfalls in modern medicine. The classic signs of a STEMI can be deceptive, especially in the presence of severe systemic illness. Clinicians must think systematically, integrating a patient's full clinical history and laboratory findings, rather than relying solely on a single diagnostic test. The success of this patient's management, leading to a full recovery of his cardiac function and normalization of his ECG, reinforces that careful, considered clinical judgment is the cornerstone of effective care and is essential to avoiding potentially fatal diagnostic errors (Rudiger & Singer, 2007).

5. Conclusion

This case study highlights the critical importance of comprehensive clinical assessment and diagnostic discernment in distinguishing sepsis-induced myocardial injury from true ST-Elevation Myocardial Infarction (STEMI). Although both conditions can present with indistinguishable electrocardiographic changes and markedly elevated cardiac biomarkers, their underlying pathophysiology and management differ substantially. The presented case clearly demonstrates that myocardial injury in sepsis results from systemic inflammation, cytokine-mediated myocardial depression, and global oxygen supply-demand imbalance, rather than a focal coronary occlusion. The patient's rapid clinical and electrocardiographic recovery following surgical source control, without the need for percutaneous coronary intervention (PCI), provides compelling evidence that timely recognition and treatment of the underlying sepsis are paramount. This reinforces the principle that aggressive infection management, rather than unnecessary invasive cardiac procedures, is the most effective therapeutic approach in such scenarios.

Clinicians should, therefore, maintain a high index of suspicion for sepsis-induced myocardial injury when confronted with ST-elevation in patients with active infection or systemic inflammatory signs. A multidisciplinary approach, integrating cardiology, infectious disease, and surgical expertise, remains essential to achieving accurate diagnosis and optimal outcomes. Ultimately, this case underscores that in complex clinical presentations, the key to successful management lies in prioritizing the systemic cause over its cardiac manifestation.

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