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Non-ST Elevation Myocardial Infarction in the Setting of Multiple Open Fractures: A Case Report

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Abstract

Myocardial infarction (MI) generally occurs due to coronary atherosclerosis. However, under certain circumstances, trauma can act as a trigger for MI. This case report discusses the occurrence of NSTEMI in a patient with multiple open fractures and explores several hypotheses that may contribute to the development of myocardial infarction in the setting of non-thoracic trauma. A 53-year-old man presented to the emergency department following a motorcycle accident. He was treated for multiple open fractures and developed a myocardial infarction on the first day of hospitalization, approximately 40 hours after the trauma. Electrocardiography (ECG) revealed no significant ST-segment changes, while high-sensitivity troponin I levels were markedly elevated at 609.5 ng/mL, and the hemoglobin level was 8.3 g/dL. The patient was co-managed with the cardiology team and received dual antiplatelet therapy (DAPT) and anticoagulation. The orthopedic procedure was postponed until hemodynamic stability was achieved. Myocardial infarction following an open fracture may occur due to rupture of an atherosclerotic plaque, an imbalance between myocardial oxygen supply and demand, and/or increased pro-inflammatory mediators resulting from the bone remodeling process or post-traumatic stress. Myocardial infarction in the setting of open fractures may result from atherosclerotic plaque rupture, oxygen supply–demand imbalance, and/or elevated pro-inflammatory mediators associated with bone remodeling or post-traumatic stress. Increased awareness of potential cardiovascular complications is essential, particularly in trauma patients with pre-existing cardiovascular risk factors.

Keywords: NSTEMI, type 2 myocardial infarction, non-thoracic trauma, fracture

INRODUCTION

Myocardial injury, identified by elevated troponin levels, is a common finding in major trauma patients. However, its detection is particularly challenging because the initial clinical focus of trauma resuscitation is directed toward the most visible injuries and immediately life-threatening conditions. This prioritization may divert attention from more subtle cardiac events. While extensive research has examined myocardial infarction (MI) following direct thoracic trauma, its occurrence in cases of isolated non-thoracic trauma, including open fractures, remains poorly documented. A possible mechanism is type 2 MI, driven by an oxygen supply–demand imbalance resulting from hemorrhage, hypotension, and anemia. Therefore, establishing a conclusive diagnosis and distinguishing between various etiologies require a thorough diagnostic workup. This case report aims to describe a potential association between an open fracture of the lower extremity and the subsequent development of non–ST-segment elevation myocardial infarction (NSTEMI) in a patient with underlying cardiovascular comorbidities. It also underscores the importance of comprehensive cardiovascular evaluation and management in patients with major trauma.

CASE REPORT

A 53-year-old male presented to the emergency department fully conscious after a motorcycle accident. He denied chest pain and reported no history of direct chest trauma. Initial radiographic evaluation revealed multiple open fractures involving the distal third of the left femur, the midshaft of the left tibia, and the base of the first phalanx of the right hand. Open reduction and internal fixation (ORIF) was planned.

On the first day in the High Care Unit (HCU), approximately 40 hours post-trauma, the patient reported sudden-onset central chest pain 30 minutes before evaluation. The pain was described as a heavy,

pressing sensation radiating to the left arm, accompanied by dyspnea unrelieved by positional changes and diaphoresis. His medical history included hypertension, treated with amlodipine 10 mg daily, and active tobacco use.

Vital signs were as follows: blood pressure, 92/59 mmHg; heart rate, 110 beats per minute; respiratory rate, 22 breaths per minute; and oxygen saturation, 98% on 2 L/min supplemental oxygen via nasal cannula. Physical examination revealed no signs of fluid overload or pulmonary edema. Electrocardiography (ECG) demonstrated sinus tachycardia at 116 beats per minute without significant ST-segment deviations, left bundle branch block (LBBB), or pathological Q waves. Serum high-sensitivity troponin I was markedly elevated at 609.5 ng/mL. Laboratory tests showed anemia (hemoglobin, 8.3 g/dL) and impaired renal function (estimated glomerular filtration rate [eGFR], 47 mL/min/1.73 m^2).

A diagnosis of NSTEMI was established. The patient's GRACE score (<140) indicated intermediate risk. The planned orthopedic procedure was postponed until hemodynamic stabilization and cardiac optimization were achieved. Co-management with the cardiology team was initiated, including dual antiplatelet therapy (DAPT) and anticoagulation. The patient also received two units of packed red blood cells.

Throughout hospitalization, the patient remained free of chest pain or shortness of breath. His predominant complaint was pain related to the fractures. ECG performed on day 4 demonstrated sinus rhythm at approximately 93 beats per minute, without significant ST-segment deviation, LBBB, or pathological Q waves. He was discharged on day 9. The planned orthopedic procedure was deferred primarily for insurance-related reasons and will be rescheduled through the outpatient clinic. Further cardiac evaluation, including serial high-sensitivity troponin measurements and echocardiography, was also deferred.

DISCUSSION

Myocardial infarction (MI) is defined as myocardial cell necrosis caused by ischemia, which may result from atherosclerotic plaque rupture or an imbalance between myocardial oxygen supply and demand. Although fractures and MI involve separate organ systems, recent studies have highlighted potential pathophysiological connections between the two. This report describes a patient who developed non–ST-segment elevation myocardial infarction (NSTEMI) following non-thoracic trauma, specifically open fractures of the lower extremities. The patient presented with typical chest pain accompanied by elevated high-sensitivity troponin I levels.

Research on MI occurring after non -thoracic trauma remains limited. In contrast, MI following blunt or penetrating thoracic trauma is well documented and is frequently attributed to direct coronary vascular injury, with the left anterior descending (LAD) artery most commonly affected. (4-6) The mechanisms in non-thoracic trauma, however, appear more complex. Previous studies have reported that trauma-induced immune dysregulation may contribute to secondary cardiac dysfunction. Interactions among pro-inflammatory cytokines, chemokines, and oxidative stress promote endothelial dysfunction and myocardial injury. Inflammatory markers such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) rise after trauma and correlate with myocardial dysfunction. These same cytokines also increase during the bone-healing process, where they serve as essential mediators of callus formation. (8,9)

Animal and human studies have demonstrated that traumatic injury triggers a systemic inflammatory response capable of causing secondary cardiac alterations. [10] Inflammatory mediators and damage-associated molecular patterns (DAMPs), including TNF-α, complement component 5a (C5a), and extracellular histones, are markedly elevated following trauma and have been implicated in post-traumatic cardiac dysfunction. These mediators may directly induce cardiac injury through cardiodepressive effects or promote maladaptive structural alterations, including dysregula-

tion of α-actinin/desmin and translocation of connexin-43, a known substrate for severe arrhythmias. Collectively, these findings suggest that NSTEMI following long-bone fractures may not be solely attributable to coronary plaque rupture but may represent a manifestation of systemic inflammatory cardiomyopathy. (10)

The patient had multiple cardiovascular risk factors, including male sex, age >45 years, hypertension, and active smoking. Previous studies have shown that the majority of MI cases occur in male patients with similar risk factors such as hypertension, diabetes mellitus, and smoking. (3,11,12) Hypertension and smoking are well-established contributors to atherosclerotic plaque formation and subsequent plaque rupture, erosion, or dissection—the hallmark mechanisms of type 1 MI. Plaque rupture may occur spontaneously or may be triggered by psychological or physical stress, both of which are commonly encountered in trauma patients. Activation of this pathological pathway may also be accelerated by increases in blood pressure and heart rate or by heightened coagulability leading to the formation of a platelet-rich thrombus over a pre-existing vulnerable plaque.(13,14)

Another plausible mechanism in this case is type 2 MI. Open fractures are frequently associated with significant blood loss, resulting in anemia. Type 2 MI occurs when myocardial oxygen demand exceeds supply, which may be precipitated by anemia. Reduced hemoglobin levels lead to anemic hypoxia, while decreased cardiac output may contribute to stagnant hypoxia. Low oxygen saturation may also cause hypoxemic hypoxia. Compensatory tachycardia increases cardiac workload and shortens diastole, thereby reducing coronary perfusion and exacerbating myocardial ischemia. (3,7,15)

The underdiagnosis of trauma-induced myocardial dysfunction remains a substantial clinical challenge, as its initial presentation is often nonspecific.(16) Although cardiac troponin (cTn) is central to diagnosing MI, current guidelines limit its use in trauma primarily to the evaluation of blunt cardiac injury. The 2012 Eastern Association for the Surgery of Trauma guidelines recommend measuring cTn only when blunt cardiac injury is suspected. While this conservative approach minimizes unnecessary testing and the risks associated with antithrombotic therapy in bleeding-prone patients, it may fail to identify MI resulting from systemic oxygen supply–demand mismatch.(17) This case highlights this limitation by demonstrating how major open fractures can precipitate NSTEMI. It underscores the need for a more vigilant, individualized diagnostic strategy that incorporates troponin testing and early cardiology consultation when clinical suspicion arises, even in the absence of thoracic trauma.

The Fourth Universal Definition of MI does not require invasive coronary angiography for diagnosis; however, differentiating type 1 from type 2 MI may be clinically challenging. Coronary angiography is often warranted to evaluate for obstructive coronary artery disease and guide management.(7) The primary limitation of this case report is the absence of coronary angiography or percutaneous coronary intervention (PCI), which would have provided definitive evidence of obstruction or the precise site of plaque rupture. As a result, the analysis remains speculative, offering plausible hypotheses that include plaque destabilization, significant anemia causing critical oxygen imbalance, and systemic inflammatory activation due to trauma and fracture healing.

Despite these limitations, the patient was managed appropriately in accordance with current clinical guidelines. Following a thorough risk–benefit assessment, the patient received dual antiplatelet therapy (DAPT), anticoagulation, and packed red blood cell transfusions, and all planned orthopedic procedures were temporarily deferred.

CONCLUSION

Myocardial infarction (MI) following non-thoracic trauma, such as open fractures of the extremities, may occur through several mechanisms. These include systemic inflammatory activation, trauma-induced

stress leading to atherosclerotic plaque rupture, and systemic conditions that create an imbalance between myocardial oxygen supply and demand. The risk of MI is further increased in patients with pre-existing cardiovascular risk factors.

Although coronary angiography was not performed, the diagnosis of non–ST-segment elevation myocardial infarction (NSTEMI) was strongly supported by clinical findings. Prompt and accurate management remains crucial for preventing deterioration of cardiovascular outcomes in trauma patients. Comprehensive monitoring and a multidisciplinary approach to the care of patients with major trauma are essential to reduce morbidity and mortality.

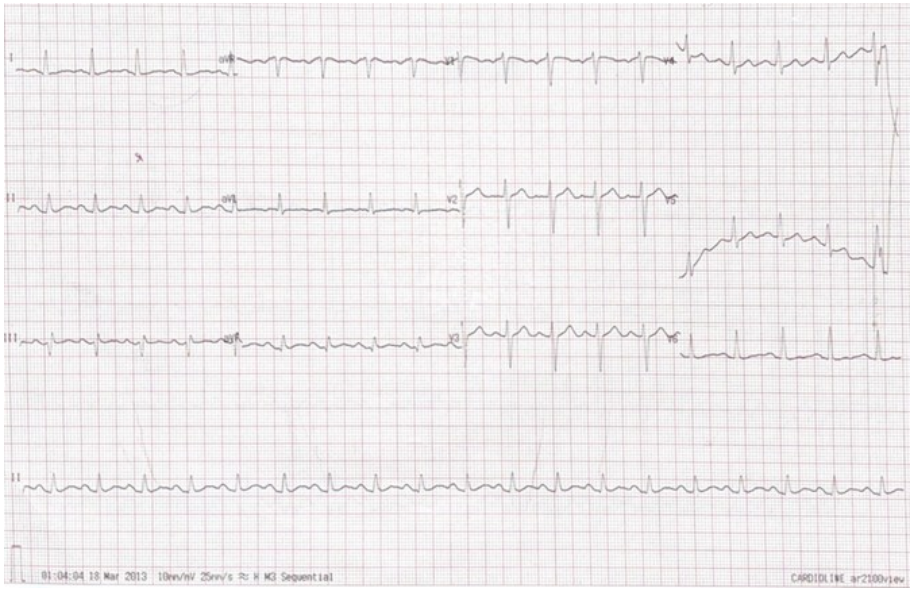

Figure 1. Electrocardiogram (ECG) recorded at the time of chest pain demonstrating sinus tachycardia at 116 beats per minute, without significant ST–T wave changes, left bundle branch block (LBBB), or pathological Q waves.

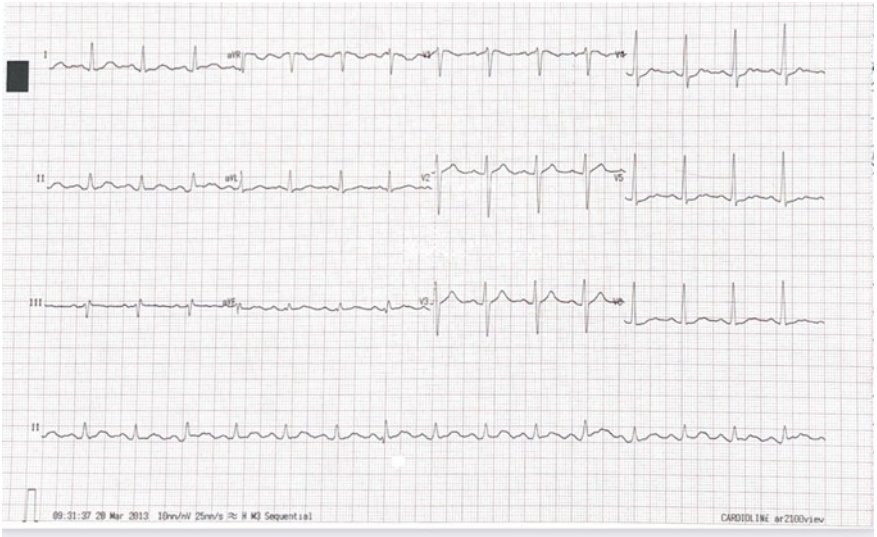

Figure 2. Electrocardiogram (ECG) recorded on day 4 of hospitalization demonstrating sinus rhythm with a heart rate of approximately 93 beats per minute, without significant ST-segment deviation, LBBB, or pathological Q waves.

REFERENCES

1. Karolewski J, Williams JK, Weaver N, Meakes S, Gane K, Balogh ZJ. Epidemiology of myocardial injury in trauma patients: proposed phenotypes for future research. European Journal of Trauma and Emergency Surgery. 2025 Dec 1;51(1).
2. Liu X, Wang A, Liu T, Li Y, Chen S, Wu S, et al. Association of Traumatic Injury and Incident Myocardial Infarction and Stroke: A Prospective Population-Based Cohort Study. Rev Cardiovasc Med. 2023 May 1;24(5).
3. Culhane JT, Mangold MA, Freeman C. Analysis of Predictors of Myocardial Infarction in Trauma With Development of a Trauma Cardiac Risk Index. Cureus. 2021 Feb 6;
4. Bhuller SB, Hasan SR, Weaver J, Lieser M. Acute myocardial infarction following penetrating thoracic trauma: A case report and review of literature. Int J Surg Case Rep. 2019 Jan 1;65:358–60.
5. Fujiwara K, Ohsaka H, Madokoro S, Yanagawa Y. Fatal acute myocardial infarction after multiple blunt injuries involving the chest. J Emerg Trauma Shock. 2018 Jul 1;11(3):230–2.
6. Li X, Wang Z, Yang Y, Meng F, He Y, Yang P. Myocardial infarction following a blunt chest trauma : A case report. Medicine (United States). 2019;98(4).
7. Chapman AR, Taggart C, Boeddinghaus J, Mills NL, Fox KAA. Type 2 myocardial infarction: challenges in diagnosis and treatment. Eur Heart J [Internet]. 2024 Dec 10; Available from: http://www.ncbi.nlm.nih.gov/pubmed/39658094
8. Elhawary H, Baradaran A, Abi-Rafeh J, Vorstenbosch J, Xu L, Efanov JI. Bone Healing and Inflammation: Principles of Fracture and Repair. Semin Plast Surg. 2021 Aug 1;35(3):198–203.
9. Tsioufis P, Theofilis P, Tsioufis K, Tousoulis D. The Impact of Cytokines in Coronary Atherosclerotic Plaque: Current Therapeutic Approaches. Vol. 23, International Journal of Molecular Sciences. MDPI; 2022.
10. Weber B, Lackner I, Knecht D, Braun CK, Gebhard F, Huber-Lang M, et al. Systemic and Cardiac Alterations After Long Bone Fracture. Shock. 2020 Dec 1;54(6):761–73.
11. Hietala P, Strandberg M, Strandberg N, Gullichsen E, Airaksinen KEJ. Perioperative myocardial infarctions are common and often unrecognized in patients undergoing hip fracture surgery. Journal of Trauma and Acute Care Surgery. 2013 Apr;74(4):1087–91.
12. Zheng ML, Du XP, Yang XC, Zheng ML. Bone fracture is associated with incident myocardial infarction in long-term follow-up. BMC Public Health. 2024 Dec 1;24(1).
13. Dutta P, Courties G, Wei Y, Leuschner F, Gorbatov R, Robbins CS, et al. Myocardial infarction accelerates atherosclerosis. Nature. 2012 Jul 19;487(7407):325–9.
14. Nicolò F, Perrone S, Geuna F, Macrini M, Muscoli S, Perrone MA. NSTEMI in a patient admitted for multiple spinal fractures: a clinical case report. J Pathol Locomot Appar-Riv Patol Appar Locomot. 2020;XIX.
15. Padda J, Khalid K, Hitawala G, Batra N, Pokhriyal S, Mohan A, et al. Acute Anemia and Myocardial Infarction. Cureus. 2021 Aug 11;
16. Bekbossynova M, Saliev T, Mukarov M, Sugralimova M, Batpen A, Kozhakhmetova A, et al. Indirect Myocardial Injury in Polytrauma: Mechanistic Pathways and the Clinical Utility of Immunological Markers. Vol. 12, Journal of Cardiovascular Development and Disease. Multidisciplinary Digital Publishing Institute (MDPI); 2025.
17. Culhane JT, Drogan J, Okeke RI, Harjai K. Elevated Cardiac Troponin Is Most Often Associated With Type 2 Myocardial Infarction in Trauma Patients. Cureus. 2023 May 30;