CASE REPORT

Myocardial Injury after Noncardiac Surgery: A Case Report of Acute Chest Pain and Elevated Troponin Levels, Challenges and Management Strategies

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ABSTRACT

Background: Perioperative myocardial injury is a common complication following noncardiac surgery, linked to significant morbidity and mortality. With over 300 million surgeries performed worldwide annually, this number has increased by more than 100 million in the past two decades.

Case: A 42-year-old woman underwent elective craniotomy for a right cerebellar hemangioblastoma and was admitted to the intensive care unit (ICU) postoperatively. On day three, she developed typical chest pain, inferior ST-elevation myocardial infarction (STEMI), and cardiogenic shock. Her medical history included the use of euthyrox following a total thyroidectomy 15 years prior, with no prior cardiac issues. Upon examination, she was conscious but had low blood pressure (58/41 mmHg), an elevated heart rate (103 bpm), and cold extremities. Her troponin I level was elevated at 9383.0 ng/mL. The diagnosis was major postoperative myocardial infarction.

Therapy: The patient received aspirin, atorvastatin, heparin, norepinephrine, and dobutamine for shock management, with close hemodynamic monitoring. By day six, her condition improved, and her electrocardiogram (ECG) was normal. By day nine, she was transferred to the high care unit.

Discussion: Myocardial injury after noncardiac surgery (MINS) includes both myocardial infarction and ischemic myocardial injury, which may not meet the Universal Definition of Myocardial Infarction. MINS usually occurs within 30 days of surgery, especially within the first 72 hours. Its causes are multifactorial, including plaque rupture, oxygen supply-demand imbalance, arrhythmias, and pulmonary embolism. Studies suggest that cardiovascular therapy, including aspirin and statins, is effective. Aspirin reduces 30-day mortality, while statins offer long-term benefits through anti-inflammatory effects. Low-dose aspirin, statins, and Renin-Angiotensin System inhibitors are recommended.

Conclusion: MINS is a serious postoperative complication, even in patients without a history of cardiovascular disease, as demonstrated in this case. Prompt recognition and appropriate therapy with aspirin, heparin, and statins, along with close monitoring, can lead to significant clinical improvement.

Keywords: chest pain; MINS; myocardial infarction; myocardial injury; STEMI

INTRODUCTION

Perioperative myocardial injury is a relatively common complication following noncardiac surgery that is associated with significant morbidity and mortality.[1] It is estimated that more than 300 million patients worldwide undergo surgery each year, an increase of more than 100 million compared to two decades ago.[2,3] Among them, approximately 3 percent are estimated to experience adverse cardiovascular and cerebrovascular events before discharge or within 30 days.[4]

Although significant advancements have been made in preoperative screening, surgical techniques, and patient monitoring, myocardial infarction remains a predominant factor contributing to high rates of cardiovascular morbidity and mortality within 30 days after surgery.[5] Atherothrombosis serves as the primary trigger for non-operative myocardial infarction; however, myocardial injury in noncardiac surgery (MINS), including perioperative myocardial infarction, arises from complex and multifaceted mechanisms that remain challenging to pinpoint in routine clinical practice.[6] From a theoretical perspective, myocardial injury can arise through four distinct mechanisms. First, it may result from the rupture of a coronary plaque, triggering thrombotic occlusion and impairing blood flow. Second, an imbalance between myocardial oxygen supply and demand can lead to ischemic stress, particularly in patients with underlying cardiovascular compromise. Third, non-ischemic cardiac abnormalities, such as atrial fibrillation episodes, may contribute to myocardial dysfunction. Lastly, non-cardiac conditions, including pulmonary embolism, can indirectly induce myocardial injury by compromising oxygenation and hemodynamic stability.[7]

CASE

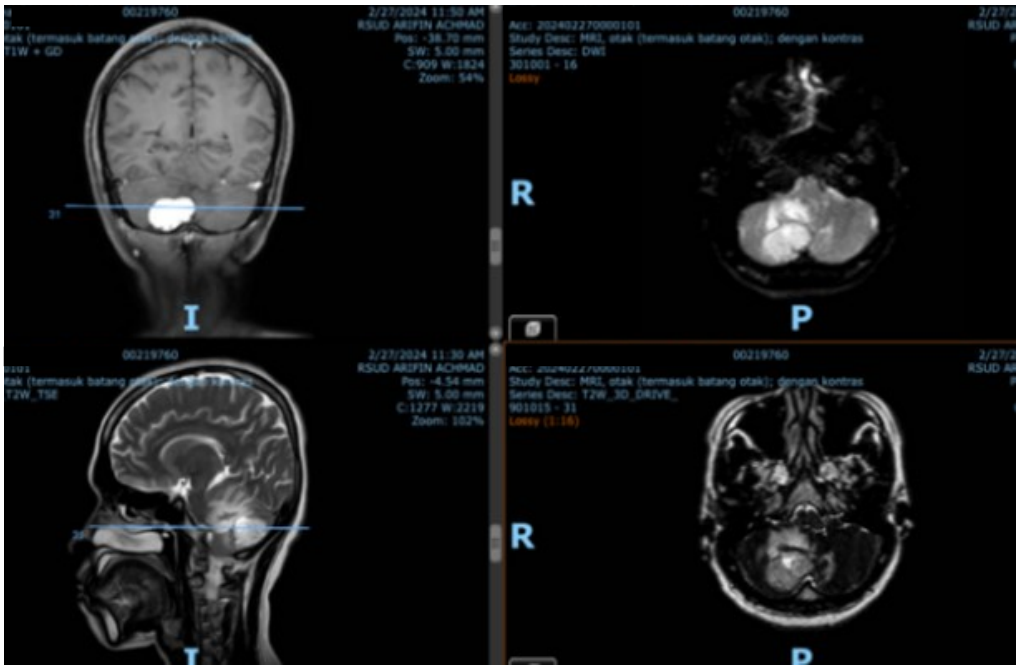


Figure 1. Head CT scan

A 42-year-old woman was admitted to the intensive care unit (ICU) after undergoing an elective craniotomy for right cerebellar hemangioblastoma. On the third postoperative day, she suddenly developed typical chest pain, ST-elevation myocardial infarction (STEMI) on monitor, and cardiogenic shock. The patient had a history of euthyrox use after total thyroidectomy 15 years ago, with no prior history of cardiac disease, chest pain, or deep vein thrombosis (DVT).

On physical examination, the patient was fully alert, with a blood pressure of 58/41 mm Hg, a heart rate of 103 beats per minute, an oxygen saturation of 95% via nasal cannula at 4 lpm, and spontaneous respirations at 20 breaths per minute. No murmurs, gallops, rales, or wheezes were noted on cardiac auscultation, and his extremities were cool. A 12-lead electrocardiogram (ECG) showed STEMI in the inferior leads, ischemic changes in the anterolateral leads, and elevated cardiac enzymes with a troponin I level of 9383.0 ng/mL.

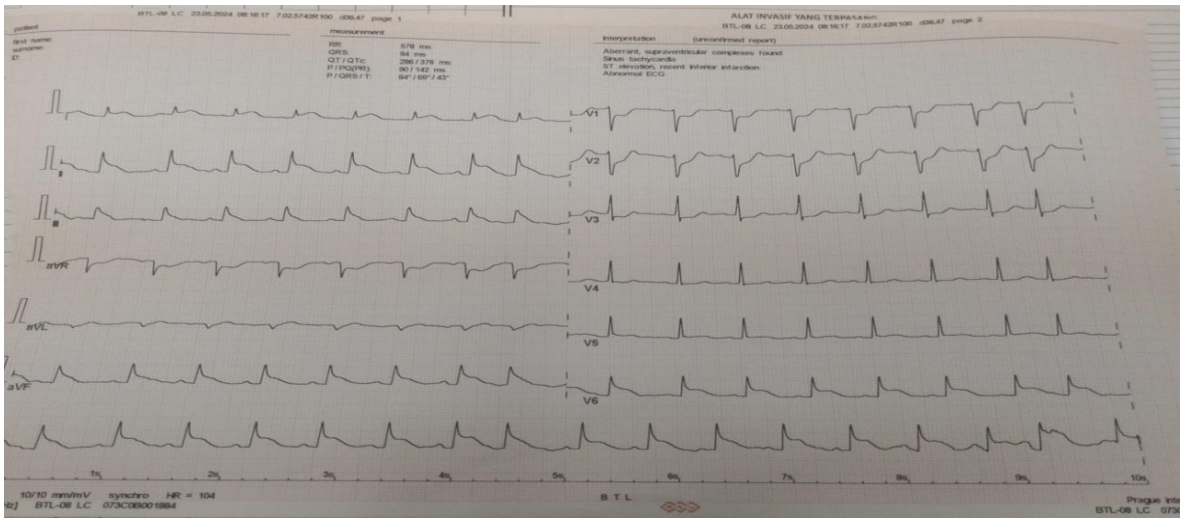


Figure 2. Monitor during an acute chest pain attack

Based on history, physical examination, ECG findings, and laboratory tests, a diagnosis of major postoperative myocardial infarction (after craniotomy for tumor removal) was made. The patient received initial doses of aspirin, atorvastatin, and heparin therapy. Hemodynamic monitoring, prothrombin time (PT), international normalized ratio (INR), and partial thromboplastin time (PTT) were performed to monitor the risk of bleeding. For cardiogenic shock, the patient was given norepinephrine 0.3 mcg/kg/min titrated and dobutamine 15 mcg/kg/min titrated. Arterial blood pressure monitoring was performed for real-time hemodynamic monitoring. On the sixth day, her condition improved, and the ECG showed resolution of the abnormalities. The patient was transferred to the High Care Unit on the ninth day of admission.

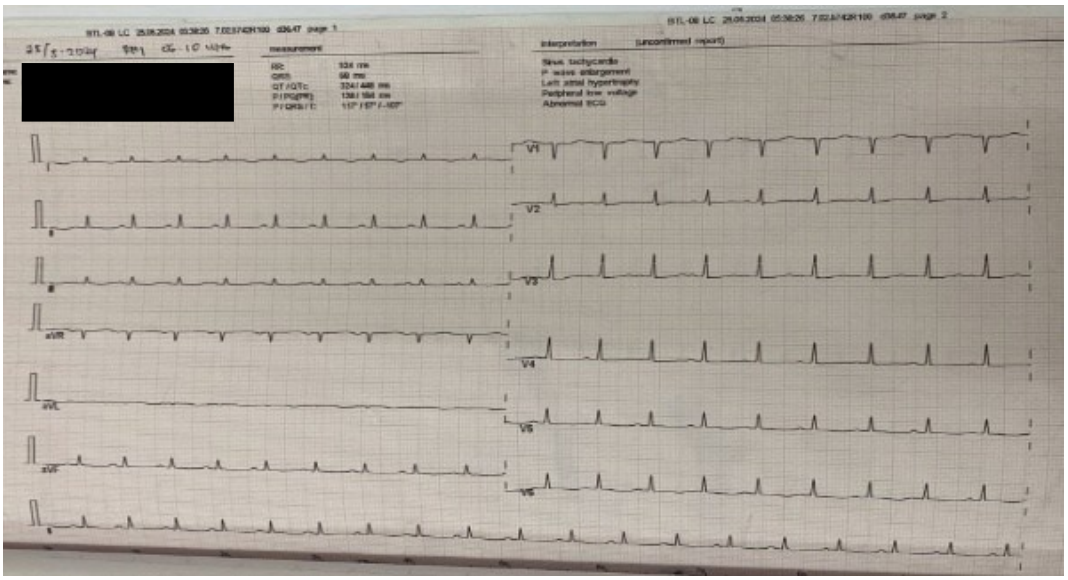


Figure 3. 12-lead EKG on the fifth day (first day post-therapy)

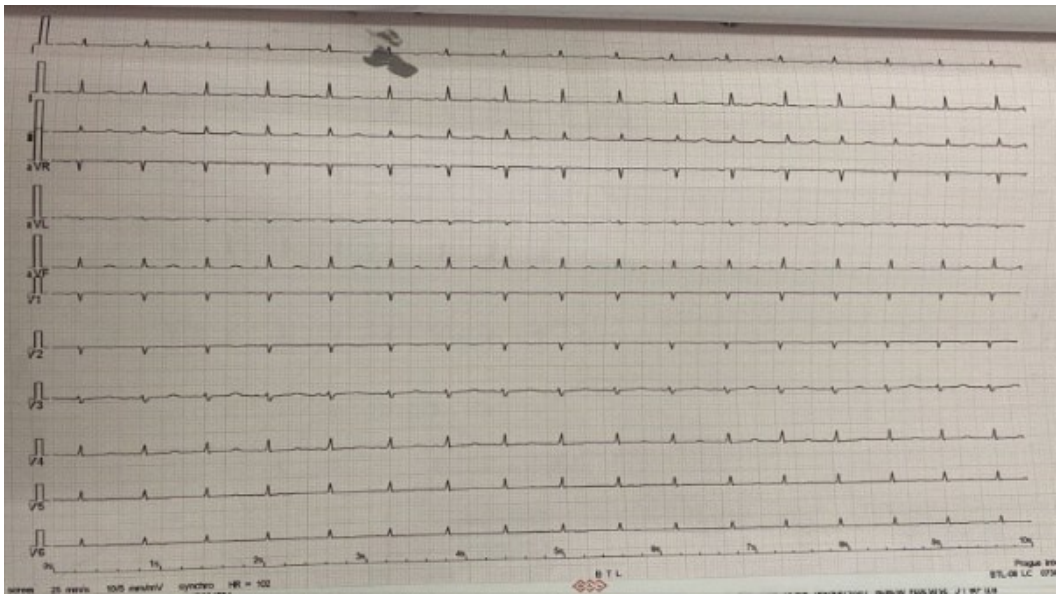


Figure 4. 12-lead EKG on the sixth day (second day post-therapy)

DISCUSSION

A woman with MINS after tumor removal via craniotomy is described in this case report. According to[8] perioperative myocardial injury is a relatively common complication after noncardiac surgery that is associated with significant morbidity and mortality. Meanwhile, according to[9] patients who experience perioperative myocardial injury within the first 7 days postoperatively have a higher risk of 30-day all-cause mortality than those who experience it within 8–29 days.[2] Each year, more than 300 million people undergo major noncardiac surgery worldwide. Despite advances in preoperative screening, surgical improvements, and improved patient monitoring, myocardial infarction remains a leading cause of cardiovascular morbidity and mortality within 30 days of surgery.

Myocardial injury after non-cardiac surgery (MINS) encompasses both myocardial infarction and ischemic myocardial injury that does not align with the universal definition of myocardial infarction. It is characterized by myocardial injury with a rise or fall in cardiac troponin (cTn) exceeding the 99th percentile of the upper reference limit, accompanied by at least one of the following indicators: ischemic symptoms, newly emerging ischemic electrocardiographic alterations, the formation of new pathological Q waves on ECG, imaging findings indicative of myocardial ischemia, or evidence of coronary thrombus confirmed through angiography or autopsy. MINS typically manifests within 30 days post-surgery, with the majority of cases occurring within the first 72 hours.[2,10]

Atherothrombosis is the leading contributor to the majority of nonoperative myocardial infarctions. However, MINS, including perioperative myocardial infarction, arises from a complex interplay of multiple factors. These mechanisms involve hemodynamic instability, inflammatory responses, and prothrombotic states, making it challenging to pinpoint specific causes in routine clinical practice.[11] Theoretically, myocardial injury can arise through four distinct mechanisms. First, it may result from the rupture of a coronary plaque, leading to thrombotic obstruction and impaired myocardial perfusion. Second, an imbalance between myocardial oxygen supply and demand can provoke ischemic stress, particularly in patients with preexisting cardiovascular conditions. Third, non-ischemic cardiac abnormalities, such as atrial fibrillation episodes, can contribute to myocardial dysfunction by disrupting normal cardiac rhythm and hemodynamics. Lastly, non-cardiac factors, including pulmonary embolism, may indirectly induce myocardial injury by impairing oxygenation and circulatory stability. These diverse pathways underscore the complexity of myocardial injury and the challenges associated with its diagnosis and management.[12] However, the angiographic, histologic, or imaging studies necessary to identify all etiological mechanisms of MINS are difficult to apply to all patients undergoing noncardiac surgery.[13] A better understanding of the causes of MINS may help develop potential preventive and therapeutic interventions.[14]

Observational studies have found that cardiovascular medical treatment is effective.[15] Increased doses or early administration of cardiovascular drugs such as antiplatelets, statins, beta-blockers, and angiotensin-converting enzyme (ACE) inhibitors have shown positive results in patients with MINS.[16] Aspirin has been linked to a reduced risk of 30-day mortality in a subgroup analysis of the perioperative ischemic evaluation (POISE) trial.[17] Statins, on the other hand, have demonstrated long-term benefits for patients who survive the perioperative myocardial injury and are discharged alive after MINS.[18] The positive effects of statins may extend beyond their lipid-lowering capabilities, potentially involving anti-inflammatory properties, as elevated levels of C-reactive protein at discharge have been correlated with higher mortality rates in this group.[19] Given these findings, the use of low-dose aspirin and statins is strongly recommended. Additionally, renin-angiotensin-aldosterone system inhibitors, including ACE inhibitors and angiotensin receptor blockers, which are commonly prescribed to hypertensive patients with comorbidities, have also been shown to offer significant benefits in this context.[20,21]

CONCLUSION

Myocardial Injury after Noncardiac Surgery (MINS) is a known complication that has significant implications for patient outcomes, as highlighted by the case of a 42-year-old woman who underwent craniotomy for the removal of a cerebellar tumor. Despite no history of cardiovascular disease, she developed acute postoperative chest pain, with elevated cTn levels confirming myocardial injury consistent with MINS. Prompt initiation of aspirin, heparin, and statin therapy, along with careful monitoring, resulted in improved clinical outcomes and decreased troponin levels. This case highlights the challenges in diagnosing and managing MINS in noncardiac surgical patients, emphasizing the importance of early recognition and tailored treatment strategies to optimize patient care.

REFERENCES

1. Nepogodiev D, Martin J, Biccard B, Makupe A, Bhangu A, Nepogodiev D, et al. Global burden of postoperative death. The Lancet. 2019 Feb;393(10170):401.
2. Weiser TG, Haynes AB, Molina G, Lipsitz SR, Esquivel MM, Uribe-Leitz T, et al. Size and distribution of the global volume of surgery in 2012. Bull World Health Organ. 2016 Mar 1;94(3):201-209F.
3. Meara JG, Leather AJM, Hagander L, Alkire BC, Alonso N, Ameh EA, et al. Global Surgery 2030: evidence and solutions for achieving health, welfare, and economic development. The Lancet. 2015 Aug;386(9993):569–624.
4. Smilowitz NR, Gupta N, Ramakrishna H, Guo Y, Berger JS, Bangalore S. Perioperative Major Adverse Cardiovascular and Cerebrovascular Events Associated With Noncardiac Surgery. JAMA Cardiol. 2017 Feb 1;2(2):181.
5. Smilowitz NR, Gupta N, Guo Y, Berger JS, Bangalore S. Perioperative acute myocardial infarction associated with non-cardiac surgery. Eur Heart J. 2017 Aug 14;38(31):2409–17.
6. Smilowitz NR, Redel-Traub G, Hausvater A, Armanious A, Nicholson J, Puelacher C, et al. Myocardial Injury After Noncardiac Surgery: A Systematic Review and Meta-Analysis. Cardiol Rev. 2019 Nov;27(6):267–73.

7. Beaulieu RJ, Sutzko DC, Albright J, Jeruzal E, Osborne NH, Henke PK. Association of High Mortality With Postoperative Myocardial Infarction After Major Vascular Surgery Despite Use of Evidence-Based Therapies. JAMA Surg. 2020 Feb 1;155(2):131.
8. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction. J Am Coll Cardiol. 2018 Oct;72(18):2231–64.
9. Smilowitz NR, Shah B, Ruetzler K, Garcia S, Berger JS. Characteristics and Outcomes of Type 1 versus Type 2 Perioperative Myocardial Infarction After Noncardiac Surgery. Am J Med. 2022 Feb;135(2):202-210.e3.
10. Nepogodiev D, Martin J, Biccard B, Makupe A, Bhangu A, Nepogodiev D, et al. Global burden of postoperative death. The Lancet. 2019 Feb;393(10170):401.
11. Puelacher C, Gualandro DM, Lurati Buse G, Bolliger D, Marbot S, Kindler C, et al. Etiology of Peri-Operative Myocardial Infarction/Injury After Noncardiac Surgery and Associated Outcome. J Am Coll Cardiol. 2020 Oct;76(16):1910–2.
12. Korsgaard S, Schmidt M, Maeng M, Jakobsen L, Pedersen L, Christiansen CF, et al. Long-Term Outcomes of Perioperative Versus Nonoperative Myocardial Infarction: A Danish Population-Based Cohort Study (2000–2016). Circ Cardiovasc Qual Outcomes. 2022 Jul;15(7).
13. Smit M, Coetzee AR, Lochner A. The Pathophysiology of Myocardial Ischemia and Perioperative Myocardial Infarction. J Cardiothorac Vasc Anesth. 2020 Sep;34(9):2501–12.
14. Puelacher C, Gualandro DM, Glarner N, Lurati Buse G, Lampart A, Bolliger D, et al. Long-term outcomes of perioperative myocardial infarction/injury after non-cardiac surgery. Eur Heart J. 2023 May 14;44(19):1690–701.
15. Korsgaard S, Schmidt M, Maeng M, Jakobsen L, Pedersen L, Christiansen CF, et al. Long-Term Outcomes of Perioperative Versus Nonoperative Myocardial Infarction: A Danish Population-Based Cohort Study (2000–2016). Circ Cardiovasc Qual Outcomes. 2022 Jul;15(7).
16. Duceppe E, Patel A, Chan MTV, Berwanger O, Ackland G, Kavsak PA, et al. Preoperative Terminal Pro–B-Type Natriuretic Peptide and Cardiovascular Events After Noncardiac Surgery. Ann Intern Med. 2020 Jan 21;172(2):96.
17. Oh AR, Park J, Shin SJ, Choi B, Lee JH, Lee SH, et al. Prediction model for myocardial injury after non-cardiac surgery using machine learning. Sci Rep. 2023 Jan 26;13(1):1475.
18. Halvorsen S, Mehilli J, Cassese S, Hall TS, Abdelhamid M, Barbato E, et al. 2022 ESC Guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery. Eur Heart J. 2022 Oct 14;43(39):3826–924.
19. Kim M, Moon I, Bae S, Seo H, Jung IH. Prognostic value of preoperative left ventricular global longitudinal strain for predicting postoperative myocardial injury and mortality in patients undergoing major non-cardiac surgery (SOLOMON study). Int J Cardiol. 2023 May;378:151–8.

20. McKenzie NL, Ward RP, Nagele P, Rubin DS. Preoperative β-Blocker Therapy and Stroke or Major Adverse Cardiac Events in Major Abdominal Surgery: A Retrospective Cohort Study. Anesthesiology. 2023 Jan 13;138(1):42–54.
21. Turan A, Cohen B, Rivas E, Liu L, Pu X, Maheshwari K, et al. Association between postoperative haemoglobin and myocardial injury after noncardiac surgery: a retrospective cohort analysis. Br J Anaesth. 2021 Jan;126(1):94–101.